Psychiatric Comorbidity in Binge-Eating Disorder as a Function of Smoking History

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Objective: To examine the comorbidity of psychiatric disorders in obese women with binge-eating disorder (BED) as a function of smoking history.

Method: A consecutive series of 103 obese treatment-seeking women with current DSM-IV diagnoses of BED were administered structured diagnostic interviews to assess all DSM-IV Axis I psychiatric disorders. Participants were classified as "never" or "daily" smokers, and lifetime rates of comorbid psychopathology were compared across smoking groups using logistic regression. The study was conducted from February 2003 to March 2005.

Results: Smokers were significantly more likely to meet criteria for co-occurring diagnoses of major depressive disorder ($p = .03$), panic disorder ($p = .01$), posttraumatic stress disorder ($p < .05$), and substance abuse or dependence ($p = .01$). Even after excluding participants with substance use disorders, significant differences remained, with lifetime smokers having significantly higher rates of co-occurring anxiety disorders.

Conclusions: It is possible that for some obese women with BED, binge eating and cigarette smoking share common functions, i.e., both behaviors may serve to modulate negative affect and/or anxiety. Although the current findings are consistent with a view of a common diathesis for the development of impulsive eating, cigarette or other substance use, and additional Axis I psychopathology, prospective longitudinal studies are needed to elucidate the nature of potential pathways.

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In the spirit of full disclosure and in compliance with all ACCME Essential Areas and Policies, the faculty for this CME article were asked to complete a statement regarding all relevant financial relationships between themselves or their spouse/partner and any commercial interest (i.e., a proprietary entity producing health care goods or services) occurring within at least 12 months prior to joining this activity. The CME Institute has resolved any conflicts of interest that were identified. The disclosures are as follows: Drs. White and Grilo have no significant commercial relationships to disclose relative to the presentation.

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Binge-eating disorder (BED) is a new eating disorder criteria set included as a research category in Appendix B of the *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition (DSM-IV).[1] BED, characterized by recurrent binge eating without inappropriate compensatory weight-control methods, is a prevalent clinically meaningful public health problem.[2,3]

Unanswered questions remain regarding the construct validity of the BED diagnosis.[4,5] The construct validity as well as important issues related to the underlying nature of BED can be approached by means of the study of comorbidity. To date, most comorbidity studies of BED have focused on rates of Axis I psychopathology in individuals with BED as compared to non–binge-eating obese controls (NBO). These studies have attempted to determine the extent to which BED may be associated with other forms of psychopathology and whether individuals with BED are more distressed than their NBO counterparts. Increased psychiatric comorbidity among individuals with BED, therefore, would be suggestive of a vulnerability that is specific to BED as opposed to obesity. Overall, research with both community and clinical samples generally has documented that obese individuals with BED differ from NBO in psychiatric comorbidity, with individuals with BED having significantly higher lifetime rates of various psychiatric disorders, most notably depressive, anxiety, and substance use disorders (SUDs).[6–8] Collectively, these findings have led some researchers to conclude that BED may be best conceptualized as a marker of psychopathology rather than a psychiatric disorder.[4]

A distinct line of research has investigated rates of SUDs and cigarette smoking among individuals with eating disorders. This research, focused thus far on individuals with anorexia nervosa and bulimia nervosa, has found a general worsening of the eating disorder profile with concurrent SUD. In community samples, cigarette smoking and substance use were correlated with several features of eating disorder pathology, including greater body dissatisfaction and binge eating, although the magnitude of the associations was weaker than generally seen for clinical samples.[9] Rates of clinically significant eating pathology are higher among individuals with SUDs than in the general population[10] and are higher in patients with SUDs than in comparison patient groups.[11] Conversely, rates of cigarette smoking[12] and alcohol abuse[13] are higher

among individuals with eating disorders than in the general population. One study reported a higher rate of smoking within a group of individuals with bulimia nervosa than among individuals with affective or anxiety disorders.[14] Some studies,[10,15] but not all,[16] have reported that rates of comorbid Axis I psychopathology are higher for individuals with both SUD and eating disorder diagnoses than with either diagnosis alone.

