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The Metabolic Syndrome and Behavioral Correlates in Obese Patients With Binge Eating Disorder

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Abstract

This study examined the frequency of the metabolic syndrome (MetSyn) and explored behavioral eating- and weight-related correlates in obese patients with binge eating disorder (BED). Ninety-three treatment-seeking obese BED patients (22 men and 71 women) with and without the MetSyn were compared on demographic features and a number of current and historical eating and weight variables. Sixty percent of the obese patients with BED met criteria for the MetSyn, with men and whites having significantly higher rates than women and African Americans, respectively. Patients with vs. without coexisting MetSyn did not differ significantly in self-reported frequency of binge eating or severity of eating disorder psychopathology. Multivariate hierarchical logistic regression analysis revealed that, after controlling for gender, ethnicity, and BMI, fewer episodes of weight cycling and regular meal skipping were significant predictors of the MetSyn. These findings suggest that lifestyle behaviors including weight loss attempts and regular meal consumption may be potential targets for prevention and/or treatment of the MetSyn in obese patients with BED.

INTRODUCTION

The metabolic syndrome (MetSyn), a cluster of symptoms including central adiposity, hypertriglyceridemia, hypertension, low levels of high-density lipoprotein (HDL) cholesterol, and elevated fasting plasma glucose levels, is associated with increased risk for heart disease (1), type 2 diabetes mellitus (2), and cardiovascular and all-cause mortality (3). Prevalence rates of the MetSyn in US adults range from ~25% of the general population (4) to 50% of obese women and 59.6% of obese men (5). While the pathogenic process involved in the MetSyn is poorly understood, insulin resistance has been identified as a central underlying mechanism (6). Lifestyle behaviors such as physical inactivity and smoking (7) and psychological factors, including anger and depression (8,9) have also been associated with the MetSyn.

Binge eating disorder (BED), which is characterized by recurrent episodes of binge eating (eating an unusually large amount of food and feeling a loss of control) in the absence of compensatory behaviors, occurs in a subset of obese individuals and is related to increased severity of obesity (10). In addition to binge eating, BED is associated with several problematic eating behaviors and patterns that distinguish obese binge eaters from nonbinge eating obese individuals (11), including frequent eating throughout the day and night (11,12) and

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maladaptive dieting behavior (13) such as meal skipping (14). Little is known about the MetSyn in obese patients with BED, but laboratory studies suggest that eating behaviors commonly found in BED patients may increase the risk for metabolic abnormalities. For example, increased eating rate has been associated with central adiposity, elevated serum lipids, and fatty liver in obese men and women (15). Furthermore, eating meals irregularly was positively associated with the MetSyn in a population-based, cross-sectional study (16). Metabolic abnormalities have even been observed in healthy, lean women following laboratory-based binge-like episodes, suggesting that metabolic dysfunction may be even more pronounced in obese individuals who binge eat and consume excessive calories on a *regular basis* (17).

Given these findings, the dearth of studies on the MetSyn in obese patients with BED is striking. To our knowledge, only one published study has examined the frequency of the MetSyn in obese patients with BED (18). Guerdjikova *et al.* (18) identified the MetSyn in 32% of weight loss treatment-seeking, obese patients with BED. Additional investigations are clearly needed as this reported rate is substantially *lower* than the ~60% reported in a population-based study of moderately obese adults in the United States (5). Moreover, to our knowledge, no studies have examined behavioral correlates associated with the MetSyn in obese patients with BED, and it is unclear whether binge eating severity and/or other maladaptive eating behaviors commonly observed in this patient group are associated with the MetSyn. Accordingly, the current study aimed to examine the frequency of the MetSyn in obese, treatment-seeking patients with BED as well as potential demographic and eating- and weight-related behavioral correlates of the MetSyn in this patient population.

METHODS AND PROCEDURES

Participants

Participants were 93 (22 men; 71 women) treatment-seeking, obese adults who met DSM-IV-TR (19) diagnostic research criteria for BED. Participants were recruited via advertisements seeking overweight persons with binge eating problems who wanted to “stop binge eating and lose weight.” Data for the current study were obtained from participants being assessed for studies evaluating behavioral weight loss or cognitive-behavioral interventions for binge eating. Participants ranged in age from 19 to 59 (M = 45.42, s.d. = 8.9); 76.3% were white (N = 71), 15.1% African American (N = 14), 4.3% Hispanic (N = 4), 3.2% Asian (N = 3), and 1.1% (N = 1) unknown. BMI (weight (kg) divided by height (m^2)) ranged from 26.34 to 53.49 (M = 37.66, s.d. = 5.5).

