Psychiatric Comorbidity in Women With Binge Eating Disorder: Prevalence Rates From a Non-Treatment-Seeking Sample

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This study provides estimates of comorbid psychiatric disorders in women with binge eating disorder (BED). Sixty-one BED and 60 control participants, who were recruited from the community, completed the Structured Clinical Interview for DSM-III-R Axis I and Axis II disorders and self-report measures of eating and general psychiatric symptomatology. Regarding psychiatric diagnoses, women with BED had higher lifetime prevalence rates for major depression, any Axis I disorder, and any Axis II disorder relative to controls. BED women also evidenced greater eating and psychiatric symptomatology than did controls. Results suggest that the prevalence of comorbid psychiatric disorders in BED may be lower than previously indicated by clinical studies.

Research on binge eating disorder (BED) has mushroomed in the past decade, and a recent review concluded that BED is a significant clinical problem in U.S. society (Castonguay, Eldredge, & Agras, 1995). Large community-based studies have reported prevalence rates for BED between 2% and 5% (Bruce & Agras, 1992; Spitzer et al., 1992, 1993), and 20% to 40% of individuals in treatment for weight control meet criteria for BED (Brody, Walsh, & Devlin, 1994; Gormally, Black, Daston, & Rardin, 1982; Marcus, Wing, & Lamansky, 1985; Spitzer et al., 1993). Moreover, research suggests that the severity of binge eating is associated with degree of overweight (Bruce & Agras, 1992; Spitzer et al., 1993; Telch, Agras, & Rossetter 1988).

Because BED is a proposed new diagnostic category based on the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994), researchers have attempted to elucidate the nature of BED, particularly with regard to comorbid psychopathology. Research using clinical samples has examined the relation of BED to eating pathology (e.g., overconcern with weight and shape) and psychiatric symptomatology (e.g., depressive and anxiety symptoms; Kolotkin, Revis, Kirkley, & Janick, 1987; Marcus et al., 1990; Schwalberg, Barlow, Alger, & Howard, 1992; Spitzer et al., 1993; Telch, Agras, 1994; Wilson, Nunas, & Rosenblum, 1993; Yanovski, Nelson, Dubbert, & Spitzer, 1993) as well as to the prevalence of psychiatric disorders (Brody et al., 1994; Marcus et al., 1990; McCann, Rossiter, King, & Agras, 1991; Schwalberg et al., 1992; Yanovski et al., 1993). These studies have generally concluded that BED is associated with elevated eating and general psychiatric symptomatology, as well as with high rates of comorbid psychiatric disorders.

However, estimates of comorbid psychiatric disorders in BED have been based primarily on studies using samples recruited for treatment trials. Within an obesity treatment trial, Marcus et al. (1990) found that a greater proportion of obese binge eaters met lifetime criteria for major depression relative to obese controls. McCann et al. (1991), in a binge eating treatment study, found that women with bulimia nervosa and female obese binge eaters did not differ on lifetime prevalence of major depression (74% and 48%, respectively). Substantial lifetime prevalence rates of major depression or dysthymia (64%) and anxiety disorders (70%) based on the revised third edition of the DSM (DSM-III-R; American Psychiatric Association, 1987) were reported in 22 female obese binge eaters seeking treatment in an eating disorders clinic (Schwalberg et al., 1992). Finally, Yanovski et al. (1993) reported on a sample of overweight men and women, one third of which was seeking treatment in a weight-loss study, and found that lifetime prevalence rates of major depression, panic disorder, bulimia nervosa, borderline personality disorder, and avoidant personality disorder were higher in participants meeting criteria for BED, compared with non-BED controls.

It has been argued that findings from individuals seeking treatment may not be representative of persons with the disorder in the general population because these samples may overestimate comorbid pathology. This argument is based on a phenomenon called Berkson’s bias, which states that persons with multiple diagnoses are more likely to seek treatment than persons with a single diagnosis (Berkson, 1946). Moreover, most individuals with bulimia nervosa (Fairburn & Cooper, 1982; Welch & Fairburn, 1994; Whitaker et al., 1990), and other psychiatric disorders (Goodman et al., 1997; Robins, Locke, & Regier, 1991; Whitaker et al., 1990), do not seek treatment. For example, the Epidemiologic Catchment Area Study (ECA;
Robins et al., 1991) suggested that only 13% of individuals with a single psychiatric disorder received treatment in the past year. Hence, data from individuals seeking treatment may not be representative of the population of persons with the disorder, potentially biasing estimates of comorbid disorders and general psychiatric symptomatology.

