Núñez-Navarro A, Jiménez-Murcia S, Álvarez-Moya E, Villarejo C, Sanchez-Díaz I, Masuet-Aumatell C, Granero R, Penelo E, Krug I, Tinahones F, Bulik C, Fernández-Aranda F. (2011). Differentiating purging and nonpurging bulimia nervosa and binge eating disorder. ***International Journal of Eating Disorders, 44,*** **488-496. doi: 10.1002/eat.20823**

ABSTRACT

Objective: To explore similarities and differences in clinical and personality variables across three groups: binge eating disorder (BED), bulimia nervosa-purging type (BN-P), and bulimia nervosa-non purging type (BN-NP).

Method: The participants were 102 female eating disorders patients (34 BED, 34 BN-P, and 34 BN-NP) consecutively admitted to the eating disorders unit, at the University Hospital of Bellvitge, and diagnosed according to DSM-IV criteria.

Results: BED patients were older, and more likely to have personal and family history of obesity. A gradient in psychopathological scores emerged with BN-P patients having higher pathological scores on the SCL-90-R, followed by

BN-NP and BED patients. No statistically significant differences were observed in personality traits.

Discussion: Our data supported that eating disorders (namely BED, BN-NP, and BN-P) followed a linear trend in general psychopathology. Whereas personality may represent a shared vulnerability factor, differences in clinical severity suggest there to be a continuum with BN-P being the most severe and BED being the least severe. VC 2010 by Wiley Periodicals, Inc.

Keywords: binge eating disorder; bulimia nervosa; personality; psychopathology; classification

Introduction

Considerable research attention has been paid to the relation between bulimia nervosa (BN) and anorexia nervosa (AN), by exploring differences and similarities in biological factors, personality traits, psychopathology, and clinical variables.1–4

However, other subcategories subsumed under eating disorders not otherwise specified (EDNOS), have received less scientific attention5 despite being more common than AN or BN.6–8 To date, the most commonly described type of EDNOS is binge eating disorder (BED), from the perspective of clinical presentation,9–13 psychopathological features9–11,14 and treatment response.11,15,16

The validity of current eating disorder diagnostic criteria has been the theme of continuous debate.11,13,16–20 Given the paucity of systematic investigations, few firm conclusions can be drawn regarding whether BED warrants an official independent diagnostic classification.11,17,21,22 Carefully designed studies are required to assist in the determination of whether BED represents a discrete and unique category, or is better subsumed under other diagnostic categories such as BN or obesity.

Clinical and Sociodemographic Features

The boundaries between BED and BN in psychopathology and diagnostic domains (and specifically between BED and BN non-purging subtype) are quite unclear.13 When comparing across all eating disorders categories (BED vs. BN, and especially purging subtype), BED is characterized by lower levels of dietary restraint,23–25 more frequent current and premorbid obesity,16,20 greater body dissatisfaction,11 lower likelihood of a previous history of AN,9,20 poorer overall physical health,26 less eating disorder symptomatology,24,27 differential cognitions and behaviors associated with binge eating,28,29 later age of onset12 and better prognosis.18,24

Although differences between the purging subtype of BN and BED are identified by the absence of compensatory behaviors,20 the boundary between the nonpurging subtype of BN and BED is much less clear.9,21 Ramacciotti et al.,21 in a crossectional clinical study with limited sample size, suggest that the differences between BED and BNNP seem to be more of degree than of type, with patients showing similar psychopathological and eating profiles and comparable levels of social and occupational maladjustment secondary to the eating disturbance. Accordingly, Hay and Fairburn,24 in a longitudinal two stage design general population study, also found no significant differences between individuals with BN-NP and those

with BED on general psychopathology (obtained by personal interviews), social adjustment, or self-esteem. They suggested that bulimic eating disorders may exist on a continuum of severity, with the BN purging representing the most extreme form, BED the least severe form, and BN nonpurging resting intermediate between the two. However, neither of these studies considered other relevant personality traits or psychopathology variables (assessed with standardized procedures).

Psychopathological Factors and Comorbidity

No consensus exists in the literature comparing BED and BN on general psychopathological indices. While some studies found no significant differences between comorbidity profiles of individuals with BN and BED,12 others report greater comorbidity and general psychopathological burden in BN.14,19,30,31 Van Hanswijck et al.,14 suggest that personality disorder difficulties are present in patients who binge eat, while obese patients who do not binge eat display significantly less personality disorder pathology. In general, studies that have examined personality disorders in BED suggest that avoidant, obsessive-compulsive, and borderline personality disorders are the most common.14,23,32,33

In general, BED patients display high self-criticism, low self-esteem, depressive symptoms, and over-evaluation of shape and weight.34–36 The relation between self-criticism and over-evaluation of shape and weight may be partly mediated or explained by low self-esteem and depressive symptoms.34

Personality Traits

Although a considerable literature exists examining personality traits in eating disorders,3,37–39 little has been done comparing personality traits in BED versus BN. Pratt et al.36 compared perfectionism in individuals with BN, BED, and obesity. All three groups demonstrated similar levels of both socially prescribed and other-oriented perfectionism. Individuals in the BN and BED groups scored significantly higher on these measures than participants in the obese group.

