Binge-Eating Disorder in the Swedish National Registers: Somatic Comorbidity

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ABSTRACT

Objective: To evaluate associations between binge-eating disorder (BED) and somatic illnesses and determine whether medical comorbidities are more common in individuals who present with BED and comorbid obesity.

Method: Cases ($n = 850$) were individuals with a BED diagnosis in the Swedish eating disorders quality registers. Ten community controls were matched to each case on sex, and year, month, and county of birth. Associations of BED status with neurologic, immune, respiratory, gastrointestinal, skin, musculoskeletal, genitourinary, circulatory, and endocrine system diseases were evaluated using conditional logistic regression models. We further examined these associations by adjusting for lifetime psychiatric comorbidity. Amongst individuals with BED, we explored whether comorbid obesity was associated with risk of somatic disorders.

Results: BED was associated with most classes of diseases evaluated; strongest associations were with diabetes [odds ratio (95% confidence interval) = 5.7 (3.8; 8.7)] and circulatory systems [1.9 (1.3; 2.7)], likely indexing components of metabolic syndrome. Amongst individuals with BED, those with comorbid obesity were more likely to have a lifetime history of respiratory [1.5 (1.1; 2.1)] and gastrointestinal [2.6 (1.7; 4.1)] diseases than those without comorbid obesity. Increased risk of some somatic disease classes in individuals with BED was not simply due to obesity or other lifetime psychiatric comorbidity.

Discussion: The association of BED with many somatic illnesses highlights the morbidity experienced by individuals with BED. Clinicians treating patients with BED should be vigilant for medical comorbidities. Nonpsychiatric providers may be the first clinical contact for those with BED underscoring the importance of screening in primary care. © 2016 The Authors International Journal of Eating Disorders Published by Wiley Periodicals, Inc.

Keywords: binge-eating disorder; gastrointestinal; cardiovascular; physical; metabolic syndrome; somatic; medical comorbidity

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Introduction

Binge-eating disorder (BED), included in the Diagnostic and Statistical Manual of Mental Disorders 5th Edition (DSM-5[1]), has a reported lifetime prevalence of 0.1–3.6%, and a point prevalence of 0.1– 5.6%.[2–6] The ratio of lifetime BED in women to men is about 1.3–3 to 1.[3,5] BED has been reported to be associated with increased risk for a range of medical comorbidities, some of which may be independent of overweight and obesity. For example, BED and binge-eating behaviors have been associated

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with increased risk of hypertension,[4,7] type II diabetes,[4,8–10] autoimmune disease,[11] and gastrointestinal disorders.[4,12–14] Pain syndromes (e.g., headaches, neck and back pain, arthritis[4,15]; limb and joint pain[13]; and fibromyalgia[12]) have also been reported to commonly co-occur in individuals with BED.

Results of the aforementioned studies have been somewhat inconsistent (see Refs. 12,16,17) and have been limited by single site clinical sampling, a focus on the presence of binge-eating behavior rather than threshold BED, or an inability to compare the somatic comorbidities in individuals with BED with and without comorbid obesity.[14,15,18,19] Moreover, other research has suggested an association between obesity and dermatological, respiratory, immune conditions[20] and metabolic syndrome. Immune problems also increase vulnerability to infectious diseases.[21] Additional evidence is required to clarify whether BED is associated with these conditions and whether any of the observed associations are partly or fully accounted for by the presence of obesity.

Determining the extent to which BED is associated with adverse health outcomes is critical for estimating the burden of disease and for service planning. Register-based studies that capture all activity in a nation's health care system allow for a complete evaluation of patterns of comorbidity in BED. Using the Swedish national population registers, the primary aim of the present study was to evaluate the lifetime associations between BED and illnesses of the neurologic, infectious/parasitic, immune, respiratory, gastrointestinal, skin, musculoskeletal, genitourinary, circulatory, endocrine systems, congenital malformations, and external causes of morbidity (i.e., injury). A secondary aim was to compare the prevalence of the observed somatic comorbidities in individuals with BED with and without comorbid obesity. We hypothesized that there would be positive lifetime associations between BED and gastrointestinal,[4,12–14] musculoskeletal,[4] circulatory,[4,7] and endocrine disorders,[4,8–10] but the other tests of association were exploratory.