Two additional methodological limitations of past studies should be noted. First, both Marcus et al. (1990) and Schwalberg et al. (1992) relied primarily on the self-report Binge Eating Scale (BES) to classify obese participants as binge eaters, and there is evidence that this scale may not reliably capture the current DSM-IV criteria for BED (Gladis, Wadden, Foster, Vogt, & Wingate, 1998). Second, the number of obese binge eaters in these studies was relatively small (Marcus et al., n = 25; McCann et al., n = 31; Schwalberg et al., n = 22; Yanovski et al., n = 43), thereby limiting our confidence in the reliability of comorbidity estimates.

The present investigation sought to provide a more representative estimate of psychiatric comorbidity in women with BED than provided by past studies, which were potentially biased by the use of clinical samples. Additionally, we improved on earlier studies by including a larger sample of participants with BED and by using a structured clinical interview to verify DSM-IV BED diagnosis. To accomplish these aims, we examined the prevalence of psychiatric disorders and the degree of general psychiatric and eating pathology symptoms in a sample of women diagnosed with BED and overweight women without BED, who were recruited for a research study on eating. We hypothesized that the rates of comorbid psychiatric disorders and the levels of eating and psychiatric symptomatology would be greater in the women with BED than in the control women. To our knowledge, this is the first study to examine comorbidity in a comparatively large sample of women with BED who were not specifically seeking treatment. We propose that individuals in our nontreatment-seeking sample will provide more representative comorbidity estimates than those reported from studies using clinical samples.

Method

Procedures

Participants in this study—61 women who met DSM-IV research criteria for BED and 60 overweight noneating-disordered (NED) controls—were recruited directly from the community to participate in a paid research study on eating behavior. Advertisements placed in local media invited overweight women between the ages of 18 and 65 to participate in a research study on eating for $150. These eating studies involved three different experimental laboratory studies designed to examine factors influencing binge eating in women with BED, and in one of these studies a small cohort of bulimia nervosa participants was included (see Telch & Agras, 1996a, 1996b, for greater details). Information from clinical interviews and an assessment battery conducted before the laboratory protocol form the basis for the data reported here. Participants were informed that interviews were conducted to learn about their eating habits and behaviors and to understand any emotional or psychological problems they may have had. Participants were informed that the $150 would be paid after completion of the laboratory protocol, which involved three separate visits.

Figure 1 illustrates the recruitment process. Participants had to (a) be female, (b) be between 18 and 65 years old, and (c) currently meet DSM-IV research diagnostic criteria for BED or criteria for an NED control (i.e., demonstrated no evidence of current or past binge eating, subjective sense of a loss of control during eating, purging, or any behavior that might meet criteria for an eating disorder not otherwise specified). Thirty-seven women with bulimia nervosa were recruited for one laboratory study but were not included here. Exclusion criteria (see below) were developed to eliminate factors that could confound interpretation of results in the experimental studies of eating behavior and were not based on exclusion for strictly clinical reasons.

Of the 993 respondents to the advertisements, 674 were successfully contacted and underwent a brief telephone screen to assess eligibility for participation. This interview included questions about age, current weight, binge eating, purging, medical conditions, and medication use. Of the 367 excluded during the screen, 160 were due to lack of interest once the study was described; 141 had a binge–purge frequency below diagnostic threshold, 33 had a physical or medical condition, such as diabetes or cancer, that affected eating; 14 were younger than 18 or older than 65, 14 were on medication that affected eating (to minimize effects on eating by starting or titrating antidepressants, we allowed only participants who were stable on antidepressants for at least 1 month into the study—the 14 excluded participants had been taking antidepressants for less than 1 month), 3 had a body mass index (BMI) below 17.5, suggesting possible anorexia nervosa, and 2 were male.