Biological Variables

A body of literature has arisen to explore genetic factors in eating disorders.4,40,41 Bulik et al. reported the heritability of binge eating to be 50 and 60% for general BN, while the remaining variance was explained by individual specific environmental factors. Shared environmental factors did not contribute to liability to binge eating,42 whereas nonshared environment may be more relevant.43 In a subsequent study, focused on obesity and binge eating, they found that there was a substantial contribution of additive genetic effects to both obesity and binge eating and they further revealed a modest overlap of genetic factors that contribute to each of these two traits.44

Despite a number of sporadic studies, no consistent body of literature has emerged to identify commonalities and differences between BED and BN across clinical, comorbidity, and personality domains. Hence, little evidence exists to assist with evaluating the validity of the current nosological differentiation across these three diagnostic categories (BED vs. BN-P vs. BN-NP).

Aims of the Study

The goals of this study were threefold: (1) to investigate whether individuals with BED or BN subtype (purging and nonpurging) differed significantly on sociodemographic, clinical, and psychopathological variables; (2) to compare personality traits of females with BED, BN-P, and BNNP.

Based on previous reports,21,45 we hypothesized that individuals with nonpurging forms of these eating disorders (i.e., BED and nonpurging BN) would exhibit similar personality traits and psychopathology, while individuals with purging profiles (i.e., BN-P) would be distinct and evidence greater psychopathology.

Method

Participants

The participants were 102 female eating disorders patients (34 BED, 34 BN-P, and 34 BN-NP) with a mean age of 28.2 years (SD5 9.4). BED and BN-NP groups consisted of patients consecutively admitted to our Department of Psychiatry, Eating Disorders unit. This adult unit is specialized in outpatient and inpatient treatment for eating disorders, in Barcelona (Spain). The BN-P group was randomly selected from the pool of BN-P patients attending our unit. To obtain equal sample sizes, given that the number of BN-P patients attending our unit is much higher than the number of patients with BED or BN-NP diagnoses, we randomly selected 34 BN-P cases, by using a SPSS computerized procedure, from a larger pool of 418 BN-P cases attended consecutively during this period of time. BN-NP patients had no history of purging behaviors.

All patients were diagnosed according to DSM-IV criteria,46 conducted by trained psychologists and psychiatrists. The majority of the patients were single (70.3%) and reported primary education (up to 8 years) (41.6%) and secondary education (up to 12 years) studies (46.5%). 83.8% were employed. Entry into the study occurred between December 2002 and December 2006. We obtained written informed consent from all participants and the study was approved by the Ethics Committee of our hospital.

For the present analysis, we excluded the following cases: (a) males (N 5 29), as the number of males with these diagnoses was too small for meaningful comparison (24 BN-P; 1 BN-NP, and 4 BED); and (b) BED patients who had fulfilled criteria for BN in the past or who presented any subthreshold BN symptoms (e.g., irregular vomiting) (n 5 7; 17.1%). BED patients with previous BN were excluded in order to obtain a more homogeneous group, and discard any possible confounding influence of previous BN in the results. No patients refused to participate.

Assessment

We developed a comprehensive battery of assessments to quantify eating disorder symptoms, general psychopathology, and personality. The battery included the Eating Attitudes Test (EAT-40),47 the Eating Disorders Inventory2 (EDI-2),48 the Bulimic Investigatory Test Edinburgh (BITE),49 the Symptom Checklist-Revised-90Revised (SCL90-R),50 and the Temperament and Character Inventory-Revised (TCI-R).51 Additional demographic information including education, occupation and living arrangements was obtained via semi-structured interviews, and also current body weight.

Eating Attitudes Test

This questionnaire contains 40 items,47 including symptoms and behaviors common to eating disordered patients and provides an index of the severity of the disorder. Scores on this questionnaire range from 0 to 120. The higher the scores, the more disturbed the eating behavior. This questionnaire was adapted to the Spanish population showing high internal consistency (Cronbach's alpha coefficient 5 0.93).52

Eating Disorders Inventory 2

This is a reliable and valid 91-item multidimensional self-report questionnaire53 that assesses different cognitive and behavioral characteristics, which are typical for eating disorders. The EDI-2 retains the 64 items (grouped into eight scales: Drive for Thinness, Bulimia, Body Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust, Interoceptive Awareness, Maturity Fears) of the EDI and adds 27 new items into three provisional scales: Asceticism, Impulse Regulation, and Social Insecurity. All of these scales are answered on a 6-point Likert scale, and provide standardized subscale scores. This instrument was validated in a Spanish population48 with a mean internal consistency of 0.63 (coefficient alpha).