Method

Procedure

Data were extracted from Swedish population registers in 2009. Unique personal identification numbers assigned to all Swedish residents enables linkage across the population registers. For this analysis, we linked (a) the eating disorders national quality registers, National Quality Register for Eating Disorders Treatment (Riksät)[22] and Stepwise,[23] which began entering patient information in 1999

and 2005, respectively; (b) the National Patient Register (NPR[24]), covering all Swedish public and private hospital inpatient admissions from 1973 onwards and outpatient specialist care from 2001 onwards; (c) the Multi-Generation Register,[25] to determine biological and adoptive relationships for all individuals living in Sweden since 1933; (d) the Migration Register,[26] containing information on emigration from or immigration to Sweden; and (e) the Cause of Death Register,[27] listing date and primary and contributing cause(s) of deaths since 1958. Details about the Swedish population registers can be found in D'Onofrio et al.[28] Body mass index (BMI, kg/m^2) was calculated from height and weight in Riksät (or Stepwise) assessed at the index date during presentation to an eating disorder service, and was only available for cases.

Defining the Sample

Cases were defined as any individual with a lifetime history of a BED diagnosis in the eating disorders registers Riksät[22] or Stepwise[23] at any point in which an evaluation occurred (i.e., initial or 1-year follow-up visit, years 1999–2009). Inclusion criteria for Riksät and Stepwise are: (a) medical or self-referral to a participating treatment unit, (b) a diagnosed eating disorder, and (c) intent to treat the patient. In cases where the clinical unit decided not to treat the patient (most often because the patient was determined not to have an eating disorder), no enrolment in Riksät or Stepwise is made. In 2009, most specialized eating disorder units (∼90%) and many general psychiatric units in Sweden reported to Riksät and or Stepwise.

In 2009, Riksät, an Internet-based register, included eating disorders-specific information on ∼8,600 patients. Follow-up assessments are annual for the duration of treatment. DSM-IV[29] eating disorder diagnoses (i.e., AN, BN, BED, and EDNOS) are given at each assessment point by a clinician. BED is a unique diagnosis; it is not subsumed under EDNOS.

Most registrants in Stepwise are in Riksät, but Stepwise contains more detailed clinical information on presentation, course, outcome, and related psychopathology. Using both registers allowed us to ascertain all registered cases. Once intent to treat is established, which typically occurs within 1 week for inpatients or three visits for outpatients, diagnosis is made by specially trained clinicians (most of whom attend a special 2-day training in using the Stepwise method battery) using a semi-structured interview (Structured Clinical Interview for DSM-IV Axis I Disorders, SCID-I[30] before 2008, or Structured Eating Disorder Interview, SEDI[31] since 2008) based on DSM-IV-TR criteria.[32]

The Multi-Generation Register allowed us to identify 10 controls for each identified case. We matched controls to cases based on sex and year, month, and county of birth. If a case was born outside of Sweden, controls were matched

TABLE 1. ICD-8, ICD-9, and ICD-10 codes for somatic illnesses from the National Patient Register

Diagnosis	Examples	ICD8	ICD9	ICD10
Neurologic diseases	Headaches, migraine, epilepsy, sleep apnea	320–358	320–349, 356–359	G00–G47, G60–G73, G91
Infectious and parasitic diseases	Colitis, gastroenteritis, viral infection, genital warts	000–136	001–139	A00–B99
Immune system disorders	Sarcoidosis, hyperimmunglobu-lin E (IgE)-syndrome		279	D80–D89
Respiratory diseases	Tonsillitis, acute respiratory infection, asthma	460–466, 470–486, 490–493, 502–508, 783	460–466, 470–478, 480–487, 490–496, 514, 786	J00–J46, R05–R06
Gastrointestinal disorders	Gallstones, appendicitis, dys-pepsia, gastritis	530–535, 540–542, 563, 570–577	530–535, 540–542, 555–558, 570–577	K25–K37, K70–K85
Skin and subcutaneous tissue disorders	Dermatitis, hives, eczema, psoriasis	680–698, 701, 708–709	680–698, 701, 708–709	L00–L54, L90–L95
Musculoskeletal system and connective tissues diseases	Lumbago (low back pain), joint pain, internal derangement of knee	710–718	710–721, 725–729	M00–M68
Genitourinary system disease	Kidney inflammation, kidney stone, kidney infection, uri-nary tract infection, ureter stone	580–584, 590, 592, 594, 601, 604	580–583, 590, 592, 594, 601, 604	N00–N08, N10–N12, N20–N21, N41, N45
Circulatory system diseases	Hypertension, tachycardia, pul-monary embolism, cardiac arrhythmia	390–448, 450	390–448, 456	I00–I79, I98.3
Endocrine system diseases	Polycystic ovarian syndrome, hyperthyroidism, thyrotoxico-sis, autoimmune thyroiditis	240–246, 250, 251–259	240–246, 250, 251–259	E00–E35
Congenital malformations	Congenital non-neoplastic nevus, congenital malforma-tion of the breast, prominent ear, congenital heart defect	740–759	740–759	Q00–Q99
Injury, poisoning, and external causes of morbidity and mortality (excluding suicide)	Fall related accidents, concus-sion, sprain of ankle	N800–N999, E807–E949, E960–E999	N800–N995, E807–E949, E960–E999	S00–T98, V01–X59, X85–Y98