Participants who were eligible following the telephone screen (n = 229) were invited to an interview to obtain informed consent, were given a description of the laboratory eating study, and were given the clinical interviews and assessment battery. Fifty-three women canceled or failed to show for the interview (51% potential BED participants, 40% potential controls, and 9% potential bulimia participants). Of the 139 participants who attended the interview, 14 were excluded due to not meeting the minimum binge frequency threshold for BED of twice per week on average during the past 6 months. Two participants were excluded during the interview for medical reasons (current cancer treatment and food allergies) not disclosed during the screen, and 1 participant was excluded due to nonpurging bulimia (regular use of excessive compensatory exercise). The interview could not be completed with 1 participant who appeared to have a cognitive disorder not otherwise specified or possible psychosis; hence, this participant was excluded and an eating disorder diagnosis could not be established.

Measures

Participants attending the interview gave voluntary written informed consent. Prior to interview, height and weight were measured using a balance beam scale. BMI was used to reflect adiposity (Garrow & Webster, 1985) and has been found to be a reliable and valid index of adiposity (Kraemer, Berkowitz, & Hammer, 1990). The interview was designed to obtain diagnostic information regarding BED and comorbid psychiatric disorders. The following instruments were administered by PhD- or master's-level psychologists trained in the administration of these instruments.

Questionnaire on Eating and Weight Patterns (QEWP, Spitzer et al., 1992). The QEWP is a self-report measure containing items that assess the individual components, duration, and frequency criteria for the proposed DSM-IV BED research criteria. The level of diagnostic agreement between the QEWP and expert rating is acceptable (Spitzer et al., 1993). To determine a diagnosis of BED, the interviewer conducted a detailed review with the participant of each response to QEWP items.

Structured Clinical Interview for DSM–III–R (SCID; Spitzer, Williams, Gibbon, & First, 1990a). The SCID is a standardized interview used extensively in research and clinical settings. The SCID assesses...
current and lifetime psychiatric status for major Axis I psychiatric disorders using criteria in accordance with the DSM. The reliability and validity of the SCID have been well documented, with interrater reliability agreement (kappas) ranging from .70 to 1.00 (Segal, Hersen, & Van Hasselt, 1994; Strakowski et al., 1993; Stukenberg, Dura, & Kiecolt-Glaser, 1990).

SCID personality disorders (SCID II; Spitzer, Williams, Gibbon, & First, 1990b). The SCID is a standardized interview used in research and clinical settings for the diagnosis of Axis II personality disorders. The SCID II has acceptable reliability and validity, with kappas ranging from .65 to 1.00 (Malow, West, Williams, & Sutker, 1989; O'Boyle & Self, 1990; Renneberg, Chumblless, Dowdall, Fauerbach, & Gracely, 1992).

Participants also completed several self-report scales designed to provide information regarding eating-related pathology and general psychiatric symptomatology. These measures were included to validate the grouping variable (BED vs. NED) and to characterize our sample.

BES (Gormally et al., 1982). The BES is a 16-item questionnaire assessing binge eating problems. It is commonly used to assess severity of binge eating in BED and discriminates among obese individuals with no, moderate, or severe binge eating problems (Gormally et al., 1982; Marcus, Wing, & Hopkins, 1988). A score of 27 and above indicates severe binge eating problems, and a score of 17 and below designates no binge eating problems.

Eating Disorder Examination—Questionnaire (EDE-Q; Fairburn & Beglin, 1994). The EDE-Q is a 38-item questionnaire measuring eating pathology and is derived directly from the Eating Disorder Examination interview (EDE; Fairburn & Cooper, 1993). The EDE has become the gold standard for assessing eating disorders. The EDE-Q focuses on the past 28 days to assess the main behavioral (binge eating and purging) and attitudinal features of eating disorders. The EDE-Q contains four subscales: Restraint, Eating Concern, Weight Concern, and Shape Concern. These scales have been found to have acceptable reliability and validity (Black & Wilson, 1996; Fairburn & Beglin, 1994).