Assessments and measures

Physical and metabolic functioning—Fasting lipid profile (total cholesterol, HDL, low-density lipoprotein, and triglycerides), and glucose levels were obtained and analyzed by Quest Diagnostics (Madison, NJ). Height, weight, waist circumference, and blood pressure were measured by trained research staff.

Eating Disorder Examination—The Eating Disorder Examination (EDE) (20), a semi-structured investigator-based interview that assesses the psychopathology of eating disorders, was used to assess binge eating and associated features. The EDE focuses on the previous 28 days, except for diagnostic items that are rated for duration stipulations of the DSM-IV-TR (19). The EDE assesses the frequency of *objective bulimic episodes* based on DSM-IV-TR criteria (19), which refer to binge eating defined as consuming unusually large quantities of food *with* a subjective sense of loss of control. To measure eating patterns, the EDE assesses the frequency of meals (breakfast, lunch, and dinner), snacks (mid-morning, mid-afternoon, and evening) and nocturnal eating (eating after having gone to sleep) over the past 28 days. The response set is based on a 7-point scale, ranging between 0 (reflecting no consumption of

the meal/snack) and 7 (reflecting daily consumption of the meal/snack). A 3 denotes having consumed the meal or snack 13−15 days over the past month.

The EDE also comprises four subscales: Restraint, Eating Concern, Shape Concern, and Weight Concern. Since the present study focuses on eating behavior and correlates, we did not include the Weight or Shape Concern subscales in our analyses but focused instead on the Restraint and Eating Concern subscales of the EDE. The Restraint subscale reflects attempts to restrict food intake to influence weight or shape; the Eating Concern subscale reflects the degree of concern about eating. These items are rated on a 7-point forced-choice format (0−6), with higher scores reflecting greater severity or frequency. The EDE is considered the best established method for assessing the features of eating disorders and has received support for its utility in assessing BED (21,22). Psychometric studies of the EDE have demonstrated its validity and inter-rater and test–retest reliability in diverse groups including obese patients with BED (23), although recent studies have found that the restraint scale is not correlated with actual caloric intake (24).

Three Factor Eating Questionnaire—The TFEQ (25) consists of 51 items that assess three factors: disinhibition, hunger, and cognitive restraint. The TFEQ-cognitive restraint scale and EDE-Restraint scale have been found to measure different aspects of dietary restraint (i.e., dieting to avoid weight gain and dieting to lose weight, respectively) (26) and was therefore included to examine both of these dimensions of dietary restraint. The TFEQ has received some psychometric support including good internal consistency (25) and predictive validity (27), although recent studies have found that TFEQ-Restraint scores are not correlated with actual caloric intake (24,28).

Questionnaire for Eating and Weight Patterns-Revised—The Questionnaire for Eating and Weight Patterns-Revised (29), used in the DSM-IV field trials, assesses each criterion for BED. The Questionnaire for Eating and Weight Patterns-Revised also assesses historical variables relevant to the current study, including demographic variables; age of onset for binge eating, dieting, and obesity; and weight cycling history (defined as an intentional weight loss of ≥20 lb followed by subsequent weight regain).

Procedures and statistical analyses

Assessments were administered by experienced doctoral level research-clinicians who were trained and monitored. Patients were categorized as having the MetSyn if they had three or more of the five criteria outlined by the National Cholesterol Education Program's Adult Treatment Panel III guidelines (30): *abdominal obesity*: waist circumference >40 inches (102 cm) in men and >35 inches (88 cm) in women; *hypertrigylceridemia*: ≥150 mg/dl (1.7 mmol/l); *low HDL cholesterol*: <40 mg/dl (1.03 mmol/l) in men and <50 mg/dl (1.30 mmol/l) in women; *hypertension*: ≥130 mm Hg systolic or ≥85 mm Hg diastolic; *high fasting glucose*: ≥110 mg/dl (6.1 mmol/l). BED research diagnoses were determined using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) (31) and confirmed by findings from the EDE. Regular meal skipping was defined as consumption of any meal less than or equal to half of the time (i.e., EDE score ≤3), and participants were dichotomized as regular meal skippers or nonskippers.