Consistent with these clinical studies, epidemiologic research has found higher rates of current[17] and lifetime[18] Axis I disorders in smokers as compared to nonsmokers. In a community sample, Black et al.[19] found significantly higher lifetime rates of major depression, agoraphobia, and SUDs among lifetime smokers than among nonsmokers. However, for prediction of smoking history/status, only history of SUD was significant, suggesting that only an SUD diagnosis was independently associated with smoking (i.e., rates of depression were no longer higher in smokers after controlling for SUDs). A larger epidemiologic study, however, found that nicotine dependence was independently associated with increased risk of anxiety and mood disorders, although the increased risk was even more profound for SUDs.[20]

Collectively, the research on eating disorders and SUDs and cigarette smoking indicates that the occurrence of one disorder increases the risk of the other. As suggested by the general epidemiologic research, it could be that a common vulnerability predisposes individuals to abuse substances, engage in disordered eating, and/or develop additional psychiatric problems. To date, no study has investigated the rate of cigarette smoking in BED, and no reports exist regarding the psychiatric comorbidity in BED as a function of smoking history. This study examined the rates of cigarette smoking and DSM-IV–defined Axis I psychiatric comorbidity in obese women with BED as a function of smoking (i.e., "never" smokers were compared to "ever" smokers). Because of the high co-occurrence of SUD and smoking, we also examined the rates of psychiatric comorbidity after controlling for lifetime SUD. It was expected that compared to nonsmokers with BED, individuals with BED and a smoking history would experience higher rates of lifetime psychopathology.

METHOD

Participants

Participants were a consecutive series of 103 obese (body mass index [BMI] ≥ 30) women recruited for treatment studies who met full DSM-IV research criteria[1] for BED. The study was conducted from February 2003 to March 2005. Mean age of the participants was 45.8 years (SD = 8.1); mean BMI was 37.8 (SD = 6.0). The racial/ethnic distribution was 79.6% white (N = 82), 10.7% African American (N = 11), 7.8% Hispanic (N = 8), 1.0% Asian (N = 1), and 1.0% "other" (N = 1). In terms of educational attainment, 17.8% (N = 18) graduated from high school, 33.7% completed some college (N = 34), and 48.5% had a college degree (N = 49) (education data available for 101 participants).

Table 1. Lifetime Prevalence of DSM-IV Axis I Psychiatric Disorders in 103 Obese Women With Binge-Eating Disorder

Diagnosis	N	%
Any Axis I disorder	71	68.9
Any mood disorder	55	53.4
Major depressive disorder	50	48.5
Dysthymic disorder	4	3.9
Depressive disorder NOS	1	1.0
Any anxiety disorder	38	36.9
Panic disorder	18	17.5
Social phobia	9	8.7
Obsessive-compulsive disorder	3	2.9
Posttraumatic stress disorder	6	5.8
Generalized anxiety disorder	5	4.9
Anxiety disorder NOS	12	11.7
Any substance disorder	14	13.6
Alcohol abuse/dependence	11	10.7
Drug abuse/dependence	8	7.8
Somatization disorder	1	1.0
Body dysmorphic disorder	1	1.0
Bulimia nervosa	7	6.8

Abbreviation: NOS = not otherwise specified.

Procedure

Axis I psychiatric disorders, including BED diagnosis, were determined by doctoral-level research-clinicians using the Structured Clinical Interview for DSM-IV Axis I Disorders.[21] BED diagnosis was confirmed with the Eating Disorder Examination Interview.[22] The EDE, a semi-structured interview, focuses on the previous 28 days except for diagnostic items, which are rated for additional duration stipulations. The EDE has well-established interrater and test-retest reliability.[23]

Smoking history was determined via self-report questionnaire. Participants were first asked if they had smoked over 100 cigarettes in their lifetime. Participants who endorsed smoking at least 100 cigarettes were then asked a series of questions assessing the rates of cigarette usage (pack years), age at onset, quit date (year), and whether they gained weight after quitting. Those who endorsed gaining weight were then asked to estimate the number of pounds gained in the first year following their quit date.

RESULTS

Table 1 summarizes the frequencies of lifetime DSM-IV Axis I diagnoses for the overall patient group. Of the 103 patients with BED, 68.9% met criteria for at least one additional lifetime psychiatric disorder; the most common comorbid disorders were mood disorders (53.4%), anxiety disorders (36.9%), and SUDs (13.6%).