The most common diagnoses within our overall cohort were used to generate the examples.

on immigration status and time of migration (controls could not immigrate later than their respective cases), regardless of origin, in addition to sex and year and month of birth. Controls had to be alive and resident in Sweden for an equivalent period of time: from birth or immigration until the time of diagnosis of their index case. Controls were required not to have received a BED diagnosis in Riksät or Stepwise at any time, but they could have had another eating disorder (which was detected in 1.0% of controls).

Inclusion in the Swedish population registers does not require informed consent. However, the rules governing the Swedish quality registers require that information about the register be provided to the patient and that the patient has the possibility to opt out of participation. For the Stepwise register, research participation is elective via an opt out procedure (about 3% decline participation[33]). The University of North Carolina Biomedical Institutional Review Board (IRB) and the Regional Ethics Committee of Karolinska Institutet both approved this study.

Somatic Diagnoses

Diagnostic information for somatic illnesses for all cases and controls were obtained from the NPR (years

1973 and onward; see Table 1 for the list of diseases and conditions and their diagnostic codes). Individuals listed in the NPR with diagnoses for these disorders based on WHO *International Classification of Diseases, Eighth Revision* (ICD-8: years 1969–1986[34]; diagnoses from ICD-8 were rare and used only for the oldest cases and controls), WHO *International Classification of Diseases, Ninth Revision* (ICD-9: years 1987–1996[35]) or WHO *International Classification of Diseases, Tenth Revision* (ICD-10: years 1997-present[36]) as a principal diagnosis at any time (i.e., lifetime history) were scored as positive for that disorder.

Psychiatric Comorbidity

Psychiatric comorbidity was coded as the presence versus absence of any lifetime psychiatric disorder or suicide-related/intentional self-harm injury and was used as a covariate. Comorbidity was obtained from the NPR and was based on the ICD-8, ICD-9, and ICD-10 classifications. The psychiatric disorders included: *schizophrenia, schizoaffective disorder, bipolar disorder, major depressive disorder* (MDD), *anxiety disorder* (minus obsessive-compulsive disorder and post-traumatic stress disorder), *obsessive-compulsive disorder*

(OCD), *post-traumatic stress disorder* (PTSD), *attention deficit hyperactivity disorder* (ADHD), *autism, alcohol use disorder, illicit drug use disorder,* and *suicide attempts/intentional self-harm*. ICD codes for the disorders are listed in Supporting Information Table 1.

Analysis

Conditional logistic regression models were applied to assess the association of BED status (case/control) with each comorbid somatic illness. Because individuals could be diagnosed with other eating disorders at other times, sensitivity analyses evaluating the association between BED status and the somatic illnesses were conducted excluding cases (and their respective controls) who had an additional eating disorder diagnosis (other than BED) in Riksät or Stepwise or who had a diagnosis of anorexia nervosa or bulimia nervosa in the NPR. These sensitivity analyses evaluated the association of BED and somatic disorders free from confounding of other eating disorders diagnosed at other times. To further investigate any comorbid associations between BED and somatic disorders, models were rerun including lifetime psychiatric comorbidity (defined as a lifetime history of any of the psychiatric illnesses) as a covariate. Given reported associations between obesity and many of the somatic illnesses included, we explored whether obesity (BMI ≥ 30 kg/m^2) was associated with increased risk of the target somatic disorders in individuals with BED with logistic regressions. In these analyses, we controlled for sex, and for county (with adjacent counties grouped into four regions based on geography, and immigrant status treated as one county) and year of birth (with years of birth grouped as before 1965, 66–75, 76–85, after 1986).