Three-Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985). The TFEQ is a 54-item questionnaire with three subscales that measure cognitive (dietary) restraint, perceived hunger, and emotionally based disinhibition of eating. Research has demonstrated that this measure has acceptable reliability and validity (Laessle, Tuschl, Kotthaus, & Pirke, 1989; Stunkard & Messick, 1985).

Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI is a 21-item scale assessing somatic, affective, cognitive, and behavioral symptoms of depression. Research has documented the reliability and validity of this scale (Beck, Steer, & Garbin, 1988). The BDI was included to provide a continuous measure of depressive symptomatology.

Symptom Checklist—90 (SCL—90; Derogatis, Lipman, & Covi, 1973). The SCL—90 is a 90-item questionnaire assessing general psychiatric symptomatology and yields an overall index of psychiatric distress, the General Symptomatic Index (GSI). The GSI was used in our study to provide a continuous measure of general psychiatric symptoms. Research has documented the reliability and validity of this scale (Derogatis, 1977).

Rosenberg Self Esteem Scale (RSE; Rosenberg, 1979). The RSE is a 10-item questionnaire that measures beliefs and attitudes regarding general self-worth. Higher scores on the RSE indicate higher self-esteem.
Research has documented the reliability and validity of this scale (Demo, 1985; Rosenberg, 1979).

**Statistical Analysis**

We analyzed SCID data on current and lifetime Axis I and Axis II diagnoses using the chi-square statistic, except in instances where the cell frequencies dropped below five cases, in which case Fisher’s exact test was performed. Relative risks were calculated for lifetime presence of Axis I diagnoses and Axis II diagnoses unless one or more cells had zero cases, thus preventing the calculation of this statistic. We analyzed continuous scales using independent sample t tests. A standard Bonferroni correction was applied when analyzing the demographic information to minimize Type I error-rate inflation associated with multiple tests of nonhypothesized relations.

**Results**

**Preliminary Analyses**

Demographic data on the BED and NED participants are presented in Table 1. The BED group did not differ significantly from the control group in terms of age, education (a proxy for socioeconomic status), weight, BMI, rates of obesity (BMI >28), ethnicity, marital status, and employment status after a standard Bonferroni correction. Additionally, the age that the participant first reported becoming overweight did not differ between the BED and NED participants (see Table 1). Eighty-eight percent of the BED participants and 78% of the NED participants were obese (BMI >28). Onset of binge eating was reported by the BED participants as ranging from ages 6 to 56, with a mode of 18. The BED participants reported an average of 4.5 (SD = 3.3) binge episodes and 3.4 (SD = 1.8) binge days per week on a calendar recall of the prior week.

Recruitment advertisements invited overweight women to participate; however, the only weight-related exclusionary criterion involved eliminating women with a BMI below 17.5. As indicated above, approximately 17% of the participants were not objectively overweight but responded to the ads because they perceived themselves to be overweight. Although analyses revealed that the BED and control groups were equally overweight, we tested whether or not weight was related to psychiatric diagnoses independent of BED status. Logistic regression analyses revealed that BMI was not significantly related to any of the individual current or lifetime Axis I or Axis II diagnoses.

Given our proposal that past studies may have overestimated the prevalence of comorbid psychiatric diagnoses in BED (because they used patients seeking treatment), it is important to examine the proportion of participants in our sample who were receiving treatment. Eighteen participants (15%) were currently receiving some form of clinical treatment (12 BED, 6 NED). Of these, 6 (5%) were involved in formal weight-loss programs exclusively; 5 (5%) were taking psychotropic medications; 2 (2%) were receiving psychotherapy; and 5 (5%) were receiving both psychotropic medications and psychotherapy. There were no significant differences between the BED women and the controls in rates of current treatment use. We also tested whether there were differences in the prevalence of comorbid psychiatric disorders between the BED participants currently in treatment and the BED participants not in treatment. No significant differences were found; however, because only 12 BED participants were currently receiving treatment, the power to detect a medium effect size for the differences between proportions was only .23 (Cohen, 1988). Finally, a chi-square test revealed that a significantly higher proportion of the BED participants had received past psychiatric treatment (n = 46, 75%) compared with the NED controls (n = 32, 53%, p < .05).