The Bulimic Investigatory Test Edinburgh

This questionnaire49 contains 33 items that measure the presence and the severity of bulimic symptoms. There are two subscales: the symptomatology scale (30 items), that determines the seriousness of the symptoms, and the severity scale (three items) that offers a severity index. The cut-off point for the symptomatology scale scores for the present study were as follows; ?10 5 no symptomatology; 10–20 subclinical symptoms and ?20 clinical symptoms. The higher the scores, the greater the severity. This questionnaire has been found to have a high internal consistency (Cronbach's alpha coefficient range: 0.96) and has been adapted to the Spanish population.54

Symptom Checklist-Revised

To evaluate a broad range of psychological problems and symptoms of psychopathology,50 the SCL-90-R was employed. This test contains 90 items and helps to measure nine primary symptom dimensions, which are: Somatization, Obsession-Compulsion, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism. In addition, it includes three global indices, which are a global severity index (GSI), designed to measure overall psychological distress; a positive symptom distress index (PSDI), designed to measure the intensity of symptoms as well as a positive symptom total (PST), which are reports of self-reported symptoms. The Global Severity Index can be used as a summary of the test. This scale has been validated in a Spanish population55 obtaining a mean internal consistency of 0.75 (Coefficient alpha).

Temperament and Character Inventory-Revised

The TCI-R56 is a 240-item, five point Likert scale, reliable and valid questionnaire that measures, as in the original TCI version,57 seven dimensions of personality: four temperament (Harm Avoidance, Novelty Seeking, Reward Dependence and Persistence) and three character dimensions (Self-Directedness, Cooperativeness, and Self-Transcendence). The performance of the Spanish version of the original questionnaire58 and the revised version have been documented. The scales in the latter showed an internal consistency (coefficient alpha) of 0.87.

Procedures

Experienced psychologists and psychiatrists, with masters or doctoral degrees in a mental health discipline, completed the anamnesis during two structured face to face interviews. The participants were assessed by means of structured face-to-face clinical interviews modeled after the Structured Clinical Interview for DSM-IV, SCID-I59 covering lifetime presence of impulsive behaviors (namely alcohol and drug abuse, comorbid, impulse control disorder and suicide attempts), as well as additional information regarding family history of obesity [defined as positive when a subject recalled having a first-degree relative (mother or father) who had ever been diagnosed with obesity by a physician].60 Both interview sessions last ?30 min. The first session established the specific eating disorder diagnosis and related clinical questions (age of onset, duration, course of the disorder, minimum and maximum body mass index [BMI: weight (Kg)/ height2 (m2)] ever achieved). Weight and height were directly measured by the interviewer during this session to calculate BMI. The second session addressed additional questions about psychopathology and family history of obesity. The above mentioned battery of tests is administered in our unit just after the second interview session and lasts ?60 min.

Statistical Analyses

All the analyses were conducted with SPSS v15. Comparison of sociodemographic, clinical, and personality features among groups was conducted with ANOVA procedures and post-hoc comparisons for quantitative variables (Scheffé). Categorical variables were compared with chi-square tests or Fisher exact test as appropriate, adjusted for age.

We conducted an associative analysis in order to determine which variables were most strongly associated with each diagnosis. Bivariate comparisons between group and clinical and personality variables were first applied to select those variables that would enter the regression analysis. Thus, age of onset of the eating disorder, EAT-40 score, EDI-2 Impulsivity subscale, BITE Severity, suicidal ideation, family history of obesity, lifetime obesity, and the SCL-90-R General Severity Index (GSI), were all entered in the regression model as independent variables, while group (BED, BN-P, BN-NP) was the dependent variable. We applied multinomial logistic regression models (BACKWARD procedure) by using BED as the reference group. To measure the differences between BN-P and BN-NP patients, binary logistic regression analyses were also performed. To correct for multiple comparisons, an alpha level of 0.01 was established.

Results

Sociodemographic Variables

The comparison of the sociodemographic characteristics across all three groups revealed statistically significant differences in age (F 5 15.2; df 5 2; p\ 0.0005) and marital status (chi2 5 16.7; df 5 4; p 5 0.002). BED patients were significantly older (mean 5 34.5; SD 5 9.0) than the other two groups (BN-NP: mean 5 25.8, SD 5 9.5; BN-P: mean 5 24.2, SD 5 6.0/Scheffe post-hoc comparison: p \ .0005 for both BN-P and BN-NP) and were more frequently married (50.0% versus 17.6% BN-NP patients and 9.1% BN-P patients).