Participants with and without the MetSyn were compared on baseline characteristics using *t*-tests for continuous variables and χ^2 analyses for categorical variables. Analysis of covariances were used to examine group differences on historical and behavioral correlates while controlling for BMI, gender, and ethnicity. Effect sizes were included for these analyses. The significant univariate findings were entered into a multivariate hierarchical logistic regression,

and odds ratios for the MetSyn were then generated. All analyses were conducted using SPSS 15.0 (Chicago, IL).

RESULTS

Descriptive characteristics

Fifty-six (60.2%) of the 93 patients met Adult Treatment Panel III criteria for the MetSyn. As summarized in Table 1, significant gender and ethnic differences emerged with men and whites having higher rates of the syndrome than women and African Americans. There were no differences in age or education levels between the two groups. As expected, patients with the MetSyn differed significantly from those without the syndrome on weight, BMI, waist circumference, HDL cholesterol, triglycerides, blood pressure, and fasting glucose. No differences emerged between the two groups on low-density lipoprotein or non-HDL cholesterol levels.

Eating and weight history

Table 2 presents the mean estimates and s.e. for eating and weight history variables in patients with and without the MetSyn. Analysis of covariance revealed that after controlling for BMI, gender, and ethnicity, patients with the MetSyn had significantly fewer episodes of weight cycling, F (1, 86) = 9.98, $P < 0.05$, partial $\eta^2 = 0.10$, than those without the MetSyn. No significant differences were found between the two groups on age of onset for obesity, binge eating, dieting, or highest or lowest adult BMI levels.

Current eating behavior

Table 3 summarizes current eating behavior in patients with and without the MetSyn. A χ^2 analysis revealed a significant association between meal skipping and the MetSyn, χ^2 (1) = 4.11, $P < 0.05$. Seventy-five percent ($N = 24$) of patients who were regular meal skippers met criteria for the MetSyn. In contrast, 53.3% ($N = 32$) of nonmeal skippers met criteria for the MetSyn. After controlling for BMI, gender, and ethnicity, no significant differences emerged between those with and without the MetSyn on current binge eating episodes or severity of eating disorder psychopathology.

Regression analyses

To explore the incremental effect of the statistically significant behavioral variables (weight cycling and meal skipping) over and above the significant demographic (gender and ethnicity) and BMI variables, a multivariate hierarchical logistic regression was conducted to predict the presence or absence of the MetSyn. Demographic variables (gender, ethnicity, and BMI) were entered in the first step, weight cycling in the second step, and meal skipping in the third step. At each step of the analysis, all predictor variables were significant. As Table 4 indicates, the significant association between regular meal skipping and the MetSyn remained after controlling for gender, BMI, ethnicity, and weight cycling history (odds ratio = 4.31, 95% confidence interval = 1.08−17.19, $P = 0.04$).

DISCUSSION

This study examined the frequency of the MetSyn and investigated potential demographic and eating- and weight-related behavioral correlates in a series of treatment-seeking, obese patients with BED. Our findings suggest that the MetSyn is common in this specific obese patient group, with ~60% of the sample meeting Adult Treatment Panel III diagnostic criteria. This rate is higher than the 32% found in a previous study of obese, BED patients (18), but is consistent with rates reported in a population-based sample of obese individuals (5). Also consistent with

previous findings from population-based studies, we found significant gender differences, with men having higher rates of the MetSyn than women (4,5); however, our findings indicate that the rates of the MetSyn in obese BED men (90.2%) are much greater than those found in obese, non-BED men in population-based (59.6%) (5) and treatment-seeking (60%) (32) samples. These differences were not found in women, and rates of the MetSyn in women (50.7%) were comparable to those reported previously in obese, non-BED women (5). Our results also suggest that whites have higher rates of the MetSyn than African Americans among obese patients with BED. Although these analyses were limited due to small numbers, this significant difference is noteworthy and is consistent with findings from population-based reports (4,5). While previous studies have identified associations between older age and the MetSyn in population-based (4,5) and treatment-seeking obese, non-BED individuals (32), we found no significant differences in age between obese BED patients with and without the MetSyn.

