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# The prevalence and correlates of binge eating disorder in the WHO World Mental Health Surveys

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### Abstract

**Background**—Little population-based data exist outside the United States on the epidemiology of binge eating disorder (BED). Cross-national data on BED are presented and compared to bulimia nervosa (BN) based on the WHO World Mental Health Surveys.

**Methods**—Community surveys with 24,124 respondents (ages 18+) across 14 mostly uppermiddle and high income countries assessed lifetime and 12-month DSM-IV mental disorders with the WHO Composite International Diagnostic Interview. Physical disorders were assessed with a chronic conditions checklist.

**Results**—Country-specific lifetime prevalence estimates are consistently (median; inter-quartile range) higher for BED (1.4%;0.8–1.9%) than BN (0.8%;0.4–1.0%). Median age-of-onset is in the late teens to early 20s for both disorders but slightly younger for BN. Persistence is slightly higher for BN (6.5 years; 2.2–15.4) than BED (4.3 years; 1.0–11.7). Lifetime risk of both disorders is elevated for women and recent cohorts. Retrospective reports suggest that comorbid anxiety, mood, and disruptive behavior disorders predict subsequent onset of BN somewhat more strongly than BED and that BN predicts subsequent comorbid psychiatric disorders somewhat more strongly than does BED. Significant comorbidities with physical conditions are due almost entirely to BN and BED predicting subsequent onset of these conditions, again with BN somewhat stronger than BED. Role impairments are similar for BN and BED. Fewer than half of lifetime BN or BED cases receive treatment.

**Conclusions**—BED represents a public health problem at least equal to BN. Low treatment rates highlight the clinical importance of questioning patients about eating problems even when not included among presenting complaints.

#### Keywords

binge eating disorder; epidemiology; WHO World Mental Health Surveys; bulimia nervosa; comorbidity; treatment

### INTRODUCTION

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) (1) recognizes only two specific eating disorders, anorexia nervosa (AN) and bulimia nervosa (BN). A provisional third diagnosis, binge eating disorder (BED), is characterized by recurrent-persistent episodes of uncontrolled binge eating with distress but without the inappropriate compensatory behaviors of BN. Based on growing evidence of comparatively high prevalence and clinical significance (2), a call has been made to include BED as a diagnosis in DSM-5 (3,4). However, most current BED epidemiological data come from Western samples (3,5,6). This report extends these data by examining the epidemiology of BED in 14 countries. To address concerns that BED may be insufficiently distinct from BN, we analyze BED and BN in tandem.

### METHODS

#### Samples

Data comes from the WHO World Mental Health (WMH) Survey Initiative (7). The 14 countries in WMH that assessed BED include one with a World Bank (8) classification as lower-middle income (Colombia), three upper-middle income (Brazil, Mexico, Romania), and ten high income (Belgium, France, Germany, Italy, the Netherlands, New Zealand, Northern Ireland, Portugal, Spain, and the United States). All surveys used adult household probability samples either nationally representative (all but three countries), representative of all urbanized areas (Colombia, Mexico), or representative of one urbanized area (São Paulo, Brazil). Sample sizes range from 466 (France) to 7,312 (New Zealand) and total 24,124. Response rates range from 45.9–87.7% and average 68.8%. More details about WMH samples are reported elsewhere (9).

Interviews had two parts. Part I, administered to all respondents, assessed core mental disorders (see below). All Part I respondents with any core disorder plus a probability subsample of other Part I respondents were then administered Part II, which assessed correlates and noncore disorders. Eating disorders were in Part II. The eating disorders sub-samples were weighted for under-sampling non-cases and for socio-demographic/geographic discrepancies with the population.

#### Measurement

**Interviews**—Interviews were administered face-to-face using consistent training and field quality control procedures (7,10,11). Informed consent procedures were approved by Institutional Review Boards of collaborating organizations. The instrument was the fully-structured lay-administered WHO Composite International Diagnostic Interview (CIDI) (12). CIDI translation, back-translation, and harmonization used standardized procedures (10).

**BN and BED**—The diagnostic algorithms and CIDI questions for DSM-IV BN and BED are presented in Supplement 1 (http://www.hcp.med.harvard.edu/ncs/ftpdir/ R896% 20APPENDIX% 20tables.pdf). Symptom questions closely parallel DSM-IV criteria with two exceptions. First, whereas DSM-IV BED requires six months of regular binge eating, the CIDI asked only about three months. However, this duration is consistent both with proposed DSM-5 criteria (3,13,14) and with DSM-IV BN criteria (15). Second, DSM-IV requires loss of control and distress regarding binges. Rather than address these symptoms directly, the CIDI questioned about attitudes and behaviors indicative of loss of control and distress (upset at out-of-control eating; feeling guilty, upset, or depressed after binging; continued eating after full; eating until uncomfortably full; eating alone because of embarrassment about volume eaten), introducing some imprecision into these assessments. DSM-IV diagnostic hierarchy rules do not allow BN or BED diagnoses in the presence of AN, or BED in the presence of BN. These hierarchy rules were implemented with retrospective age-of-onset (AOO) reports based on probing methods found to improve dating accuracy (16).