All analyses were conducted using SAS version 9.4[37] and all tests were two-tailed. Although we conducted multiple tests, we sought to minimize the probability of making a Type II error in this observational study,[38] thus we did not perform familywise error rate correction for exploratory analyses (only for hypothesis-based analyses and we used the false discovery rate method[39]). Note that based on the fixed sample size for the conditional logistic regression, the odds ratio (OR) detectable with statistical inference, calculated using Lachin's method,[40] ranged from 1.22 (most prevalent) to 1.59 (least prevalent medical conditions), indicating that there may be inadequate power to detect some associations.

Results

The overall sample included 9,350 individuals (95% female). At the time of diagnosis of the index case, the average age of the overall sample was 29 (SD = 9.6) years and 96% were adults (aged 18+ years). Thirty percent of adults were cohabiting (married/de facto) or living with children, 28% were living with their parents, 37% lived alone, and 5% reported other living arrangements. Eight percent were immigrants.

The case sample, described in Welch et al.[41] comprised 850 individuals (39 males, 811 females) with BED. Cases were diagnosed between 14 and 72 years of age. Men were significantly older (mode = 22 years for women and 25 years for men) and had significantly higher BMI values at the time of evaluation than women. Significantly more men were immigrants.

Table 2 presents the results of the conditional logistic regressions evaluating the associations between BED and each somatic disorder in the full sample. BED was significantly associated with all classes of disease evaluated except genitourinary system disorders and congenital malformations. Immune system disorders could not be evaluated due to their rarity. The strongest associations were with endocrine (diabetes mellitus and other disorders) and circulatory system diseases. Sensitivity analyses excluding individuals with BED who also had lifetime diagnoses of other eating disorders (i.e., AN or BN) yielded similar findings: the strongest associations were with endocrine and circulatory system diseases and no association was found for the genitourinary system.

To further investigate the comorbid associations between BED and somatic disorders, we adjusted for any lifetime psychiatric comorbidity, which was present in 47% of cases compared with 11% of controls ($p < 0.001$). The associations between BED and respiratory, skin, musculoskeletal, circulatory, and endocrine problems all remained statistically significant but reduced slightly (**Table 2**). The associations between BED and neurological, infectious/parasitic, and gastrointestinal disorders reduced in magnitude and became statistically nonsignificant, suggesting limited unique variance accounted for by BED.

Table 3 presents the results of logistic regression analyses comparing individuals with BED with ($n = 361$) and without ($n = 489$) comorbid obesity. Individuals with BED with comorbid obesity were more likely to have a lifetime history of respiratory, gastrointestinal, and skin disorders than individuals with BED without comorbid obesity. Sensitivity analyses excluding individuals with BED who also had lifetime diagnoses of other eating disorders (i.e., AN or BN) yielded similar findings: the

TABLE 2. *N* (%) of individuals with each somatic illness by group (case = 850; control = 8,500) and results of conditional logistic regressions evaluating the association of binge-eating disorder (BED) with each somatic illness [OR (95% CI)], and adjusted for lifetime psychiatric comorbidity [AOR (95% CI)]