We also examined differences on historical and current eating- and weight-related variables in individuals with and without the MetSyn while controlling for current BMI, gender, and ethnicity. Consistent with previous reports from a population-based sample (16), regular meal skipping was associated with higher rates of the MetSyn in obese patients with BED. Our findings also suggest that individuals with the MetSyn had fewer episodes of weight cycling than patients without the MetSyn but did not differ on any other historical variables. Notably, no differences were found between the groups on binge eating frequency or severity of eating disorder psychopathology.

Findings from a multivariate hierarchical logistic regression analysis suggest that even after controlling for differences in gender, BMI, and ethnicity, weight cycling and meal skipping continued to be significant predictors of the MetSyn in obese patients with BED. Our finding that regular meal skipping is associated with the MetSyn is particularly noteworthy given that this is a modifiable behavior that could routinely be advised to patients. Not only does regular meal consumption appear to be significantly associated with the MetSyn and other cardiovascular disease risk, but it also is a crucial component of cognitive-behavioral treatment for BED (33). Moreover, obese BED patients who consume three meals per day have been found to weigh less and have fewer binge episodes than those who do not (14).

While our findings also suggest that more frequent episodes of weight cycling are associated with lower rates of the MetSyn, these findings should be interpreted cautiously. There is considerable controversy surrounding the health implications of weight cycling, and evidence linking weight cycling to cardiovascular disease and mortality rates has been mixed (34,35). It has been suggested that the conflicting findings reported in the literature can be largely attributed to differences in the measurement of weight cycling as well as not distinguishing between unintentional and intentional weight loss (36). In the current study, weight cycling was defined as episodes of intentional weight loss of ≥20 lb followed by weight regain and was based on self-report. Our findings provide preliminary evidence suggesting that more frequent episodes of intentional weight loss (followed by eventual weight regain) may be associated with lower rates of the MetSyn in obese patients with BED. Additional research is needed to replicate and extend our findings to better understand the relationship between weight cycling and the MetSyn in obese patients with and without BED.

This study has several strengths, including the use of a semi-structured diagnostic and investigator-based clinical interview to determine BED diagnosis and to assess current eating patterns, the inclusion of men and women, and the use of well-validated measures. Several limitations, however, should be noted. Our study group consisted of nondiabetic, treatment-seeking, obese patients with BED, and future research is needed to extend our findings to other groups (e.g., nontreatment-seeking BED, treatment-seeking obese patients without BED, and community samples). The reliance on self-reported eating behaviors, both through

interviewing and paper-and-pencil methods, is also a limitation. Although the EDE method for determining binge eating is generally thought to be reliable, research has found that current measures of restraint (i.e., EDE restraint and TFEQ-Restraint) are not correlated with actual recent eating behaviors (24). In addition, the sample size was relatively small, and power may have limited our ability to detect small differences between the groups. We considered and followed recommendations to address such well-known power limitations in behavioral studies (37,38). First, we focused on a limited number of demographic (gender, ethnicity, and BMI) and behavioral (binge eating and meal irregularity) variables supported in the literature and augmented this exploratory (hypothesis generating) study by focusing on clinically relevant eating- and weight-related behaviors. Second, the variables selected for inclusion in the omnibus multiple regression analyses were (i) statistically significant in the univariate analyses, and (ii) not highly correlated (*r*s ranged from 0.02 to 0.31). Third, we supplemented our traditional significance tests with effect size measures and confidence intervals. Lastly, small numbers of Hispanic and Asian American patients in our sample prohibited us from examining rates of the MetSyn in these specific ethnic groups, although our findings for African-American and white groups parallel the literature. Future research should address these limitations with larger and diverse samples ideally in prospective designs.

In summary, our findings suggest that roughly 60% of obese patients with BED meet Adult Treatment Panel III criteria for the MetSyn, with men and whites having higher rates than women and African Americans. Frequency of binge eating episodes and severity of eating disorder psychopathology did not differ significantly between those with and without the MetSyn; however, fewer episodes of weight cycling and regular meal skipping were significant predictors of the MetSyn. These findings suggest that advising patients to lose weight and to consume regular meals may have important implications for prevention and/or treatment of the MetSyn in obese patients with BED.