Clinical Variables

Table 1 presents descriptive parameters and results of the comparison of clinical variables across the three groups.

ANOVA comparisons yielded significant group differences on age of onset of the eating disorder (later for BED patients), current BMI (higher in BED patients), maximum and minimum BMI ever achieved (higher in BED patients), presence of current and lifetime obesity and presence of family history of obesity (all higher in BED patients). The observed difference in age of onset was not significant after adjustment for age.

Psychometric Tests

Table 2 presents means, standard deviations, and results of ANOVA analyses comparing psychometric variables across groups. In general, BN-P patients reported the most pathological scores on all tests, followed by BN-NP patients, with BED patients showing the least pathological scores. These differences were statistically significant at p \ .01 level on the EAT-40 total score, EDI-2 Impulsivity subscale, BITE Severity score, and SCL90-R Paranoid Ideation and Psychoticism subscales. After adjusting for age, the difference on SCL-90-R Psychoticism subscale was no longer significant. No statistically significant differences on personality traits (as measured by the TCI-R) were observed across groups.

Associative Analysis

The results of multinomial analyses measuring which combination of clinical variables was associated with clinical diagnosis (BN-P vs. BN-NP vs. BED) are presented in Table 3. BITE severity scores and lifetime obesity were significantly associated with group membership. Thus, the presence of lifetime obesity was associated with a diagnosis of BED, while greater severity of bulimic symptoms (as measured by the BITE) was associated most strongly with BN-P and also with BN-NP diagnoses. The final model explained 56.8% of variability in group membership and was statistically significant (chi2 5 56.96; df 5 4; p <.0005). Binary logistic regression analyses comparing BN-P and BN-NP patients (BACKWARD -Likelihood ratio procedure) indicated that higher BITE Severity (OR 5 1.187; 95% CI 5 1.060–1.328; p =.003) and, at a trend level, presence of suicidal ideation (OR 5 3.774; 95%CI 5 1.094–13.014; p = .035) were associated with a BN-P diagnosis in relation to BN-NP. This model was statistically significant (chi2 5 23.39; df 5 3; p< .0005) and explained 40.8% (Nagelkerke R2 5 0.408) of the variability in diagnosis (other variables were automatically selected for the final model but they did not reach statistical significance).

Discussion

We examined clinical, psychopathological and personality differences in three groups of patients with eating disorders (ED), namely BED, BN-P, and BNNP, to determine the extent to which the three groups represent different diagnostic categories.

Sociodemographic and Clinical Features

As reported in previous studies12 BED patients were the older than participants with both subtypes of BN. In agreement with previous studies,16,20 and were significantly more likely to report a family history of obesity and lifetime obesity. Obesity was the one clinical dimension that clearly differentiated BED from both subtypes of BN. After excluding those individuals who developed BED after BN, which could possibly represent a subgroup of patients with residual symptomatology (where a combination of subthreshold BN symptoms might be present),61 rather than a distinct psychiatric syndrome, the greater personal and family history of obesity is remarkable. A bivariate twin study,44 identified a modest genetic correlation of 1.34 (95%CI 5 0.19–0.50) between the obesity and binge eating. These results suggest a qualitative difference between BED and BN subtypes on the dimension of obesity.

Psychopathology and Personality Traits

In terms of psychometric results, BN-P patients showed the highest scores on all clinical and psychopathological tests, followed by BN-NP patients and, finally, BED patients, who reported the least pathological profile. Specifically, BN-P patients showed the highest scores on Paranoid Ideation, Impulsivity (EDI-2), severity of bulimic psychopathology, and general eating symptomatology, as measured by the EAT mean score. This confirms previous reports,11,24,27 in which BN-purging subtype was associated with higher impulsivity and some psychopathological traits. Unlike the findings for obesity, the observed results on psychometric indices suggest more of a continuum of clinical severity across the three diagnostic groups rather than discrete differences, with BED patients on the least severe end and BN-P patients on the most severe. In the personality domain, no differences were

found across groups. These results are intriguing and suggest that none of our measured personality dimensions was able to distinguish among these three diagnostic groups. Personality might represent a shared vulnerability factor for these eating disorders but not play a role in the emergence of differential symptom expression across these three subtypes.

Associative Analysis

The associative analysis yielded few clinical differences across groups. BN-P and BN-NP patients distinguished themselves from BED patients with more severe bulimic symptoms and a lower risk of lifetime obesity, as expected. Therefore, severity of the eating disorder and obesity were the main differential factors between the diagnoses of BED, on the one hand, and BN, on the other.11,17,62 The only differences between the two BN subtypes were the severity of clinical symptoms (higher in the purging type).