Somatic Illness Category	BED (Cases) N (%)	Controls N (%)	OR (95% CI)	p Values	AOR (95% CI)	p Values
Neurologic diseases	68 (8.0)	411 (4.8)	1.7 (1.3; 2.2)	0.0002	1.1 (0.8; 1.5)	0.38
Infectious and parasitic diseases	181 (21.3)	1394 (16.4)	1.4 (1.2; 1.6)	0.0005	1.1 (0.9; 1.4)	0.20
Immune system disorders	<4	20 (0.2)	NA[a]	—	NA[a]	—
Respiratory diseases	254 (29.9)	1941 (22.8)	1.4 (1.2; 1.7)	0.0001	1.3 (1.1; 1.5)	0.004
Gastrointestinal disorders	120 (14.1)	903 (10.6)	1.4 (1.1; 1.7)	0.0053[b]	1.1 (0.9; 1.4)	0.82[b]
Skin and subcutaneous tissue disorders	133 (15.7)	926 (10.9)	1.5 (1.2; 1.8)	0.0001	1.3 (1.1; 1.6)	0.014
Musculoskeletal system and connective tissues diseases	178 (20.9)	1156 (13.6)	1.7 (1.4; 2.0)	0.0002[b]	1.5 (1.3; 1.9)	0.0002[b]
Genitourinary system diseases	39 (4.6)	285 (3.4)	1.4 (1.0; 2.0)	0.09	1.2 (0.8; 1.7)	0.42
Circulatory system diseases	42 (4.9)	217 (2.6)	1.9 (1.3; 2.7)	0.0006[b]	1.6 (1.1; 2.4)	0.02[b]
Endocrine system diseases (excluding diabetes mellitus)	84 (9.9)	341 (4.0)	1.8 (1.3; 2.4)	0.0003	1.5 (1.0; 2.0)	0.04[b]
Diabetes mellitus	40 (4.7)	74 (0.9)	5.7 (3.8; 8.7)	0.0002[b]	5.8 (3.6; 9.4)	0.0002[b]
Congenital malformations	37 (4.4)	327 (3.9)	1.1 (0.8; 1.6)	0.50	1.1 (0.7; 1.5)	0.77
Injury, poisoning and external causes of morbidity and mortality (excluding suicide)	402 (47.3)	3154 (37.1)	1.5 (1.3; 1.8)	0.0001	1.1 (1.0; 1.3)	0.15

[a]Cell size < 5, analysis not applied. Psychiatric comorbidity refers to any lifetime psychiatric disorder or suicide-related behavior recorded in the National Patient Register. Controls were matched to cases on sex and year, month, and county of birth. AOR = adjusted odds ratio, CI = confidence interval, OR = odds ratio.
[b]p values adjusted by the method of false discovery rate.

association with gastrointestinal disorders remained significant; however, skin and subcutaneous tissue disorders were no longer significant.

Discussion

Research on the co-occurrence of BED with somatic illnesses may help us conceptualize biological bases for disease, potentially generate novel directions for therapeutic intervention, and inform our understanding of morbidity. This first national register study revealed elevated risk across a range of somatic illnesses in individuals with lifetime BED. Our observations are consistent with previous studies of gastrointestinal,[4,12–14] musculoskeletal,[4] circulatory,[4,7] and endocrine,[4,8–10] disorders but extend the literature by documenting additional increased risk for neurologic, infectious, respiratory, and skin diseases. Of the latter series, skin and respiratory conditions were uniquely associated with BED, whereas the remainder was

dependent on the presence of lifetime psychiatric comorbidity.

The strongest associations with BED and somatic comorbidities were with endocrine (diabetes mellitus and other disorders) and circulatory system diseases, which were not fully accounted for by obesity. This pattern may reflect components of the "metabolic syndrome" which comprises abdominal obesity, atherogenic dyslipidemia, elevated blood pressure, insulin resistance with or without glucose intolerance, and proinflammatory and prothrombotic states. Our findings converge with longitudinal research suggesting that BED confers risk for metabolic syndrome beyond that conferred by obesity alone.[16] Even among children, binge eating predicts subsequent development of metabolic syndrome, which is partly explained by the excess weight gain associated with children's binge eating.[42] Preventing or successfully treating binge eating may have important clinical implications for mitigating the development of metabolic syndrome.

TABLE 3.   Results of logistic regression evaluating the
association of obesity (BMI ≥ 30) with each outcome,
controlling for sex, county, and year of birth[a]

Somatic Illness Category	OR (95% CI)	*p* Values
Neurologic diseases	0.8 (0.5; 1.4)	0.47
Infectious and parasitic diseases	1.1 (0.7; 1.5)	0.77
Immune system disorders	NA[b]	—
Respiratory diseases	1.5 (1.1; 2.1)	0.017
Gastrointestinal disorders	2.7 (1.7; 4.2)	0.0005[c]
Skin and subcutaneous tissue disorders	1.6 (1.1; 2.4)	0.021
Musculoskeletal system and connective tissues diseases	1.1 (0.8; 1.6)	0.82[c]
Genitourinary system diseases	1.1 (0.6; 2.3)	0.74
Circulatory system diseases	1.4 (0.7; 2.9)	0.82[c]
Endocrine system diseases (excluding diabetes mellitus)	1.0 (0.5; 1.8)	0.82[c]
Diabetes mellitus	0.9 (0.4; 1.9)	0.82[c]
Injury, poisoning and external causes of morbidity and mortality (excluding suicide)	1.3 (0.9; 1.7)	0.12

Total *N* = 850; 361 comorbid obesity, 489 no comorbid obesity.