**Psychiatric Diagnoses**

Current and lifetime prevalence rates of Axis I psychiatric disorders for the BED and NED participants are presented in Table 2. Analyses revealed that the BED participants evidenced a significantly higher lifetime prevalence rate of major depression (49%) and a lifetime history of any Axis I diagnosis (59%) compared with the NED controls (28% and 37%, respectively). For the BED participants, the relative risk of receiving a lifetime diagnosis of major depression or any Axis I disorder was about twice that of the controls. However, there were no significant differences between the BED and NED groups in current major depression, bipolar disorder (lifet ime or current), dysthymia, alcohol abuse or dependence (lifetime or current), substance abuse or dependence (lifetime or current), panic disorder (lifetime or current), agoraphobia (lifetime or current), social phobia (lifetime or current), obsessive–compulsive disorder (lifetime or current), or bulimia nervosa (lifetime).

The prevalence rates of Axis II personality disorders for the BED and NED participants are presented in Table 3. No significant between-groups differences were found in the rates of any individual Axis II personality disorders, including avoidant, de-
Table 2
Frequency of Diagnostic Levels of Axis I Disorders for the Binge Eating Disorder (BED) Group and the Control Group

<table>
<thead>
<tr>
<th>Axis I Diagnosis</th>
<th>BED group (n = 61)</th>
<th>Control group (n = 60)</th>
<th>Relative risk*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depression (lifetime)</td>
<td>30 49*</td>
<td>17 28</td>
<td>1.74</td>
</tr>
<tr>
<td>Major depression (current)</td>
<td>3 5</td>
<td>1 2</td>
<td>2.88</td>
</tr>
<tr>
<td>Bipolar (lifetime)</td>
<td>1 2</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Bipolar (current)</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Dysthymia</td>
<td>4 7</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Alcohol abuse or dependence (lifetime)</td>
<td>9 15</td>
<td>6 10</td>
<td>1.48</td>
</tr>
<tr>
<td>Alcohol abuse or dependence (current)</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Substance abuse or dependence (lifetime)</td>
<td>6 9</td>
<td>2 3</td>
<td>2.97</td>
</tr>
<tr>
<td>Substance abuse or dependence (current)</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Panic (lifetime)</td>
<td>7 12</td>
<td>4 7</td>
<td>1.72</td>
</tr>
<tr>
<td>Panic (current)</td>
<td>1 2</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Agoraphobia (lifetime)</td>
<td>2 3</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Agoraphobia (current)</td>
<td>1 2</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Social phobia (lifetime)</td>
<td>3 5</td>
<td>3 5</td>
<td>0.98</td>
</tr>
<tr>
<td>Social phobia (current)</td>
<td>3 5</td>
<td>3 5</td>
<td>0.98</td>
</tr>
<tr>
<td>Obsessive–compulsive (lifetime)</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Obsessive–compulsive (current)</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Bulimia nervosa (lifetime)</td>
<td>1 2</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Any Axis I (lifetime)</td>
<td>36 59*</td>
<td>22 37</td>
<td>1.61</td>
</tr>
</tbody>
</table>

* It is not possible to calculate the relative risk if there are any cells with a 0 frequency.
* p < .05.

Table 3
Frequency of Diagnostic Levels of Axis II Disorders for the Binge Eating Disorder (BED) Group and the Control Group

<table>
<thead>
<tr>
<th>Axis II Diagnosis</th>
<th>BED group (n = 61)</th>
<th>Control group (n = 60)</th>
<th>Relative risk*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avoidant</td>
<td>4 7</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Dependent</td>
<td>2 3</td>
<td>1 2</td>
<td>1.94</td>
</tr>
<tr>
<td>Obsessive–compulsive</td>
<td>3 5</td>
<td>1 2</td>
<td>2.88</td>
</tr>
<tr>
<td>Passive–aggressive</td>
<td>2 3</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Self-defeating</td>
<td>3 5</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Paranoid</td>
<td>3 5</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Schizotypic</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Schizoid</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Histrionic</td>
<td>0 0</td>
<td>2 3</td>
<td></td>
</tr>
<tr>
<td>Narcissistic</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Borderline</td>
<td>4 7</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Antisocial</td>
<td>0 0</td>
<td>0 0</td>
<td></td>
</tr>
<tr>
<td>Any (lifetime)</td>
<td>12 20*</td>
<td>3 5</td>
<td>3.94</td>
</tr>
</tbody>
</table>

* It is not possible to calculate the relative risk if there are any cells with a 0 frequency.
* p < .05.