Limitations

Limitations of this study include the relatively small sample size for the subtype comparisons, and the assessment procedures did not allow us to evaluate either specific psychopathological symptoms or comorbid disorders more broadly (namely affective and anxiety disorders). Additional assessments might have been useful for a more comprehensive characterization of eating disorder symptomatology (e.g., the Eating Disorders Examination). Furthermore, the evaluation of nosological accuracy of these three diagnostic categories could be enhanced with the inclusion of biological and genetic indices in adequately powered studies. Our measure of family history of obesity was also imprecise as many individuals may not be aware of discussions between their family members and their physicians-thus leading to an underestimate of the frequency with which family members suffered from obesity. Finally, the use of two different recruitment methods (consecutive admissions for BED patients and randomized selection of BN patients) may have introduced a sampling bias.

Conclusion

Our measurement of three dimensions—sociodemographics, clinical presentation, and personality—allowed us to build a comprehensive picture of commonalities and differences across these three diagnostic subgroups.

Overall, it appears that there are no differences in personality traits across these three disorders. This could suggest an underlying shared personality style that indexes vulnerability to any eating disorder characterized by binge eating. The dimensions of clinical severity suggest dimensional differences across the three diagnosis with BN-P representing the most severe and BED the least severe. The sole but important difference that emerged was on obesity and related family history of obesity. Obesity is much more strongly associated with BED than with either form of BN. Furthermore, since our results point to the similarities and differences across those eating disorders, future studies should explore their response to treatment, and underlying biological indices, allowing enhanced tailoring of interventions (both behavioral and pharmacological).

References

1. Cassin SE, von Ranson KM. Personality and eating disorders: A decade in review. Clin Psychol Rev 2005;25:895–916.
2. Fassino S, Amianto F, Gramaglia C, Facchini F, Abbate Daga G. Temperament and character in eating disorders: Ten years of studies. Eat Weight Disord 2004;9:81–90.
3. Alvarez-Moya EM, Jimenez-Murcia S, Granero R, Vallejo J, Krug I, Bulik CM, et al. Comparison of personality risk factors in bulimia nervosa and pathological gambling. Compr Psychiatry 2007;48:452–457.
4. Ribases M, Fernandez-Aranda F, Gratacos M, Mercader JM, Casasnovas C, Nunez A, et al. Contribution of the serotoninergic system to anxious and depressive traits that may be partially responsible for the phenotypical variability of bulimia nervosa. J Psychiatr Res 2008;42:50–57.
5. le Grange D, Binford RB, Peterson CB, Crow SJ, Crosby RD, Klein MH, et al. DSM-IV threshold versus subthreshold bulimia nervosa. Int J Eat Disord 2006;39:462–467.
6. Anderson AE, Bowers WA, Watson T. A slimming program for eating disorders not otherwise specified. Reconceptualizing a confucing, residual diagnostic category. Psychiatric Clinics N Am 2001;24:271–280.
7. Grilo CM, Devlin MJ, Cachelin FM, Yanovski SZ. Report of the National Institutes of Health (NIH) workshop on the development of research priorities in eating disorders. Psychopharmacol Bull 1997;33:321–333.
8. Rodriguez-Cano T, Beato-Fernandez L, Belmonte-Llario A. New contributions to the prevalence of eating disorders in Spanish adolescents: Detection of false negatives. Eur Psychiatry. 2005; 20:173–178.
9. Santonastaso P, Ferrara S, Favaro A. Differences between binge eating disorder and nonpurging bulimia nervosa. Int J Eat Disord 1999;25:215–218.
10. Tobin DL, Griffing A, Griffing S. An examination of subtype criteria for bulimia nervosa. Int J Eat Disord 1997;22:179– 186.
11. Fichter MM, Quadflieg N, Brandl B. Recurrent overeating: An empirical comparison of binge eating disorder, bulimia nervosa, and obesity. Int J Eat Disord 1993;14:1–16.
12. Barry DT, Grilo CM, Masheb RM. Comparison of patients with bulimia nervosa, obese patients with binge eating disorder, and nonobese patients with binge eating disorder. J Nerv Ment Dis 2003;191:589–594.
13. Mond JJ, Hay PJ, Rodgers B, Owen C, Mitchell J. Correlates of the use of purging and non-purging methods of weight control in a community sample of women. Aust NZ J Psychiatry 2006; 40:136–142.
14. van Hanswijck de Jonge P, Van Furth EF, Lacey JH, Waller G. The prevalence of DSM-IV personality

pathology among individuals with bulimia nervosa, binge eating disorder and obesity. Psychol Med 2003;33:1311–1317.