Respondents with lifetime BN or BED were asked if they ever received treatment for eating problems. Respondents with 12-month disorders were additionally asked about 12-month treatment and administered a modified version of the Sheehan Disability Scales (SDS) (17) to assess severity of recent eating problems. The modified SDS used a 0–10 visual analogue scale from *none* (0) to *very severe* (10) to characterize severity of impairment in each of four areas of living (work, home management, social life, close relationships). The SDS has

excellent internal consistency reliability (17–19) and good concordance with objective measures of role functioning (17–21). Height and weight were also assessed by self-report.

**Other DSM-IV disorders**—Fourteen other DSM-IV/CIDI disorders considered here include mood disorders (major depressive/dysthymia, bipolar I–II), anxiety disorders (panic disorder with/without agoraphobia, specific phobia, social phobia, generalized anxiety, post-traumatic stress, separation anxiety), disruptive behavior disorders (attention-deficit/ hyperactivity, oppositional-defiant, conduct, and intermittent explosive), and substance disorders (alcohol and drug abuse with or without dependence). Diagnostic hierarchy rules and organic exclusion rules were used in all diagnoses excepting oppositional-defiant disorder (diagnosed with/without conduct disorder) and substance abuse (diagnosed with/ without dependence). AOO was assessed using the same probing method as for eating disorders (16). A blinded clinical reappraisal study using the Structured Clinical Interview for DSM-IV (SCID) (22) in four WMH surveys found generally good concordance between CIDI and SCID diagnoses (23).

**Chronic physical conditions**—Comorbidities were also examined with 15 lifetime chronic physical conditions assessed using a checklist based on the US National Health Interview Survey (24,25). AOO was also assessed, allowing study of time-lagged associations with eating disorders. The checklist asked respondents whether they ever had a series of symptom-based conditions (e.g., chronic headaches) and whether a health professional ever said they had a series of silent conditions (e.g., hypertension). Such checklists yield more complete and accurate reports than reports from open-ended questions (26) and have good concordance with medical records (27–29).

**Treatment**—In addition to questions about treatment of eating disorders, respondents were asked if they saw a series of professionals "for problems with your emotions, nerves, or your use of alcohol or drugs" ever or in the past 12 months. These reports were not validated against treatment records. Treatment was divided into four sectors: specialty, general medical, human services, and complementary-alternative.

#### **Statistical Analyses**

Cross-tabulations were used to estimate prevalence, persistence, and treatment. The actuarial method (30) was used to generate AOO and persistence curves. Discrete-time survival analysis with a logistic link function and person-year the unit of analysis (31) was used to estimate predictors of onset and persistence. Survival coefficients and standard errors were exponentiated to create odds-ratios (ORs) with 95% confidence intervals. Design-based standard errors were estimated with the Taylor series method (30) using the SUDAAN software system (32). Multivariate significance was evaluated using Wald  $\chi^2$  tests based on design-corrected coefficient variance-covariance matrices. Statistical significance was consistently evaluated using two-sided .05-level tests.

## RESULTS

#### Prevalence

Lifetime prevalence estimates average 1.0% for BN and 1.9% for BED across surveys. Range and inter-quartile range (IQR; $25^{th}$ -75<sup>th</sup> percentiles) of lifetime prevalence estimates across surveys are 0.0–2.0% (0.4–1.0%) for BN and 0.2–4.7% (0.8–1.9%) for BED. (Table 1) Twelvemonth prevalence estimates (IQR) average 0.4% (0.1–0.3%) for BN and 0.8% (0.2–1.0%) for BED. Both lifetime and 12-month prevalence estimates are higher for BED than BN in virtually all countries.

#### Age-of-onset and persistence

Both mean and median AOO are consistently earlier for BN than BED (means: 20.6 vs. 23.3, t=3.4, p<.001; medians: 18.0 vs. 19.3). (Detailed results of these and other analyses not reported in tables are available at http://www.hcp.med.harvard.edu/ncs/ftpdir/ R896%20APPENDIX%20tables.pdf.) AOO curves show the slope to be less steep for BED than BN and narrower AOO IQR for BN (14.5–22.9 years of age) than BED (15.5–27.2), although country-specific AOO curves show considerable variability around these central tendencies.

Persistence is somewhat higher for BN than BED, with median (IQR) years in episode 6.5 (2.2–15.4) for BN and 4.3 (1.0–11.7) for BED. However, the mean (IQR) ratio of 12-month/ lifetime prevalence, an indirect measure of persistence, is lower for BN [37.3% (32.2–42.3)] than BED [44.3% (38.7–50.0)]. Cross-national differences in speed-of-recovery, as assessed in survival models predicting years in episode, suggest that BN recovery is most rapid in France and Portugal and least rapid in Italy and the Netherlands. (Table 2) The comparatively slow speed-of-recovery in Italy and Netherlands also applies to BED, with Colombia, France and Germany also having relatively slow BED speed-of-recovery.

#### Socio-demographic correlates

Multivariate survival models to predict lifetime risk were estimated only in the aggregate due to small within-country samples. (Table 3) Age-at-interview is inversely related to risk of both disorders ( $\chi^2_3$ =98.1–149.4, p<.001), with highest ORs for respondents ages 18–29 compared to ages 60+ (14.6–21.4), indicating either increased lifetime prevalence over time or differential recall and/or differential mortality. ORs are also consistently higher for females than males (2.4–3.6), students than non-students (1.6–3.5), and respondents with no college than some college (1.6–1.9). BN ORs are additionally elevated for some college (1.7) versus college graduates. Marital status is not significantly related to either disorder ( $\chi^2_2$ =1.6–1.9, p=.39–.68).