With the relative risk indicating that the women with BED were almost four times as likely as the NED controls to receive an Axis II diagnosis. Almost 20% of the women with BED met criteria for an Axis II disorder compared with 5% of the controls.

Eating Pathology and General Psychiatric Symptomatology

Results of the t tests performed on continuous measures of eating disorder and general psychiatric symptomatology are presented in Table 4. As expected, the women with BED reported significantly greater eating disorder symptomatology than did the NED controls. The BED participants evidenced a higher mean score on the BES, indicating greater binge eating severity compared with the controls. As shown in Table 4, significant between-groups differences were found on each of the EDE-Q subscales of Restraint, Eating Concern, Weight Concern, and Shape Concern, each indicating more eating disturbance in the women with BED relative to the controls. The BED participants demonstrated greater disinhibition of eating and perceived hunger on the TFEQ subscales compared with the controls, although the difference for the TFEQ Restraint subscale did not reach significance.

As shown in Table 4, the women with BED demonstrated significantly lower self-esteem, a higher level of general psychiatric symptoms measured by the GSI of the SCL–90, and higher depression scores on the BDI relative to the NED controls.
Table 4

Differences Between the Binge Eating Disorder (BED) Group and the Control Group on Continuous Measures of Eating Disturbances and Psychopathology

<table>
<thead>
<tr>
<th>Pathology and measure</th>
<th>BED group (n = 61)</th>
<th>Control group (n = 60)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EDE-Q Restraint</td>
<td>2.31</td>
<td>1.64</td>
</tr>
<tr>
<td>EDE-Q Weight Concern</td>
<td>3.75</td>
<td>2.67</td>
</tr>
<tr>
<td>EDE-Q Shape Concern</td>
<td>4.36</td>
<td>3.31</td>
</tr>
<tr>
<td>TFEQ Restraint</td>
<td>7.46</td>
<td>6.89</td>
</tr>
<tr>
<td>TFEQ Hunger</td>
<td>8.07</td>
<td>7.05</td>
</tr>
<tr>
<td>EDE-Q Eating Concern</td>
<td>2.80</td>
<td>2.31</td>
</tr>
<tr>
<td>EDE-Q Shape Concern</td>
<td>4.36</td>
<td>3.31</td>
</tr>
<tr>
<td>EDE-Q Weight Concern</td>
<td>3.75</td>
<td>2.67</td>
</tr>
<tr>
<td>EDE-Q Restraint</td>
<td>2.31</td>
<td>1.64</td>
</tr>
<tr>
<td>Rosenberg Self Esteem Scale</td>
<td>28.84</td>
<td>32.96</td>
</tr>
<tr>
<td>Symptom Checklist—90</td>
<td>0.56</td>
<td>0.29</td>
</tr>
<tr>
<td>Beck Depression Inventory</td>
<td>11.04</td>
<td>5.78</td>
</tr>
</tbody>
</table>

Note. TFEQ = Three Factor Eating Questionnaire; EDE-Q = Eating Disorder Examination—Questionnaire.
+ p < .05. *** p < .001.

Discussion

This study investigated comorbidity of psychiatric disorders and eating and general psychiatric symptomatology in a sample of women with BED and overweight controls who were not from a clinical sample. Our aim was to provide estimates of psychiatric comorbidity that are more representative than estimates reported in past studies that used participants seeking treatment. According to Berkson’s bias, participants with more than one diagnosis are more likely to seek treatment. Thus, previous studies of BED that used clinical samples likely overestimated the rates of comorbid psychiatric disorders. Our sample included women in the community who responded to advertisements for paid research studies. Although participants were not randomly selected, our sampling design was less likely to be vulnerable to Berkson’s bias. Although having a comorbid psychiatric disorder would logically increase the likelihood of seeking treatment, there is no obvious reason why it would increase the likelihood of responding to advertisements for a research study on eating. Note that, although ours is not a random sample, even individuals randomly selected from the community to participate in research must respond to recruitment and consent to participate, possibly introducing some bias. Whereas our sampling procedure is less likely to result in overestimates of comorbid pathology than past research, future studies should use large community-based random samples to yield optimal estimates of comorbid disorders.