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REFERENCES

1. McNeill AM, Rosamond WD, Girman CJ, et al. The metabolic syndrome and 11-year risk of incident cardiovascular disease in the Atherosclerosis Risk in Communities Study. Diabetes Care 2005;28:385–390. [PubMed: 15677797]
2. Meigs JB, Wilson PWF, Fox CS, et al. Body mass index, metabolic syndrome, and risk of type 2 diabetes or cardiovascular disease. J Clin Endocrinol Metab 2006;91:2906–2912. [PubMed: 16735483]
3. Malik S, Wong ND, Franklin SS, et al. Impact of the metabolic syndrome on mortality from coronary heart disease, cardiovascular disease, and all causes in United States Adults. Circulation 2004;110:1245–1250. [PubMed: 15326067]
4. Ford ES, Giles WH, Dietz WH. Prevalence of the metabolic syndrome among US adults: findings from the Third National Health and Nutrition Examination Survey. JAMA 2002;287:356–359. [PubMed: 11790215]
5. Park Y, Zhu S, Palaniappan L, et al. The metabolic syndrome: prevalence and associated risk factor findings in the US population from the Third National Health and Nutrition Examination Survey, 1988−1994. Arch Intern Med 2003;163:427–436. [PubMed: 12588201]
6. Grundy SM. Hypertriglyceridemia, insulin resistance, and the metabolic syndrome. Am J Cardiol 1999;83:25F–29F.
7. Zhu S, St-Onge M, Heshka S, Heymsfield SB. Lifestyle behaviors associated with lower risk of having the metabolic syndrome. Metabolism 2004;53:1503–1511. [PubMed: 15536610]

8. Kinder LS, Carnethon MR, Palaniappan LP, King AC, Fortmann SP. Depression and the metabolic syndrome in young adults: findings from the Third National Health and Nutrition Examination Survey. Psychosom Med 2004;66:316–322. [PubMed: 15184689]
9. Raikkonen K, Matthews KA, Kuller LH. The relationship between psychological risk attributes and the metabolic syndrome in healthy women: antecedent or consequence? Metabolism 2002;51:1573–1577. [PubMed: 12489070]
10. Hudson JI, Hiripi E, Pope J, Harrison G, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. Biol Psychiatry 2007;61:348–358. [PubMed: 16815322]
11. Allison KC, Grilo CM, Masheb RM, Stunkard AJ. Binge eating disorder and night eating syndrome: a comparative study of disordered eating. J Consult Clin Psychol 2005;73:1107–1115. [PubMed: 16392984]
12. Grilo CM, Masheb RM. Night-time eating in men and women with binge eating disorder. Beh Res Ther 2004;42:397–407.
13. Masheb RM, Grilo CM. On the relation of flexible and rigid control of eating to body mass index and overeating in patients with binge eating disorder. Int J Eat Disord 2002;31:82–91. [PubMed: 11835301]
14. Masheb RM, Grilo CM. Eating patterns and breakfast consumption in obese patients with binge eating disorder. Behav Res Ther 2006;44:1545–1553. [PubMed: 16376851]
15. Kral JG, Buckley MC, Kissileff HR, Schaffner F. Metabolic correlates of eating behavior in severe obesity. Int J Obes Relat Metab Disord 2001;25:258–264. [PubMed: 11410829]
16. Sierra-Johnson J, Undén AL, Linestrand M, et al. Eating meals irregularly: a novel environmental risk factor for the metabolic syndrome. Obesity (Silver Spring) 2008;16:1302–1307. [PubMed: 18388902]
17. Taylor AE, Hubbard J, Anderson EJ. Impact of binge eating on metabolic and leptin dynamics in normal young women. J Clin Endocrinol Metab 1999;84:428–434. [PubMed: 10022396]
18. Guerdjikova AI, McElroy SL, Kotwal R, Keck PE Jr. Comparison of obese men and women with binge eating disorder seeking weight management. Eat Weight Disord 2007;12:e19–e23. [PubMed: 17384521]
19. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Vol. 4th edn. American Psychiatric Association; Washington, DC: 2000. Text Revision
20. Fairburn, CG.; Cooper, Z. The eating disorder examination.. In: Fairburn, CG.; Wilson, GT., editors. Binge Eating: Nature, Assessment, and Treatment. Vol. 12th edn. Guilford Press; New York: 1993. p. 317-360.
21. Grilo CM, Masheb RM, Lozano-Blanco C, Barry DI. Reliability of the Eating Disorder Examination in patients with binge eating disorder. Int J Eat Disord 2004;35:80–85. [PubMed: 14705160]
22. Grilo CM, Masheb RM, Wilson GT. A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. J Consult Clin Psych 2001;69:317.
23. Grilo CM, Lozano C, Elder KA. Inter-rater and test–retest reliability of the Spanish language version of the Eating Disorder Examination interview: clinical and research implications. J Psych Pract 2005;11:231–240.
24. Sysko R, Timothy Walsh B, Terence Wilson G. Expectancies, dietary restraint, and test meal intake among undergraduate women. Appetite 2007;49:30–37. [PubMed: 17188400]
25. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res 1985;29:71–83. [PubMed: 3981480]
26. Safer DL, Agras WS, Lowe MR, Bryson Susan. Comparing two measures of eating restraint in bulimic women treated with cognitive-behavioral therapy. Int J Eat Disord 2004;36:83–88. [PubMed: 15185276]
27. Foster GD, Wadden TA, Swain RM, et al. The Eating Inventory in obese women: clinical correlates and relationship to weight loss. Int J Obes Relat Metab Disord 1998;22:778. [PubMed: 9725638]
28. Stice E, Fisher M, Lowe MR. Are dietary restraint scales valid measures of acute dietary restriction? Unobtrusive observational data suggest not. Psychol Assess 2004;16:51–59. [PubMed: 15023092]
29. Yanovski S. Binge eating disorder: current knowledge and future directions. Obes Res 1993;1:306–324. [PubMed: 16350580]

30. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001;285:2486–2497. [PubMed: 11368702]

31. First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for DSM-IV Axis I Disorders: Patient Edition (SCID-I/P, Version 2.0). New York State Psychiatric Institute; New York: 1996.

32. Tsai AG, Wadden TA, Sarwer DB, et al. Metabolic syndrome and health-related quality of life in obese individuals seeking weight reduction. Obesity (Silver Spring) 2008;16:59–63. [PubMed: 18223613]

33. Fairburn, CG.; Marcus, MD.; Wilson, GT. Cognitive-behavioral therapy for binge eating and bulimia nervosa: a comprehensive treatment manual.. In: Fairburn, CG.; Wilson, GT., editors. Binge Eating: Nature, Assessment, and Treatment. Guilford Press; New York: 1993. p. 361-404.

34. Brownell KD, Rodin J. Medical, metabolic, and psychological effects of weight cycling. Arch Intern Med 1994;154:1325–1330. [PubMed: 8002684]

35. Wing RR, Jeffery RW, Hellerstedt WL. A prospective study of effects of weight cycling on cardiovascular risk factors. Arch Intern Med 1995;155:1416–1422. [PubMed: 7794091]

36. Williamson DF. “Weight cycling” and mortality: how do the epidemiologists explain the role of intentional weight loss? J Am Coll Nutr 1996;15:6–13. [PubMed: 8632117]

37. Jaccard J, Guilamo-Ramos V, Johansson M, Bouris A. Multiple regression analyses in clinical child and adolescent psychology. J Clin Child Adol Psych 2006;35:456–479.

38. Maxwell SE. Sample size and multiple regression analysis. Psych Meth 2000;5:434–458.

Table 1

Baseline characteristics of treatment-seeking binge eating disorder patients with and without the metabolic syndrome

	No metabolic syndrome (n = 37)	Metabolic syndrome (n = 56)	P value	Effect size[a]
Gender, number (%)				
Women	35 (94.6)	36 (64.3)	0.001	0.35
Men	2 (5.4)	20 (35.7)		
Ethnicity[b], number (%)				
Black	9 (27.3)	5 (9.6)	0.03	0.23
White	24 (72.7)	47 (90.4)		
Education, number (%)				
Some HS	1 (2.7)	0 (0)	0.42	0.18
HS or GED	3 (8.1)	9 (16.1)		
Some college	11 (29.7)	18 (32.1)		
College graduate/postgraduate	21 (56.8)	28 (50)		
Unknown	1 (2.7)	1 (1.8)		
Age	46.36 (8.47)	44.82 (9.20)	0.42	0.17
BMI (kg/m^2)	35.81 (5.51)	38.82 (5.22)	0.01	0.56
Waist circumference (in)	41.66 (5.93)	46.46 (5.99)	0.002	0.81
LDL cholesterol (mg/dl)	128.51 (31.35)	119.26 (28.9)	0.15	0.31
Non-HDL cholesterol (mg/dl)	147.65 (33.53)	154.88 (31.41)	0.29	0.22
HDL cholesterol (mg/dl)	62.59 (11.27)	42.10 (9.71)	<0.001	1.95
Triglycerides (mg/dl)	92.49 (36.18)	190.26 (120.11)	<0.001	1.10
Systolic blood pressure (mm Hg)	130.16 (16.30)	146.22 (15.83)	<0.001	1.0
Diastolic blood pressure (mm Hg)	78.97 (10.24)	92.11 (11.46)	<0.001	1.21
Glucose (mg/dl)	91.82 (10.52)	106.72 (15.93)	<0.001	1.10