Parallel models were estimated to predict speed-of-recovery as defined by number of years with disorder. (Table 3) Age-at-interview is positively related to speed-of-recovery ( $\chi^2_3$ =67.2–88.4, p<.001), indicating either decreased persistence over time or differential recall and/or differential mortality. Odds of recovery of BED (0.6) but not BN (1.0) are inversely related to age-of-onset, suggesting that early-onset BED cases recover more quickly than later-onset cases. Speed-of-recovery is unrelated, though, to gender (0.8–1.2) or marital status, but is higher among students than non-students (2.9–3.0).

#### **Associations with Body-Mass Index**

Respondents with BN and BED have significantly higher current BMI than respondents without a history of eating disorder. (Table 4) Lower proportions of respondents with lifetime and 12-month BN and BED are underweight (0.5-1.5% vs. 2.8%, z=1.4-6.4, p=.16-(.001) or normal weight (25.0-33.7% vs. 47.3%, t=4.6-7.7, p<.001) and higher proportions obese (32.8-41.7% vs. 15.8%, t=4.5-7.5, p<.001) than those without lifetime eating disorders. No significant BN vs. BED differences exist in proportions underweight, normal weight, overweight, obese, or severely obese (t=0.3-1.0, p=.29-.78). Disaggregated results (reported in tables in Supplement 1) found equivalent patterns in the US, Latin America, Europe, and New Zealand.

#### Comorbidity with other mental disorders

The vast majority of respondents with lifetime BN (84.8%) and BED (79.0%) meet lifetime criteria for other DSM-IV/CIDI disorders. (Detailed results reported in tables in Supplement 1.) ORs of BN and BED with each of the other disorders are positive and significant (BN

Median=4.3, IQR=3.5–4.6; BED Median=2.8, IQR=2.7–3.3). Retrospective AOO reports were used to estimate reciprocal survival models of (i) temporally primary eating disorders predicting subsequent first onset of other mental disorders and (ii) temporally primary other mental disorders predicting subsequent first onset of eating disorders. The time-lagged ORs were mostly elevated and significant in both directions, but generally higher for other disorders predicting eating disorders than the reverse. Median (IQR) ORs of BN predicting other disorders were generally higher than those of BED predicting the same disorders (BN 3.3[2.8–4.4]) vs. BED (2.3[2.0–2.7]). ORs in reciprocal models showed that other disorders predicted subsequent first onset of BN somewhat more powerfully than first onset of BED (BN 5.1[3.8–6.0] vs. BED 3.4[3.3–3.9]).

#### Comorbidity with chronic physical conditions

Lifetime BN and BED have significant cross-sectional associations with numerous physical conditions. Reciprocal survival models documented generally insignificant associations between temporally primary physical conditions and subsequent onset of BN and BED, but significant associations between temporally primary BN-BED and subsequent onset of some physical conditions. (Table 5) ORs of BN and BED are generally comparable in predicting musculoskeletal conditions (1.6–2.8), other pain conditions (1.9–2.8), diabetes (2.9–3.1), hypertension (2.2–2.2), and ulcers (1.8–1.9). BN is associated with subsequent heart attack (4.1) and stroke (3.3), but BED is not. Neither BN nor BED is associated with subsequent respiratory disorders (0.3–2.2). The introduction of other temporally primary (to the onset of the outcome condition) DSM-IV/CIDI disorders as controls reduces the associations of BN and BED with these outcomes. However, a number of adjusted associations of BN and BED remain significant, including BN and BED predicting chronic back/neck pain (1.5–1.7), other chronic pain conditions (1.5–1.8), diabetes (2.4), and hypertension (1.6–1.8), and of BED predicting chronic headaches (1.8).

#### Impairment in role functioning

Role impairment due to eating disorder was reported by 54.5% of respondents with 12month BN and 46.7% with 12-month BED. (Detailed results reported in tables in Supplement 1.) Severe impairment was reported by 21.8% of respondents with 12-month BN and 13.2% with 12-month BED. The only significant BN vs. BED difference was more severe impairment in personal relationships for BN than BED (12.9% vs. 5.4%, t=2.1, p=. 038).

#### Treatment

The majority of respondents with lifetime BN (67.3%) and BED (57.7%) received lifetime treatment for emotional problems. (Detailed results reported in tables in Supplement 1.) The most common sector was mental health specialty (43.3–57.7%) followed by general medical (34.3–43.2%), with lower proportions in the human services (10.3–14.3%) and complimentary-alternative medicine (15.0–18.8%) sectors. Twelve-month treatment among 12-month cases was lower (BN 45.0%, BED 36.6%), most common in the general medical sector (24.9–31.0%), less in the mental health specialty sector (17.6–25.2%), and least in the human services and complementary-alternative medicine sectors (5.5–7.4%).