Our data suggest that there are few differences in rates of comorbid psychiatric disorders between women with BED and similarly obese controls. Across the spectrum of Axis I and Axis II disorders, a significant difference was demonstrated for only one diagnostic category: Women with BED evidenced a significantly higher lifetime prevalence rate of major depression than did controls. Results from our nontreatment-seeking sample corroborate findings from clinical samples (e.g., Marcus et al., 1990; Yanovski et al., 1993), suggesting that BED is associated with an elevated lifetime prevalence rate of major depression. When collapsing across individual Axis I and Axis II disorders, we found that analyses indicated that the BED participants were significantly more likely to meet lifetime diagnostic criteria for any Axis I or II disorder compared with the controls. Our finding that BED participants were more likely than controls to meet criteria for lifetime diagnosis of any Axis I disorder converges with findings from two past studies that used clinical samples (Marcus et al., 1990; Yanovski et al., 1993). Between-group differences on any Axis II disorder were evidenced in both our own and the Yanovski et al. study, although the percentage of BED participants in the Yanovski et al. study who met criteria for any Axis II disorder (35%) was higher than in our BED participants (20%).

There were also noteworthy differences between our results and those from past clinical studies. Yanovski et al. (1993) reported greater lifetime prevalence of panic disorder for BED versus non-BED participants, and Schwalberg et al. (1992) reported that 70% of obese binge eaters met lifetime criteria for a DSM-III-R anxiety disorder. However, no differences on anxiety disorders were found in our sample, and lifetime prevalence rates for anxiety disorders in our nontreatment-seeking sample were much lower than reported by Schwalberg et al. Additionally, Yanovski et al. found that prevalence rates of borderline personality disorder and avoidant personality disorder were higher in BED participants compared with controls, yet we found no differences for these personality disorders in our sample.

Because we propose that past studies that used clinical samples may have overestimated psychiatric comorbidity in BED, it is important to examine the proportion of participants receiving clinical treatment in our sample. Approximately 15% of our participants were receiving some form of clinical services. This rate is nearly identical to that reported in the ECA (Robins et al., 1991), which indicated that 16% of women with any single disorder and 19% of the total affected sample had mental health care in the past year. This increases our confidence in the representativeness of our sample. Moreover, the ECA data indicate that 6% of women without a diagnosis had been in treatment in the past year, highlighting that a sample without any individuals currently receiving treatment would be unrepresentative. Relatively, there were no differences in rates of current treatment between the BED and control participants in our sample, suggesting that any effect of having a portion of participants in current treatment was essentially equivalent across groups.

Although the critical difference between our study and past research involves the use of participants who were not recruited for a treatment trial, it is important to consider other differences.

1 It may be noted that when the 18 participants (12 with BED and 6 control participants) who were currently receiving treatment were excluded from the analysis, the results were identical with the exception of one additional finding: The women with BED were also more likely to meet criteria for lifetime substance abuse or dependence compared with the control women (p < .05). However, because this subsample of participants who were not currently receiving treatment was less representative, we give precedence to the results from the entire sample.
that may explain why our results differed from past studies. For example, Marcus et al. (1990), Yanovski et al. (1993), and Schwalberg et al. (1992) excluded participants who did not meet standard criterion for overweight, whereas the only weight criterion in our study was that participants not meet criteria for anorexia nervosa. Thus, participants in the past studies may have been more obese, possibly accounting for some of the differences in findings. However, the lack of relationship between BMI and psychiatric diagnoses in our sample suggests that weight differences do not account for observed differences in psychiatric comorbidity. Although it might be argued that differences in diagnostic instruments could contribute to differential findings, the fact that both we and Yanovski et al. used the SCID and the SCID II indicates that this cannot explain the contrary findings. Finally, because our sample (n = 121) was larger than Schwalberg et al.'s (n = 82), Marcus et al.'s (n = 50), and McCann et al.'s (n = 51), and comparable with Yanovski et al.'s (n = 128), the fewer significant findings in our study cannot be explained by a lack of statistical power. Power calculations (Cohen, 1988) indicated that our power to detect a medium effect size with an alpha of .05 was .78, suggesting adequate statistical power and increasing our confidence that the present findings are likely the most stable estimates of comorbid disorders available to date.