15. Hay PJ, Bacaltchuk J, Stefano S. Psychotherapy for bulimia nervosa and binging. Cochrane Database Syst Rev 2004:CD000562.
16. Schneider M. Bulimia nervosa and binge-eating disorder in adolescents. Adolesc Med 2003;14:119–131.
17. Latzer Y, Tzchisinki O. [Binge eating disorder (BED)–new diagnostic category]. Harefuah 2003;142:544–549, 564.
18. Fairburn CG, Cooper Z, Doll HA, Norman P, O'Connor M. The natural course of bulimia nervosa and binge eating disorder in young women. Arch Gen Psychiatry 2000;57:659–665.
19. Crow SJ, Stewart Agras W, Halmi K, Mitchell JE, Kraemer HC. Full syndromal versus subthreshold anorexia nervosa, bulimia nervosa, and binge eating disorder: A multicenter study. Int J Eat Disord 2002;32:309–318.
20. Striegel-Moore RH, Cachelin FM, Dohm FA, Pike KM, Wilfley DE, Fairburn CG. Comparison of binge eating disorder and bulimia nervosa in a community sample. Int J Eat Disord 2001;29:157– 165.
21. Ramacciotti CE, Coli E, Paoli R, Gabriellini G, Schulte F, Castrogiovanni S, et al. The relationship between binge eating disorder and non-purging bulimia nervosa. Eat Weight Disord 2005; 10:8–12.
22. Williamson DA. Does the evidence point to a binge eating phenotype?: Comment on Gordon et al. (2007) and Wonderlich et al. (2007). Int J Eat Disord 2007;40 (Suppl):S72–S75.
23. Wilfley DE, Friedman MA, Dounchis JZ, Stein RI, Welch RR, Ball SA. Comorbid psychopathology in binge eating disorder: relation to eating disorder severity at baseline and following treatment. J Consult Clin Psychol 2000;68:641–649.
24. Hay P, Fairburn C. The validity of the DSM-IV scheme for classifying bulimic eating disorders. Int J Eat Disord 1998;23:7–15.
25. Masheb RM, Grilo CM. Binge eating disorder: A need for additional diagnostic criteria. Compr Psychiatry 2000;41:159–162.
26. Wilfley DE, Wilson GT, Agras WS. The clinical significance of binge eating disorder. Int J Eat Disord 2003;34 (Suppl):S96– S106.
27. Hay PJ, Fairburn CG, Doll HA. The classification of bulimic eating disorders: A community-based cluster analysis study. Psychol Med 1996;26:801–812.
28. 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.
29. Fitzgibbon ML, Blackman LR. Binge eating disorder and bulimia nervosa: Differences in the quality and quantity of binge eating episodes. Int J Eat Disord 2000;27:238–243.
30. Dunn EC, Larimer ME, Neighbors C. Alcohol and drug-related negative consequences in college students with bulimia nervosa and binge eating disorder. Int J Eat Disord 2002;32:171– 178.
31. Fontenelle LF, Mendlowicz MV, Moreira RO, Appolinario JC. An empirical comparison of atypical bulimia nervosa and binge eating disorder. Braz J Med Biol Res 2005;38:1663–1667.
32. Telch CF, Stice E. Psychiatric comorbidity in women with binge eating disorder: Prevalence rates from a non-treatment-seeking sample. J Consult Clin Psychol 1998;66:768–776.
33. 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.
34. Dunkley DM, Grilo CM. Self-criticism, low self-esteem, depressive symptoms, and over-evaluation of shape and weight in binge eating disorder patients. Behav Res Ther 2007;45:139– 149.
35. Hilbert A, Saelens BE, Stein RI, Mockus DS, Welch RR, Matt GE, et al. Pretreatment and process predictors of outcome in interpersonal and cognitive behavioral psychotherapy for binge eating disorder. J Consult Clin Psychol 2007;75:645–651.
36. Pratt EM, Telch CF, Labouvie EW, Wilson GT, Agras WS. Perfectionism in women with binge eating disorder. Int J Eat Disord 2001;29:177–186.

37. Woodside DB, Bulik CM, Thornton L, Klump KL, Tozzi F, Fichter MM, et al. Personality in men with eating disorders. J Psychosom Res 2004;57:273–278.

38. Bulik CM, Sullivan PF, Joyce PR. Temperament, character and suicide attempts in anorexia nervosa, bulimia nervosa and major depression. Acta Psychiatr Scand 1999;100:27–32.

39. Bulik CM, Sullivan PF, Joyce PR, Carter FA. Temperament, character, and personality disorder in bulimia nervosa. J Nerv Ment Dis 1995;183:593–598.

40. Ribases M, Gratacos M, Badia A, Jimenez L, Solano R, Vallejo J, et al. Contribution of NTRK2 to the genetic susceptibility to anorexia nervosa, harm avoidance and minimum body mass index. Mol Psychiatry 2005;10:851–860.

41. Bulik CM. Exploring the gene-environment nexus in eating disorders. J Psychiatry Neurosci 2005;30:335–339.

42. Bulik CM, Sullivan PF, Kendler KS. Heritability of binge-eating and broadly defined bulimia nervosa. Biol Psychiatry 1998;44: 1210–1218.