Smaller proportions of lifetime cases (BN 47.4%, BED 38.3%) ever received treatment specifically for eating disorders, representing roughly two-thirds of those who received lifetime treatment for emotional problems. About one-fourth (25.9%) of respondents with 12-month BN received 12-month treatment for their eating disorder, representing 57.6% of those who received 12-month treatment for any emotional problems (i.e., 25.9/45.0=57.6%). An even smaller proportion (9.8%) of respondents with 12-month BED received 12-month

treatment for their eating disorder, representing 26.8% of those who received any 12-month treatment for emotional problems (i.e., 9.8/36.6=26.8%). Survival analysis showed that women are significantly more likely than men to obtain treatment for BN (OR=1.5) and BED (1.6), but no other significant socio-demographic correlates of treatment were found.

### DISCUSSION

Results must be interpreted in the context of several study limitations. Response rates varied considerably across surveys. Sample sizes were too small to produce country-specific results other than for estimates of prevalence, AOO, and persistence. Because of confounding between country and socio-demographic variables, it was impossible to explore potentially important relationships of eating disorders with race/ethnicity or socio-economic status (2,33) excepting aggregate associations with education. Lifetime disorders were assessed retrospectively using fully-structured rather than semi-structured interviews, probably leading to under-estimation of lifetime prevalence and over-estimation of AOO and of the effects of age-at-interview and comorbidity. Although an earlier version of CIDI under-diagnosed eating disorders (34), the more recent version of CIDI used here modified probes to address this problem (12). Nonetheless, the absence of CIDI validation studies is a limitation. Prevalence estimates might be considered lower bounds because of the above limitations, although the three-month rather than six-month duration requirement in the CIDI is anti-conservative.

Notwithstanding these limitations, the data reported here provide the first population-based estimates of the prevalence and correlates of BED among adults across a diverse set of countries. These results extend findings from previous epidemiological research, which has largely concentrated on females (35–43), adolescents (see 2,6 for reviews), and particular races or ethnic groups (41,42,44–47). Indeed, apart from the DSM field trial, which established a population prevalence of 2.0% for BED (48) and some seminal but now dated Western studies (49–52), few epidemiological studies describe eating disorders in broadly representative samples of men and women. Although one recent regional Italian study examined adult mixed-gender rates (53), the overly restrictive screener in that study undermined the representativeness of the tiny resultant sample of 32 cases. To our knowledge, other contemporary mixed-gender surveys are limited to WMH sub-samples in Europe and the US, with estimates between 0.5% to 1.0% for BN and 1.1% to 2.6% for BED (54,55), consistent with the WMH pooled estimates reported here of 1.0% for BN and 1.9% for BED.

Data on eating disorder AOO are also not well established given methodological heterogeneity of studies (4), although the WMH finding of relatively early AOO is broadly consistent with previous research. Our finding of somewhat earlier AOO for BN than BED is also consistent with previous studies of DSM-defined eating *disorders* (43,52,56–60), but differs from more general studies of eating *behaviors*, which tend to show binging behaviors peaking before compensatory behaviors (36,61,62). The wider AOO range found here for BED than BN is consistent with previous large-scale population-based studies (55,63), possibly reflecting diagnostic heterogeneity of BED (43,57,64–67).

The WMH finding of relatively low persistence of BED and BN is consistent with most epidemiologic research (55,68–70), but clinical samples show higher persistence. Our somewhat lower persistence of BED than BN is consistent with previous population studies (36,54), but contrasts with results in two non-representative samples (43,57). It is noteworthy in this regard that lower persistence of BED than BN was also found in several longitudinal clinical studies that found lower remission for BN than BED (71,72), higher

relapse for BN than BED (62), and higher diagnostic crossover from BED to BN than the reverse (13,72,73).

The finding that BN and BED are more prevalent among females than males is consistent with much previous research (54,55,74,75). The WMH gender differences are smaller, though, than in treatment studies (76,77), presumably reflecting the lower help-seeking rates of men than women found both here and elsewhere (78). The higher proportion of males found here in BED than BN (i.e., binge-eating males less likely to use compensatory behaviors than binge-eating females) is consistent with previous findings (79,80), further supporting the clinical differentiation of BED from BN.

The WMH finding that students have elevated risk of eating disorders is indirectly consistent with the widely accepted notion that peer norms shape maladaptive eating behaviors (81–83). Our finding that marital status is unrelated to eating disorders also accords with previous studies (6,36,55,77), as does our finding that BN and BED are inversely related to education (84,85). The finding that age-at-interview is inversely related to lifetime risk is consistent with previous evidence suggesting that BN (74,77,86) and BED (54,74,87) have increased in prevalence over time, although the fact that our ORs for age-at-interview are highest for the most recent ages is inconsistent with other data suggesting a leveling of risk in recent years (88).

Our finding that BN and BED are associated with equivalently high rates of current overweight and obesity is consistent with some (89–91), but not all (36,38,52,60), previous community-based surveys. All of these other studies found high rates of obesity in BED but only some also found high rates of obesity in BN. The exception is a pair of national trend surveys in Norway, where equivalently elevated rates of obesity were found for BN and BED in the first survey (1991) but only for BN (i.e., not for BED) in the second survey (2004) (91). It is striking in light of these diverse results that the WMH finding of equivalently high BMI in BN and BED was found consistently in surveys in the US, Latin America, Western Europe, and New Zealand. It is noteworthy in light of this consistency that none of the previous community surveys other than the two in Norway was based on a nationally representative mixed-gender sample, raising the possibility that the inconsistent association across segments of the population that were differentially sampled in these surveys. Future studies should include systematic investigations of such possible specifications.