For control participants in our study, lifetime prevalence rates for psychiatric disorders appeared comparable with rates reported in epidemiological studies (see Robins et al., 1991; APA, 1994). Moreover, for women with BED in our study, lifetime prevalence rates of psychiatric disorders were similar to those reported in the ECA and the DSM-IV for all disorders, with the exception of those rates found to be higher relative to the controls in our study. The ECA reported a lifetime prevalence rate of 9% for major depression in women (Weissman, Bruce, Leaf, Florio, & Holzer, 1991), and the DSM-IV cites the lifetime risk for major depression in community samples to be 10–25% for women. Women with BED in our study were well above this rate (49%), whereas our controls (28%) were close to the rates stated in the DSM-IV. The ECA data indicated a lifetime prevalence of 32% for any Axis I disorder (Robins et al., 1991), which is comparable with the rate found in our control group (37%) but markedly lower than that found in the BED sample (59%). Thus, it appears that women with BED are generally at risk for experiencing an Axis I disorder, but this risk is not specific to any single diagnostic category, with the exception of major depression. The rate of any Axis II personality disorder reported in a large-community epidemiological study was 18% (Zimmerman & Coryell, 1989), which is similar to the rate found in our BED sample (20%) but higher than the rate found in our control sample (5%). It is thus possible that the effect for any Axis II disorder in our study reflects the lower rate for controls in our study rather than a meaningful elevation for women with BED in our study. In sum, it appears that BED is associated with higher lifetime prevalence rates of major depression and any Axis I disorder when compared with rates from community-based studies and with non-BED controls. However, the conclusion from past research that BED is associated with higher rates of anxiety disorders and Axis II disorders is equivocal.

The significant differences evidenced on all but one (TFEQ Restraint subscale) of the continuous-eating pathology measures validates the grouping variable (BED vs. NED). Most centrally, scores on the BES (Gormally et al., 1982), a measure of binge eating severity, were higher for the participants with BED. Results from our sample of non-treatment-seeking women also revealed differences on the continuous measures of depressive symptomatology and general psychiatric symptoms that converge with past findings from clinical samples (Brody et al., 1994; Marcus et al., 1988; Spitzer et al., 1993; Wilson et al., 1993). Collectively, findings for the continuous measures strengthen our conclusion that individuals with BED evidence psychopathology specific to eating disorders and depression, as well as elevated levels of general but nonspecific psychiatric symptoms.

Regarding clinical implications, our findings highlight the importance of diagnosing BED in overweight individuals, given the association between BED and depression. Relatedly, note that most treatment outcome studies have reported no changes in depression-related measures despite improvements in binge eating (Agras et al., 1994, 1995; Telch, Agras, Rossitter, Wilfley, & Kenardy, 1990). Depressive symptomatology may render individuals more vulnerable to binge eating relapse, yet standard treatments for BED do not directly target the relation between binge eating and depression. Another model posits that binge eating arises in the context of affect dysregulation and because individuals with BED lack adaptive affect regulation skills, they use binge eating to regulate their mood. Thus, teaching adaptive emotion regulation skills to people with BED may allow them to replace binge eating with healthier skills and reduce depression that might threaten binge abstinence.

Although our sampling procedures and relatively large sample improved on past studies, our design was not optimal. Ideally, an even larger sample would be used to enhance statistical power and the stability of the comorbidity estimates. Moreover, use of a random sample would have further reduced potential sampling biases. Future studies might use random-digit dialing procedures to generate an even more representative sample than provided here. Finally, because we excluded men from our sample, generalization of findings should be limited to women with BED.

References


Received July 7, 1997
Revision received December 17, 1997
Accepted April 8, 1998

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