Table 2. Smoking Status and Smoking History in 103 Obese Women With Binge-Eating Disorder

Variable	N	%
Smoking status[a]		
Never smokers	52	50.5
Former smokers	45	43.7
Current smokers	6	5.8
Smoker characteristics (N = 51)		
Frequency		
Less than daily	8	15.7
Daily	43	84.3
Gained weight after quit		
No	9	20.0
Yes	22	48.9
Quit due to pregnancy	5	11.1
Unable to estimate	9	20.0
	Mean (SD)	Range
Estimated weight gain, lb	13.9 (12.4)	0–50
Age at first cigarette, y	15.2 (2.6)	10–25
Daily no. of cigarettes (at highest)	17.2 (11.1)	1–50
Time at highest rate, y	9.8 (7.9)	1–30
Time smoked, y	13.6 (8.9)	2–32
Time since quit date, y	15.6 (10.1)	0–35

[a]Smokers were defined as those who reported smoking more than 100 cigarettes during their lifetime.

Table 2 summarizes the findings regarding smoking status and smoking history. Participants who denied smoking more than 100 cigarettes in their entire lifetime were classified as "never" smokers. Lifetime smokers were classified according to smoking history (current vs. former) and according to their reported frequency of use. Those smokers who reported smoking on a "social" or infrequent basis (N = 8) were classified as "less-than-daily" smokers and excluded from analyses. We chose to exclude less-than-daily smokers from the current analyses due to the body of research reporting important differences between daily smokers and "chippers" or social/occasional smokers.[24–27] These results are available from the authors upon request; the results show a similar pattern in rates of comorbid psychopathology, although the significant differences (p values) are attenuated slightly.

Table 3 summarizes the frequencies of co-occurring lifetime psychiatric disorders in the obese women with BED as a function of their smoking status. A series of logistic regression analyses was conducted to test the hypothesis that lifetime daily smokers would differ from never-smokers in terms of lifetime psychiatric diagnosis. Analyses were first conducted comparing never-smokers to those who endorsed smoking on at least a daily basis at some point in their lives. Compared to nonsmokers, lifetime smokers were significantly more likely to have experienced major depressive disorder, anxiety disorders, and SUDs.

A parallel set of analyses, conducted only with those participants who had not reported a history of substance abuse or dependence, is summarized in Table 4. With the patients who had SUDs (N = 14) excluded, the smoking group differed from the nonsmoking group in incidence of panic disorder and anxiety disorder not otherwise specified.

A series of exploratory analyses was conducted to examine the age at onset of Axis I disorders across smoking groups. The smoking groups did not differ significantly in age at onset for BED ($t = 1.05$, $df = 92$, $p = .30$) or age at onset of any comorbid Axis I disorder ($t = 1.60$, $df = 64$, $p = .11$). Limited sample sizes preclude meaningful statistical analyses of the effect of smoking on the age at onset of specific disorders. For descriptive purposes and to generate hypotheses, we summarize these data for specific psychiatric disorders in Figure 1.

DISCUSSION

The goal of this study was to determine whether obese women with BED who had smoked at some point in their lifetime differed from their nonsmoking counterparts in lifetime rates of comorbid psychopathology. Lifetime smokers with BED were significantly more likely than never-smokers with BED to meet criteria for Axis I psychiatric disorders overall, and specifically for major depressive disorder, anxiety disorders, and SUDs. Due to the possibility that the presence of SUD may have confounded the pattern of results, we also compared the groups after removing individuals with lifetime SUD from the analysis. Although attenuated, the results showed a pattern of increased lifetime risk of anxiety disorders among lifetime smokers. These results are particularly interesting when contrasted to the findings of Black et al.,[19] who reported that of the Axis I disorders, only SUDs were independently associated with smoking in a community sample. It may be that the combination of eating disorder with smoking may engender a more complex pattern of comorbid psychopathology.

Overall, the observed rates of DSM-IV Axis I psychiatric disorders are strikingly similar to those reported previously for DSM-III-R–defined disorders in obese BED patient groups also assessed with structured diagnostic interviews.[7,28] For example, our overall lifetime frequencies of any psychiatric disorder (68.9%), major depressive disorder (48.5%), and anxiety disorders (36.9%) are similar to those previously reported by Wilfley et al.[28] (77%, 58%, 29%, respectively), while our rates of SUDs (13.6%) fall between those previously reported by Wilfley et al.[28] (33%) and Yanovski et al.[7] (12%). It is worth noting that for some psychiatric disorders, the base rate in this sample was extremely low (e.g., there were no cases of agoraphobia), and many cases of nonsignificance across smoking groups corresponded with these low base rate diagnoses.