Values are means and s.d. based on t-tests for continuous variables and χ^2 analyses for categorical variables.

GED, General Educational Development; HDL, high-density lipoprotein; HS, high school; LDL, low-density lipoprotein.

[a] Effect sizes are reported as Φ for categorical variables and Cohen's d for continuous variables.

[b] Ethnicity analyses contain data only from black and white patients due to small numbers of other ethnic groups in this sample.

Table 2

Eating and weight history variables in treatment-seeking binge eating disorder patients with and without the metabolic syndrome

	No metabolic syndrome (n = 37)	Metabolic syndrome (n = 56)	P value	Partial η^2
Age of obesity onset	12.91 (1.55)	16.43 (1.21)	0.10	0.03
Age of binge eating onset	22.74 (2.11)	24.18 (1.62)	0.61	0.003
Age of dieting onset	17.93 (1.50)	20.38 (1.15)	0.23	0.02
Weight cycling	3.58 (0.16)	2.90 (0.12)	0.002	0.10
Highest adult BMI	41.06 (1.13)	39.35 (0.87)	0.26	0.02
Lowest adult BMI	23.71 (0.68)	24.09 (0.52)	0.68	0.002

Values are mean estimates and s.e. based on analysis of covariance that controlled for BMI, gender, and ethnicity.

Table 3

Current eating behavior variables in treatment-seeking obese binge eating disorder patients with and without the metabolic syndrome

	No metabolic syndrome (n = 37)	Metabolic syndrome (n = 56)	P value	Effect size[a]
EDE-Restraint	1.94 (0.22)	1.62 (0.17)	0.28	0.01
EDE-Eating Concerns	1.97 (0.24)	2.03 (0.18)	0.85	0.000
TFEQ-Restraint	6.89 (0.54)	7.79 (0.42)	0.22	0.02
TFEQ-Disinhibition	14.22 (0.47)	13.61 (0.36)	0.34	0.01
TFEQ-Hunger	10.32 (0.63)	10.59 (0.48)	0.75	0.001
OBE in past month	16.52 (1.52)	15.53 (1.17)	0.63	0.003
Meal skipping, number, (% yes)	8 (21.6)	24 (42.9)	0.04	0.21

Values are mean estimates and s.e. for continuous variables obtained through analysis of covariances (ANCOVAs) in which BMI, gender, and ethnicity were covariates.

Categorical variables are based on numbers and percentage values obtained through χ^2 analyses.

EDE, Eating Disorders Examination; OBE, Objective Bulimic Episodes; TFEQ, Three Factor Eating Questionnaire.

[a] Effect sizes are reported as partial η^2 for ANCOVAs and Φ for χ^2 analyses.

Table 4

Odds ratios associated with demographic, weight cycling, and meal skipping variables in the prediction of the metabolic syndrome

Variable	Odds ratios	95% CI	*P* value
Full model			
Gender	29.55	3.03−287.99	0.004
Ethnicity[a]	0.04	0.005−0.38	0.005
BMI	1.19	1.04−1.35	0.009
Weight cycling	0.38	0.17−0.83	0.016
Meal skipping	4.31	1.08−17.19	0.038

Values based on hierarchical logistic regression analyses with demographic variables entered in the first step, weight cycling in the second step, and meal skipping in the third step.

CI, confidence interval.

[a]Ethnicity analyses contain data only from black and white patients due to small numbers of other ethnic groups in this sample.