43. Wade TD, Bulik CM, Sullivan PF, Neale MC, Kendler KS. The relation between risk factors for binge eating and bulimia nervosa: A population-based female twin study. Health Psychol 2000;19:115–123.

44. Bulik CM, Sullivan PF, Kendler KS. Genetic and environmental contributions to obesity and binge eating. Int J Eat Disord 2003;33:293–298.

45. Ramacciotti CE, Coli E, Passaglia C, Lacorte M, Pea E, Dell'Osso L. Binge eating disorder: Prevalence and psychopathological features in a clinical sample of obese people in Italy. Psychiatry Res 2000;94:131–138.

46. APA. DSM-IV: Diagnostic and Statistical Manual of Mental Disorders,4th ed. Washington, DC: American Psychiatric Association, 1994.

47. Garner DM, Garfinkel PE. The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. Psychol Med 1979;9:273– 279.

48. Garner DM, Inventario de Trastornos de la Conducta Alimentaria (EDI-2)-Manual. Madrid: TEA, 1998.

49. Henderson M, Freeman CP. A self-rating scale for bulimia. The 'BITE'. Br J Psychiatry 1987;150:18–24.

50. Derogatis L, SCL-90-R. A bibliography of research reports 1975– 1990. Baltimore, MD: Clinical Psychometric Research, 1990.

51. Cloninger CR. A systematic method for clinical description and classification of personality variants. A proposal. Arch Gen Psychiatry 1987;44:573–588.

52. Castro J, Toro J, Salamero M, Guimerá E. The Eating Attitudes Test: Validation of the spanish version. Evaluacion Psicologica/ Psychological Assessment 1991;7:175–190.

53. Garner DM, Eating Disorder Inventory-2. Odessa: Psychological Assessment Resources, 1991.

54. Rivas T, Bernabé R, Jiménez M. Fiabilidad y validez del test de investigación bulímica de Edimburgo (BITE) en una muestra de adolescentes españoles. Psicología Conductual 2004;12:447– 461.

55. Derogatis L, SCL-90-R. Cuestionario de 90 síntomas-Manual. Madrid: TEA Editorial, 2002.

56. Cloninger CR. The Temperament and Character Inventory-Revised. St Louis, MO: Center for Psychobiology of Personality, Washington University, 1999.

57. Cloninger CR, Svrakic DM, Przybeck TR. A psychobiological model of temperament and character. Arch Gen Psychiatry 1993;50:975–990.

58. Gutierrez F, Torrens M, Boget T, Martin-Santos R, Sangorrin J, Perez G, et al. Psychometric properties of the Temperament and Character Inventory (TCI) questionnaire in a Spanish psychiatric population. Acta Psychiatr Scand 2001; 103:143–147.

59. First M, Gibbon M, Spitzer R, Williams J, Users guide for the structured clinical interview for DSM IV Axis I disorders— Research version (SCID-I, version 2.0). New York: New York State Psychiatric Institute, 1996.

60. Fernandez-Aranda F, Turon V, Trastornos alimentarios. Guia basica de tratamiento en anorexia y

bulimia. Barcelona: Masson, 1998.

61. Stice E, Marti CN, Shaw H, Jaconis M. An 8-year longitudinal study of the natural history of threshold, subthreshold, and partial eating disorders from a community sample of adolescents. J Abnorm Psychol 2009;118:587–597.

62. Cooper Z, Fairburn CG. Refining the definition of binge eating disorder and non-purging bulimia nervosa. Int J Eat Disord 2003;34 (Suppl):S89–S95.

Table 1

	BED ($N = 34$) Mean (SD)	BN-NP ($N = 34$) Mean (SD)	BN-P ($N = 34$) Mean (SD)	F; df	Significance
Age of onset	27.4 (10.9)[a,b]	19.7 (8.9)	17.6 (4.7)	12.0; 2	<0.001
Duration of ED	6.9 (4.9)	6.1 (5.1)	6.7 (5.0)	0.2; 2	0.803
Weekly bingeing	6.7 (4.2)	7.6 (6.3)	7.7 (5.8)	0.3; 2	0.752
Current BMI	36.2 (4.9)[a,b]	26.5; 5.6	23.9; 6.0	45.1; 2	<0.001
Maximum BMI	37.7 (5.6)[a,b]	28.5; 5.6	27.2; 5.8	33.7; 2	<0.001
Minimum BMI	24.4 (4.0)[a,b]	19.9; 2.6	18.9; 3.1	25.5; 2	<0.001
	BED ($N = 34$) %	BN-NP ($N = 34$) %	BN-P ($N = 34$) %	Chi[b]; df	Significance
Impulsive behaviors	67.6%	66.7%	79.4%	1.7; 2	0.436
Substance abuse	15.2%	24.2%	29.4%	2.0; 2	0.374
Alcohol abuse	5.9%	12.1%	17.6%	2.3; 2	0.325
Current obesity[c]	90.9[1,2]	18.8	9.4	54.1; 2	<0.001
Lifetime obesity[c]	93.9[1,2]	32.3	24.2	38.1; 2	<0.001
Fam.Hist.[d] Obesity	54.5[1]	17.6	29.4	10.6; 2	0.005