A complicating factor in such future studies is the possibility of time trends. As noted above, the Norwegian trend studies showed that BN vs. BED differences in obesity changed in that country in the years between those two surveys. In addition, while clinical studies have traditionally found high obesity rates among patients with BED but not BN (58,92), recent studies have begun to find rising obesity rates also among patients with BN (93,94). This might reflect changing patterns of differential selection into treatment on the basis of BMI among community cases with BN versus BED. Another possibility is that age patterns of BN prevalence and/or help-seeking are changing, as the association of BN with obesity is stronger among older cases (95). Future studies of these possibilities, while well beyond the scope of the current report, are clearly warranted in light of inconsistences across community and clinical studies in the associations of BN and BED with obesity.

The high comorbidity of BN and BED with other DSM-IV/CIDI disorders is broadly consistent with previous studies of both BN (49,50,86,96) and BED (97–99). However, only a few of these previous studies examined associations between early-onset mental disorders and subsequent eating disorders (100–102) or between early-onset eating disorders and other

subsequent mental disorders (103). The WMH results are consistent with those few studies in finding significant reciprocal bivariate time-lagged associations, although our multivariate analysis did not find the dominance of major depression as the primary predictor of eating disorders suggested in one previous study (101).

Although we know of no previous study of the relative sizes of cross-lagged ORs between eating disorders and other mental disorders, one cross-lagged evaluation of eating behaviors and substance-use problems revealed no concurrent or temporal associations between binge eating symptoms and alcohol or illicit substance use (104). In addition, two separate papers from one prospective study (101,103) found other mental disorders predicting eating disorders somewhat more strongly than vice-versa. To the extent that this asymmetry reflects causal influences of other mental disorders on subsequent eating disorders, a question can be raised whether successful early intervention to treat other disorders would help prevent subsequent eating disorders. This question is of considerable public health importance and deserves empirical investigation (105).

Despite a growing clinical literature on adverse effects of eating disorder on physical health (106,107), few epidemiological studies have examined comorbidity between eating disorders and physical disorders. Existing cross-sectional studies are consistent with the WMH results in documenting significant associations between eating disorders and numerous chronic physical disorders (108–111). The few prospective epidemiological studies that examine time-lagged associations of this sort are consistent with WMH in finding positive associations between eating disorders and subsequent cardio-metabolic disorders and chronic pain conditions. Previous studies differ from WMH, though, in finding insignificant associations with later respiratory disorders (103,112,113). Although it is unclear whether these significant time-lagged associations are causal, clinical studies document plausible biological pathways that are consistent with a causal interpretation (114,115).

The possibility of significant adverse effects of eating disorders with subsequent physical health problems contrasts with the finding that current role impairments of BN and BED are quite modest. As shown in a previous WMH report (19), the proportions of WMH respondents reporting severe impairment associated with a wide variety of other 12-month anxiety, mood, and disruptive behavior disorders range between 18.6% (specific phobia) and 68.3% (bipolar disorder), with a mean across disorders of 41.3%. The 13.2% rate of severe impairment associated with 12-month BED is well below that range, and the 21.8% rate associated with 12-month BN is near the low end of that range. This might reflect the well-known lack of insight and tendency of people with eating disorders to minimize their symptom severity (116,117).

The finding that low proportions of people with eating disorders receive treatment is broadly consistent with previous reports from component WMH surveys in the US (54) and Western Europe (36,55). These are lower treatment rates than for many other mental disorders found in the WMH surveys (118). Consistent with previous research on health service utilization in eating disorders (119), a substantial proportion of WMH eating disorder cases who received treatment were treated for comorbid emotional problems rather than for their eating disorders. This finding, coupled with the observation that physicians seldom assess and often fail to recognize eating disorders (120,121), highlights the importance of clinicians questioning about eating problems even when patients do not include them among presenting complaints.

The WMH findings provide strong evidence for the clinical and public health importance of BED. In contrast to some earlier studies in young women suggesting that BED might be

relatively transient (35,36), we find that BED is more common than BN, nearly as persistent and comorbid as BN, and roughly comparable in impairment to BN across a wide range of countries. Combined with broadly similar evidence from other studies (4), WMH findings suggest that BED represents a public health problem at least equal to that of BN. BED is also distinguishable from BN by higher prevalence, lower persistence, and distinctive patterns of comorbidity and recovery. This, taken together with evidence of its clinical significance, supports the case for elevating binge eating disorder from a provisional entity to an official diagnosis in DSM-5 (122).

### Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Lifetime and 12-month prevalence of DSM-IV/CIDI bulimia nervosa (BN) and binge eating disorder (BED) in the WMH surveys

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		BN	B	ED	1	17-11 3N	aonth B	ED	
	%	(se)	%	(se)	%	(se)	%	(se)	I(u)
I. Lower-middle incor	l me								
Colombia	0.4	(0.1)	0.9	(0.2)	0.2	(0.1)	0.3	(0.1)	(1,217)
II. Upper-middle inco	me								
Brazil (São Paulo)	2.0	(0.2)	4.7	(0.3)	0.9	(0.2)	1.8	(0.3)	(2,942)
Mexico	0.8	3 (0.2)	1.6	(0.4)	0.3	(0.1)	0.5	(0.2)	(1,236)
Romania	0.0	(0.0)	0.2	(0.0)	0.0	(0.0)	0.1	(0.0)	(2,357)
III. High income									
Belgium	1.6	) (0.5)	1.2	(0.4)	0.3	(0.3)	0.7	(0.4)	(518)
France	0.7	(0.4)	1.7	(0.8)	0.2	(0.2)	0.3	(0.2)	(466)
Germany	0.3	(0.1)	0.5	(0.2)	0.2	(0.1)	0.1	(0.1)	(658)
Italy	0.1	(0.1)	0.7	(0.3)	0.0	(0.0)	0.2	(0.1)	(000)
Netherlands	0.5	(0.5)	0.9	(0.5)	0.1	(0.1)	0.1	(0.1)	(540)
New Zealand	1.3	3 (0.1)	1.9	(0.2)	0.5	(0.1)	1.0	(0.1)	(7,312)
Northern Ireland	0.5	(0.1)	1.5	(0.3)	0.2	(0.1)	0.8	(0.3)	(1,432)
Portugal	0.8	(0.3)	2.4	(0.6)	0.5	(0.2)	1.1	(0.4)	(509)
Spain	0.7	(0.4)	0.8	(0.3)	0.1	(0.1)	0.5	(0.3)	(1,057)
NS	1.0	(0.2)	$2.6^{2}$	(0.3)	0.3	(0.1)	1.2	(0.2)	(2,980)
IV. Total									
Total	1.0	(0.1)	1.9	(0.1)	0.4	(0.1)	0.8	(0.1)	(24,124)
I Sample sizes reported	repre	sent the t	total nun	uber of I	people 1	who wei	re asses	sed for p	sresence of BN and BED in the survey, not the number of people who met criteria for eating disorders.
2									
An earlier paper on the	e US	survey in	correctl	y reporte	ed this :	as 2.8%	rather	than 2.6	% (Hudson JI, Hiripi E., Pope HG, et al., 2007. Prevalence and Correlates of Eating Disorders in the Nation

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Comorbidity Study Replication. Biological Psychiatry, 61(3), 348–358) due to prevalence of BED but not BN being defined without diagnostic hierarchy exclusions for Anorexia Nervosa. Corrected results for BED from that paper are now posted at http://www.hcp.med.harvard.edu/ncs/ftpdir/PA368%20appendix%20tables.pdf.

Cross-national differences in speed-of-recovery of DSM-IV/CIDI bulimia nervosa (BN) and binge eating disorder (BED) in the WMH surveys  $^{I}$ 

		BN		BED
	OR	(95% CI)	OR	(95% CI)
I. Lower-middle incom	ne			
Colombia	1.1	(0.7–2.0)	0.6*	(0.4–0.9)
II. Upper-middle incom	ne			
Brazil (São Paulo)	1.8	(1.0–3.2)	0.7	(0.4–1.1)
Mexico	1.0	(0.5–1.9)	0.7	(0.4–1.2)
Romania		2	1.2	(0.6–2.2)
III. High income				
Belgium	1.2	(0.8–2.0)	1.2	(0.6–2.2)
France	3.1*	(1.9–5.0)	0.5*	(0.2–0.9)
Germany	1.9	(0.7–5.7)	0.3*	(0.2–0.5)
Italy	0.4*	(0.3–0.7)	0.6*	(0.4–0.8)
Netherlands	0.3*	(0.2–0.4)	0.4*	(0.3–0.7)
New Zealand	0.8	(0.6–1.2)	1.0	(0.7–1.5)
Northern Ireland	1.1	(0.7–1.7)	1.0	(0.7–1.4)
Portugal	2.4*	(1.6–3.6)	0.8	(0.4–1.6)
Spain	0.8	(0.5–1.2)	1.0	(0.3–3.1)
(n)		(457)		(722)

Significantly different from the US at the .05 level, two-sided test

<sup>1</sup>Based on a multivariate discrete-time survival model to predict number of years with the disorder in the subsample of respondents with a lifeitme history of the disorder. Coefficients represent the relative-odds of continuation of the disorder one additional year in the country indicated in the row heading compared to the US. Controls were included in the model for age-at-interview, age-of-onset, gender, education, student status, and marital status. Education, and student status were time-varying covariates.

<sup>2</sup>The coefficient for Romania could not be estimated due to sparse data.

Socio-demographic correlates of lifetime onset and speed-of-recovery of DSM-IV/CIDI bulimia nervosa (BN) and binge eating disorder (BED) in the WMH surveys<sup>1</sup>