Note: analyses for congenital malformations were conducted using exact logistic regression models controlling only for age group: OR (95% CI): 0.3 (0.1; 0.7), *p* < 0.004.

[a]County of birth was grouped according to region: north, middle, south, outside Sweden; year of birth was grouped as: 1965 and earlier, 1966–1975, 1976–1985, 1986 and later.

[b]Cell size < 5, analysis not applied.

Exact logistic regression analyses conducted controlling only for age group.

[c]*p* values adjusted by the method of false discovery rate.

We confirm and extend observed associations between BED and a broad range of somatic illness categories. Future research should explore more granular diagnoses to further elucidate the exact nature and timing of comorbid somatic illnesses. It was beyond the scope of this study to assess associations between specific somatic illnesses and BED, rather categories for somatic illness according to ICD were used which are broad and inclusive. A second future research direction is to explore the temporality of the associations between BED and these somatic illnesses using prospective evaluations of disease onset in longitudinal cohorts, which could also help clarify whether associations are causal or reflect underlying shared vulnerabilities.

Parsing out the role of obesity and somatic comorbidity is another important step to understanding the biological basis of BED. Within the sample of individuals with BED, we found that the presence of obesity was associated with increased risk for respiratory, gastrointestinal, and skin disorders, but not other classes of illness. This observation reinforces that increased risk for some diseases in individuals with BED, including components of metabolic syndrome, is not simply due to the effects of obesity.

These results must be interpreted within the context of the study limitations. First, only individuals who received a BED diagnosis and had diagnostic information in the eating disorders quality registers (years 1999–2009) were included. Individuals who do not present for treatment may have different patterns of somatic comorbidity. Furthermore, spuriously high rates of comorbidity may occur among clinically ascertained individuals because one comorbidity may be detected via treatment contact for the other or the burden of two comorbid disorders may increase the probability of treatment-seeking.[43] Second, ~95% of the sample were women, thus, results may not be generalizable to men because the prevalence of comorbidities may differ across sexes. Our sample was a young adult sample, hence results are specific to this age group; the magnitude of the associations between BED and somatic illnesses might be affected by age-related factors (e.g., duration and persistence of BED) and length of surveillance. Third, controls were not screened for eating disorders, which may have biased associations between BED and somatic illnesses downward. Fourth, small cell sizes for some disorders resulted in greater imprecision in estimates or inability to apply the analytic model. Fifth, information on BMI (obesity) was not available for controls. Thus, the effects of obesity on the associations between BED and somatic illnesses could not be clarified further. Sixth, care must be taken in interpreting the findings: some somatic problems emerge prior to BED. For this study, controls were required to be unaffected individuals in the cohort who were still alive and under surveillance at the time of diagnosis of the case. Suppose that individuals who do not develop BED (or would not go on to develop BED if they survived until adulthood) are more likely to develop certain severe somatic problems (that result in death) in childhood/adolescence. These individuals would not be eligible for inclusion in analyses, potentially leading to bias in the form of stronger associations between somatic problems and BED in the results. Finally, the time between symptom onset and treatment seeking varies across disorders. Thus, results related to medical comorbidities must be interpreted with caution.

These limitations are balanced by several strengths. First, because registers were used, the sample is large and based on the total population of Sweden. Second, BED diagnoses were made by clinicians using a structured clinical interview. These diagnoses captured subthreshold information and were entered in the eating disorders

quality registers, allowing us to separate BED from other presentations covered by the historical EDNOS diagnosis. Third, somatic illness diagnoses were made by clinicians and were not based on self-report; this increases diagnostic validity and limits recall bias.

Our results demonstrate that BED is associated with a broad range of somatic illnesses. These results have important implications for both mental-health care providers and nonpsychiatric health care providers. For mental-health care providers, our results underscore the importance of recognizing and referring appropriately for somatic complaints. For non-psychiatric, general medical care providers, our results encourage the routine screening for binge eating or excessive overeating in patients presenting for medical care or weight loss. Given the wide range of observed comorbidities, our results support screening for binge eating in primary care. Improving detection of BED in primary care settings could lead to prompt referral, reduce duration of exposure to the illness, and reduce lifetime psychiatric and somatic illness burden.

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