Table 2

	BED (*N* = 34) (Mean; SD		BN-NP (*N* = 34) Mean; SD		BN-P (*N* = 34) Mean; SD		F; df	Signif.
EAT-40	34.1	10.6[a]	43.2	18.0	52.2	19.6	9.3; 2	<0.001
EDI-2								
Drive for thinness	12.5	4.6	14.4	4.4	14.6	5.8	1.7; 2	0.190
Body dissatisfaction	21.4	5.7	19.8	5.8	18.1	7.7	2.1; 2	0.127
Interoceptive awareness	10.5	6.6	11.2	6.1	12.1	5.9	0.5; 2	0.611
Bulimia	9.2	4.2	10.3	4.6	9.9	5.4	0.4; 2	0.648
Interpersonal distrust	5.2	4.1	5.4	4.3	5.1	4.3	0.0; 2	0.955
Inefficacy	10.1	6.4	10.5	6.1	11.5	6.7	0.5; 2	0.637
Maturation fears	6.1	4.3	7.9	5.0	8.0	4.9	1.6; 2	0.207
Perfectionism	4.4	3.7	4.6	4.0	6.5	3.7	3.0; 2	0.055
Impulsivity	4.4	3.4[a]	7.0	6.1	9.5	6.1	7.1; 2	0.001
Asceticism	6.5	3.1	7.7	3.8	8.0	4.3	1.3; 2	0.285
Social insecurity	6.7	4.3	7.0	5.1	8.2	4.3	0.9; 2	0.431
EDI-2 total	97.1	32.2	105.4	34.0	111.4	34.7	1.4; 2	0.241
BITE								
Symptoms	23.1	4.3	25.1	2.5	24.0	4.3	2.5; 2	0.085
Severity	7.3	3.4[a]	9.5	5.3	15.4	6.3	21.8; 2	<0.001
SCL-90-R								
Somatization	1.9	0.9	1.5	1.1	1.9	0.9	1.4; 2	0.251
Obsessive-compulsive	1.8	0.9	1.8	0.9	2.1	0.8	1.4; 2	0.244
Interpersonal sensitivity	1.8	1.0	1.9	0.9	2.3	0.8	3.1; 2	0.050
Depression	2.2	1.0	2.0	0.9	2.5	0.8	3.4; 2	0.038
Anxiety	1.5	0.8	1.5	0.8	1.9	0.8	3.9; 2	0.023
Hostility	1.2	0.9	1.4	0.9	1.7	0.9	3.5; 2	0.034
Phobic anxiety	0.7	0.7	1.0	0.8	1.3	0.9	3.5; 2	0.035
Paranoid ideation	1.1	0.9[a]	1.3	0.9	1.7	0.8	4.9; 2	0.009
Psychoticism	1.0	0.7[a]	1.2	0.7	1.6	0.8	5.3; 2	0.006
GSI	1.6	0.7	1.6	0.7	2.0	0.6	4.0; 2	0.021
PST	59.0	19.7[a]	58.6	18.0	70.2	14.1	4.8; 2	0.010
PSDI	2.3	0.6	2.3	0.5	2.5	0.5	2.0; 2	0.136
TCI-R								
Novelty seeking	100.8	16.1	103.3	17.2	104.6	13.1	0.5; 2	0.586
Harm avoidance	118.7	16.8	115.0	22.1	120.1	21.3	0.6; 2	0.570
Reward dependence	107.3	16.5	106.3	12.8	105.3	15.0	0.1; 2	0.866
Persistence	103.9	18.4	101.4	19.0	111.0	20.8	2.2; 2	0.115
Self-directedness	121.6	23.6	115.3	23.4	110.9	13.8	2.3; 2	0.108
Cooperativeness	137.1	14.4	137.7	18.6	132.9	15.7	0.9; 2	0.426
Self-transcendence	64.7	15.3	64.6	16.6	68.9	12.7	0.9; 2	0.403

Table 3

	BN-NP vs. BED			BN-P vs. BED		
	OR	95%CI	Significance	OR	95%CI	Significance
Lifetime obesity	0.032	0.005–0.187	<0.0005	0.016	0.002 to 0.121	<0.0005
BITE severity	1.201	1.014–1.422	0.034	1.417	1.179 to 1.702	<0.0005