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		Ons	et			Speed of	recovei	y
		BN	_	BED		BN		BED
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
Age-at-interview								
18–29	$21.4^{*}$	(11.5 - 39.6)	14.6	(9.1 - 23.6)	$0.0^{*}$	(0.0 - 0.2)	$0.1^{*}$	(0.1 - 0.2)
30-44	$9.9^*$	(5.6–17.5)	$6.6^*$	(4.4 - 10.1)	$0.1^*$	(0.0-0.4)	0.2	(0.1 - 0.4)
45-59	7.9*	(4.3 - 14.6)	$2.6^*$	(1.8–3.8)	$0.2$ $^{*}$	(0.0-0.8)	0.6	(0.3 - 1.0)
60+	1.0	ł	1.0	1	1.0	I	1.0	I
$\chi^{2}{}_{3}$		98.1*	÷	49.4 *		67.2 <sup>*</sup>		88.4
Age-of-onset (Decade) <sup>2</sup>	0							
A00	ł		ł		1.0	(0.9 - 1.0)	$0.6^*$	(0.5 - 0.7)
Gender								
Female	3.6	(2.6-4.8)	2.4*	(1.9–3.2)	0.8	(0.6 - 1.1)	1.2	(0.9 - 1.6)
Male	1.0	ł	1.0	ł	1.0	I	1.0	I
Education (Completed t	o date)							
< Secondary	1.8	(1.2–2.9)	$1.7$ $^{*}$	(1.2 - 2.3)	$1.6^*$	(1.2–2.3)	1.3	(0.9-1.9)
Secondary	$1.9^*$	(1.1–3.1)	$1.6^*$	(1.2 - 2.3)	$1.7^{*}$	(1.2–2.5)	$1.5$ $^{*}$	(1.0–2.2)
Some college	$1.7$ $^*$	(1.0–2.9)	1.2	(0.8-1.9)	$2.0^*$	(1.3–3.1)	1.3	(0.9 - 1.8)
College graduate	1.0	1	1.0	ł	1.0	I	1.0	I
$\chi^{2}{}_{3}$		8.3 *	-	11.0*		12.9*		$13.8^{*}$
Currently a student (Ye	(oN/s							
Student	3.5*	(2.3–5.5)	$1.6^*$	(1.0-2.4)	$3.0^*$	(1.9–4.7)	$2.9^*$	(1.6–5.2)
Marital status								
Never married	1.1	(0.7 - 1.8)	1.2	(0.9-1.7)	0.8	(0.6 - 1.1)	0.8	(0.6 - 1.0)
Previously married	1.4	(0.8-2.2)	1.1	(0.8-1.6)	1.1	(0.7 - 1.7)	0.7	(0.5 - 1.1)
Currently married	1.0	:	1.0	;	1.0	I	1.0	I

		Ons	et			Speed of 1	recover	y
		BN		BED		BN		BED
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
$\chi^{2}_{2}$		1.6		1.9		2.7		4.8
(u)	0	24,124)	(2	4,124)		(457)		(722)
				-				

Significant at the .05 level, two-sided test

/Based on a multivariate discrete-time survival model to predict either first onset of each lifetime disorder in the total sample or persistence of the disorder in the subsample of respondents with the disorder. additional year. Results are pooled across countries due to the instability of coefficients within individual countries. Education, student status, and marital status are time-varying covariates. Dummy Coefficients in the onset model represent the relative-odds of onset of the disorder, while coefficients in the speed-of-recovery model represent the relative-odds of continuation of the disorder one predictors are included to control for between-country differences in overall risk.

<sup>2</sup>Due to the small changes in relative-odds associated with differences in age-of-onset (AOO) of only one year, results are expressed here for differences over ten years (i.e., the tenth power of the OR associated with AOO predicting number of years with the disorder).

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Current body mass index (BMI) among respondents with versus without DSM-IV/CIDI bulimia nervosa (BN) and binge eating disorder (BED) in the WMH surveys

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		B	z			DE	2		INO CALILI	ion inem Bi
	Ľ	fetime	12-	·month	Ľ	fetime	12	-month		
	%	(se)	%	(se)	%	(se)	%	(se)	%	(se)
I. Distribution of BMI										
< 18.5 (underweight)	1.1	(0.4)	$0.5$ $^{*}$	(0.3)	1.3	(0.6)	1.5	(6.0)	2.8	(0.2)
18.5-24.9 (normal)	33.7*	(2.7)	26.6	(4.1)	31.7*	(2.8)	25.0	(2.9)	47.3	(0.5)
25 - 29.9 (overweight)	32.4	(2.9)	34.9	(4.9)	30.7	(2.5)	31.8	(3.8)	34.1	(0.4)
30 – 34.9 (obese class I)	19.7*	(2.5)	22.8 <sup>*</sup>	(4.5)	$23.1^{*}$	(2.5)	25.2	(3.0)	11.2	(0.3)
35 – 39.9 (obese class II)	8.5*	(1.5)	9.7*	(2.6)	7.3*	(1.1)	8.8	(1.7)	3.3	(0.2)
40+ (obese class III)	4.5*	(1.1)	5.6	(2.3)	$5.8^*$	(1.1)	7.6	(1.9)	1.3	(0.1)
35+ (obese class II–III)	$13.0^*$	(1.8)	$15.3^{*}$	(3.2)	$13.1^{*}$	(1.5)	16.4	(2.4)	4.6	(0.2)
30+ (total obese)	32.8*	(2.7)	$38.1^{*}$	(4.9)	36.2	(2.7)	41.7	(3.7)	15.8	(0.4)
II. Logistic regression of BN	or BED	vs. No lifetim	ie eating	disorder) on E	IMI <sup>1</sup>					
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)		
< 18.5 (underweight)	0.6	(0.2 - 1.3)	0.3	(0.1 - 1.1)	0.7	(0.3-1.8)	1.0	(0.3 - 3.5)		
18.5-24.9 (normal)	1.0	I	1.0	I	1.0	1	1.0	1		
25 - 29.9 (overweight)	1.3	(1.0-1.8)	1.8	(1.1 - 3.0)	1.3	(1.0-1.8)	1.7	(1.2–2.5)		
30 – 34.9 (obese class I)	2.4 *	(1.7 - 3.5)	3.8*	(2.0–6.9)	3.1 *	(2.2–4.3)	4.1	(2.8-6.0)		
35 – 39.9 (obese class II)	$3.2$ $^{*}$	(2.1–5.1)	$5.0^*$	(2.6–9.6)	$3.0^*$	(2.0-4.6)	4.5*	(2.6–7.5)		
40+ (obese class III)	4.7 *	(2.7–8.2)	8.3*	(3.3 - 21.1)	$6.6^*$	(4.2 - 10.2)	$10.2^{*}$	(5.8 - 18.1)		
$\chi^2{}_5$	••	58.9*	4	16.1 <sup>*</sup>	1	$05.9 ^{*}$		)4.2 *		
(u)	-	(457)	Ŭ	(158)		722)		(344)	(22	.,949)