We note potential strengths and limitations in our study to consider as context for interpreting the findings. Our

Table 3. Lifetime Rates of DSM-IV Axis I Psychiatric Disorders in Obese Women With Binge-Eating Disorder, Never Smokers Versus Daily Smokers[a]

	Smoking History							
	Never (N = 52)		Daily (N = 43)					
Lifetime Diagnosis	N	%	N	%	χ^2	p	OR	95% CI
Any Axis I psychiatric disorder	30	57.7	35	81.4	6.32	.01	3.21	1.25 to 8.25
Any mood disorder	24	46.2	28	65.1	3.45	.06	2.18	0.95 to 5.00
Major depressive disorder	21	40.4	27	62.8	4.77	.03	2.49	1.09 to 5.71
Any anxiety disorder	12	23.1	22	51.2	8.15	.01	3.49	1.45 to 8.42
Panic disorder	4	7.7	12	27.9	7.02	.01	4.65	1.37 to 15.71
Social phobia	2	3.8	6	14.0	3.19	.07	4.05	0.77 to 21.23
Posttraumatic stress disorder	1	1.9	5	11.6	3.96	.05[b]	6.71	0.75 to 59.82
Generalized anxiety disorder	3	5.8	1	2.3	0.73	.39	0.39	0.04 to 3.88
Anxiety disorder NOS	2	3.8	8	18.6	5.66	.02	5.71	1.14 to 28.55
Bulimia nervosa	3	5.8	3	7.0	0.06	.81	1.23	0.23 to 6.40
Any substance disorder	3	5.8	11	25.6	7.60	.01	5.62	1.45 to 21.70
Alcohol abuse/dependence	3	5.8	8	18.6	3.85	.05	3.73	0.92 to 15.08
Drug abuse/dependence	1	1.9	7	16.3	6.81	.01	9.92	1.17 to 84.14

[a]For some DSM-IV diagnoses, the base rate in the sample was too low to permit meaningful statistical comparisons; for diagnoses in which there were fewer than 5 (i.e., < .05) cases in the sample, analyses were not conducted.
[a]Value has been rounded to nearest tenth and is significant (p = .047, rounded to .05).
Abbreviations: CI = confidence interval, NOS = not otherwise specified, OR = odds ratio.

Table 4. Lifetime Rates of DSM-IV Axis I Psychiatric Disorders in Obese Women With Binge-Eating Disorder, Never Smokers Versus Daily Smokers: Reanalysis With Substance Use Disorder Exclusion

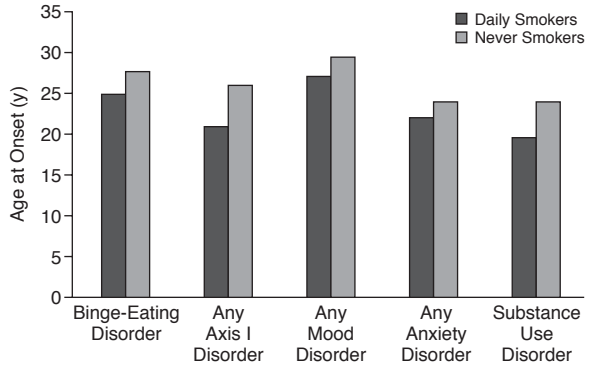
	Smoking History							
	Never (N = 49)		Daily (N = 32)					
Lifetime Diagnosis	N	%	N	%	χ^2	p	OR	95% CI
Any Axis I psychiatric disorder	27	55.1	24	75.0	3.38	.07	2.44	0.92 to 6.50
Any mood disorder	23	46.9	19	59.4	1.20	.27	1.65	0.67 to 4.07
Major depressive disorder	20	40.8	18	56.3	1.86	.17	1.86	0.76 to 4.59
Any anxiety disorder	12	24.5	16	50.0	5.53	.02	3.08	1.19 to 7.98
Panic disorder	4	8.2	10	31.3	7.12	.01	5.11	1.44 to 18.15
Social phobia	2	4.1	4	12.5	1.95	.16	3.36	0.58 to 19.53
Posttraumatic stress disorder	1	2.0	3	9.4	2.19	.14	4.97	0.49 to 50.01
Generalized anxiety disorder	3	6.1	0	0	3.09	.08	…[a]	…[a]
Anxiety disorder NOS	2	4.1	8	25.0	7.85	.01	7.83	1.54 to 39.81
Bulimia nervosa	2	4.1	2	6.3	0.19	.66	1.57	0.21 to 11.73