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I Based on a series of pooled (across countries) logistic regression models, each one comparing respondents with the eating disorder defined by the column heading with respondents without a history of

eating disorders controlling for dummy variables for country

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Survival analysis predicting subsequent onset of chronic physical conditions from temporally primary DSM-IV/CIDI bulimia nervosa (BN) and binge eating disorder (BED) in the WMH surveys  $(n = 21, 183)^{I}$ 

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		BN				BE	Q	
	Without contro CID1	ols for other DSM-IV/ [ disorders	With controls CIDI	for other DSM-IV/ disorders	Without control CIDI	s for other DSM-IV/ disorders	With controls CIDI	for other DSM-IV/ disorders
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
I. Musculoskeletal								
Arthritis	$1.6^*$	(1.1-2.3)	1.1	(0.7 - 1.5)	1.7 *	(1.3 - 2.2)	1.3	(1.0-1.6)
Chronic back/neck pain	2.8*	(2.1 - 3.6)	$1.7^{*}$	(1.3-2.2)	$2.0^*$	(1.5–2.7)	1.5 *	(1.1-2.0)
II. Other pain conditions								
Chronic headaches	$1.9$ $^{*}$	(1.4–2.7)	1.2	(0.9 - 1.7)	$2.3^{*}$	(1.7–3.3)	$1.8^{*}$	(1.3–2.5)
Other chronic pain	2.8*	(1.9-4.1)	$1.5^{*}$	(1.1-2.2)	2.7 *	(1.8-4.0)	$1.8^*$	(1.2–2.7)
III. Respiratory								
Asthma	0.8	(0.5-1.4)	0.8	(0.4 - 1.3)	1.4	(0.9-2.2)	1.3	(0.9-2.0)
Seasonal allergies	0.9	(0.6-1.3)	0.8	(0.5-1.1)	1.2	(0.8 - 1.7)	1.1	(0.8-1.5)
Tuberculosis	0.3	(0.0-2.3)	0.2	(0.0-1.4)	2.2	(0.6 - 8.2)	1.6	(0.4-5.9)
Other (e.g., COPD)	0.8	(0.2-2.8)	0.5	(0.1 - 1.7)	1.0	(0.4-2.5)	0.7	(0.3 - 1.6)
IV. Cardio-metabolic								
Diabetes	$3.1^{*}$	(2.0-4.8)	2.4*	(1.5 - 3.8)	$2.9^{*}$	(1.9–4.6)	2.4 *	(1.5–3.7)
Hypertension	$2.2^{*}$	(1.6–3.1)	$1.6^*$	(1.1–2.3)	$2.2^{*}$	(1.7 - 3.0)	$1.8^{*}$	(1.3–2.4)
Heart attack	$4.1^*$	(1.7 - 10.1)	2.5	(0.9 - 7.0)	0.0	(0.3 - 3.1)	0.7	(0.2 - 2.5)
Heart disease	1.1	(0.5-2.4)	0.7	(0.3-1.5)	1.3	(0.6-2.5)	0.9	(0.5-1.8)
Stroke	3.3 *	(1.2 - 9.0)	1.9	(0.7 - 5.2)	1.6	(0.7 - 3.5)	1.2	(0.5-2.5)
V. Other								
Cancer	1.8	(1.0-3.4)	1.3	(0.6-2.6)	1.8	(1.0-3.3)	1.5	(0.8-2.8)
Ulcer	1.8	(0.9 - 3.5)	1.0	(0.5 - 1.9)	$1.9^{*}$	(1.2 - 3.0)	1.2	(0.7 - 1.9)
* Significant at the .05 level, tv	vo-sided test							

temporally primary DSM-IV/CIDI mental disorders included as predictors. Results are pooled across 13 countries and 12 dummy predictors are included to control for between-country differences along with controls for the socio-demographic variables in Table 3. São Paulo was excluded from the analysis because AOO of the physical conditions was not dated in the São Paulo survey. /Based on multivariate discrete-time survival models to predict first onset of the physical conditions from temporally primary lifetime eating disorders without and with dummy variablers for other