[a]Rate of diagnosis too low to permit meaningful analysis.
Abbreviations: CI = confidence interval, NOS = not otherwise specified, OR = odds ratio.

study group consisted of a consecutive series of obese women with BED who presented for treatment for binge eating at a medical school. Our findings may not generalize to community or to non–treatment-seeking groups. It is also possible that obese patients with BED who seek alternative forms of treatment (e.g., weight control programs or smoking cessation programs) may have different patterns of comorbidity than our study group. In addition, whether similar patterns would characterize men is uncertain. Finally, it is possible that a larger sample size might have allowed for detection of additional differences in comorbidity for the low base rate diagnoses. Our consecutive sampling and our use of reliably administered structured diagnostic interviews performed by trained and monitored doctoral research clinicians represent notable strengths.

Our findings have potential implications for future investigations of the pathophysiology of binge eating. Future research should investigate potential underlying mechanisms predisposing individuals to both binge eating and smoking. It is possible that a common vulnerability (genetic or environmental) or common neurobiological mechanism underlies both behaviors. Since the smoking literature has indicated that the noradrenergic system is involved in nicotine addiction,[29] it is possible that the noradrenergic system is also involved for some individuals with BED and that these individuals are more prone to develop additional psychopathology. The smoking research has found that cigarettes may be used as an attempt to "self-medicate" in response to negative affect,[30,31] and a similar mechanism may be operating with regard to binge eating. Future research should also explore mediating factors or potential third variables predisposing individuals to both binge eating and smoking, and the potential interconnections with additional psychopathology.

Figure 1. Mean Age at Onset of Axis I Disorders in Smoking Groups[a]



[a]Sample sizes vary for each diagnosis. See Table 3 for specific numbers of cases in each diagnostic category.

The finding that smokers with BED in the sample were more likely to have experienced additional psychopathology than nonsmokers suggests that smoking may be a marker of a psychological vulnerability, or that smoking and binge eating are insufficient coping strategies for negative life events. Although the current study did not employ sufficient sample sizes to explore the temporal ordering of smoking, eating disorder onset, and other Axis I psychopathology, the current data tentatively suggest that smoking may precede the development of both binge eating and additional psychopathology. Direct tests of the temporality of clinical features are clearly needed, with an aim of delineating potential causal pathways. For example, longitudinal studies should investigate the acquisition of negative coping strategies, i.e., assess whether smoking or disordered eating developed first and whether different patterns of onset are associated with meaningful biopsychosocial sequelae. Isensee et al.,[29] for example, examined the temporal ordering (bidirectional nature) of smoking and panic disorder onset. Their results replicated other comorbidity studies reporting a significant association between lifetime panic disorder and cigarette smoking. Importantly, examination of the directionality of the disorders indicated that smoking was associated with subsequent development of panic disorder, whereas preexisting panic was not associated with later smoking behaviors. Similar findings have been reported in longitudinal studies of smoking with agoraphobia, generalized anxiety disorder, and panic.[24,32]

A review of smoking among adolescents indicates that smoking may exacerbate or serve as a marker for the development of psychiatric disorders.[33] The current cross-sectional study found that among patients with BED, history of smoking is associated with increased risk of comorbid psychopathology, but did not test whether smoking is associated with increased risk of binge eating. A prospective longitudinal study found that smoking in adolescence is associated with increased risk for anxiety disorders.[34] Prospective studies on smoking and binge eating, involving larger psychiatric or community samples, should explore this hypothesis. Additional research is needed to test whether the relationship between smoking, binge eating, and additional psychopathology is unique to binge eating. We cautiously note that our exploratory analyses suggest that the age at onset of particular disorders may be slightly earlier for smokers than for never-smokers; future research is required to empirically test this hypothesis.

Clinically, the results of this study indicate that women with BED and a history of smoking may be especially distressed, with higher rates of comorbid lifetime psychiatric diagnosis. These patients may have a deficit in coping strategies, making them more prone to using substances and/or food to regulate emotion. There is a great need for treatments to address multiple impulse control behaviors[10] as clinicians and patients struggle with important unanswered questions around treatment formulation and planning.[35]

Disclosure of off-label usage: The authors of this article have determined that, to the best of their knowledge, no investigational information about pharmaceutical agents that is outside U.S. Food and Drug Administration–approved labeling has been presented in this article.

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