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Metabolic Syndrome in Obese Men and Women with Binge Eating Disorder: Developmental Trajectories of Eating and Weight-Related Behaviors

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Abstract

The metabolic syndrome (MetSyn), characterized by vascular symptoms, is strongly correlated with obesity, weight-related medical diseases and mortality, and has increased commensurately with secular increases in obesity in the U.S. Little is known about the distribution of MetSynin obese patients with binge eating disorder (BED) or its associations with different developmental trajectories of dieting, binge eating, and obesity problems. Further, inconsistencies in the limited data necessitate elucidation. This study examined the frequency and correlates of MetSyn in a consecutive series of 148 treatment-seeking obese men and women with BED assessed with structured clinical interviews. Almost half of the participants met criteria for MetSyn. Participants with MetSyn did not differ from those without MetSyn on demographic variables or disordered eating psychopathology. However, our findings suggest that MetSyn is associated with a distinct developmental trajectory, specifically a later age at BED onset and shorter BED duration. Although the findings from this study shed some light on MetSyn and its associations with developmental trajectories of eating and weight-related behaviors, notable inconsistencies characterize the limited literature. Prospective studies are needed to examine causal connections in the development of the MetSyn in relation to disordered eating in addition to excess weight.

Obesity has reached epidemic proportions, with more than one-third of U.S. adults considered obese (BMI ≥ 30 kg/m^2) [1]. The level of obesity is directly related to metabolic syndrome (MetSyn), a group of vascular risk factors which increase the risk of cardiovascular disease, including hypertension, elevated fasting blood glucose, central adiposity, hypertriglyceridemia, and low serum high-density lipoprotein (HDL) cholesterol level [2, 3]. MetSyn is a significant risk factor for cardiovascular disease, type II diabetes, all-cause mortality [4] and a growing economic health-care burden in the United States [5]. MetSyn may also be related to health related quality of life (HRQOL) [6], although this is equivocal [5].

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The prevalence of MetSyn has increased with the increasing rates of obesity. Estimates indicate that 24% of U.S. population has MetSyn [7]. The rates of MetSyn among obese individuals are much higher, with 50% of women and nearly 60% of men meeting criteria [8]. There also appears to be significant ethnic differences in the prevalence of MetSyn with Hispanic American women having the highest rates (35.6%) and Caucasian women having the lowest rates (22.8%) [7].

Obesity is a multifaceted problem with complex causes [9]. An important subgroup of obese persons are those who have binge eating disorder (BED) [10]. BED is characterized by recurrent episodes of binge eating (consuming unusually large amounts of food while experiencing a subjective loss of control) without inappropriate compensatory behaviors. BED is associated with severity of obesity, heightened risk for psychiatric symptoms, as well as elevated levels of self-reported medical symptoms [10]. Obese individuals with BED differ significantly from their non-BED counterparts in their non-binge eating patterns and behaviors [11].

Despite the relationship between obesity and BED, little is known about the association of specific eating behaviors and patterns characteristic of BED to MetSyn. Existing literature suggests that certain eating behaviors associated with metabolic abnormalities are similar to some of the features characteristic of BED. For example, eating large amounts of food in a discreet period of time is associated with exaggerated insulin secretion, increased fasting glucose levels, decreased glucose tolerance, and elevated serum lipids [12, 13]. Eating rapidly is associated with elevated serum lipids, higher waist-hip circumference ratio, and fatty liver in obese individuals [14]. Additionally, irregular meal patterns are associated with MetSyn in the general population [15].

To our knowledge, only four published studies have examined MetSyn in obese persons with BED. The first study [16] compared obese men and women with BED seeking weight loss treatment and reported that 32% of participants met criteria for MetSyn. However, this study observed rates of MetSyn much lower than those typically reported in obese populations (50–60%) and did not examine correlates of the MetSyn [8]. Another study followed overweight and obese individuals with and without BED over a 5-year period and assessed self-reports about components of MetSyn [17]. Individuals with BED, compared to those without BED, were significantly more likely to self-report new diagnoses of MetSyn components (e.g., dyslipidemia, hypertension) at follow-up. Hudson and colleagues [17] concluded that BED may confer added risk for metabolic irregularities independent of weight.

In an examination of obese patients seeking treatment for BED at a university-based research clinic, 60% met criteria for MetSyn [18]. MetSyn was significantly associated with ethnicity, BMI, fewer episodes of weight cycling, and meal skipping [18]. Most recently, the fourth study reported that 43% of patients recruited from primary care centers for BED and weight loss treatment met criteria for MetSyn [19]. Patients without MetSyn started dieting at a significantly younger age, spent more of their adult lives dieting, and reported more current dietary restriction than patients with MetSyn. It is unclear if the differences between Roehrig et al. [18] and Barnes et al. [19] were due to small samples sizes, particularly of men, or because participants were recruited from different sources (i.e., specialty clinic vs. primary care centers).

Due to the limited and inconsistent research on metabolic abnormalities in obese persons with BED, more research is needed to understand MetSyn in BED. The current study explores the timing of binge eating, diet, and overweight onset to elucidate potential developmental correlates of BED with MetSyn. Research has found distinct developmental

trajectories of BED that suggest potentially important clinical implications for an earlier age at binge onset [20–22]. Individuals who reported an earlier age at binge onset also reported a binge-first (versus diet-first) developmental trajectory [20–22], a younger age when first met full BED criteria [21, 22], and younger overweight onset [21, 22]. The current study sought to replicate and extend previous research with a larger sample of both women *and* men with BED by investigating the frequency of MetSyn and its potential association with eating and weight-related pathology and BED developmental trajectories.

METHODS

Participants

Participants were 148 consecutively evaluated, treatment-seeking obese individuals who met full *DSM-IV* research diagnostic criteria for BED. Participants were recruited via newspaper advertisements seeking obese men and women who eat "out of control" and "want to lose weight" for treatment studies at a medical school-based specialty clinic. Inclusion criteria were a BMI of 30–55 (kg/m^2) and a *DSM-IV* research diagnosis of binge eating disorder (BED). Exclusion criteria were pregnancy or breastfeeding, uncontrolled hypertension, significant cardiovascular disease, coronary arterial disease, significant neurological history, regular use of purging behaviors, severe psychiatric disorders (e.g., bipolar disorder, schizophrenia, substance dependence), and current use of anti depressants. Participants were aged 21 to 65 years ($M = 48.2$, $SD = 8.8$) and 71.6% (n=106) were women and 28.4% (n=42) were men. Participants were 78.4% (n=115) Caucasian, non-Hispanic, 14.2% (n=21) African-American/Black, non-Hispanic, 3.4% (n=5) Caucasian, Hispanic, 0.7% Asian (n=1), and 3.4% (n=5) other or of mixed race. Educationally, 0.7% (n=1) reported some grammar and junior high school, 2.0% (n=3) some high school, 15.5% (n=23) high school or GED, 35.1% (n=52) some college or associates degree, 45.3%(n=67) college, and 1.4% (n=2) unknown. Participants' mean body mass index (BMI) was 39.6 (SD=6.0).

Assessment and Measures

The study received full review and approval by the Yale Human Investigation Committee and all participants provided informed-written consents. Assessment procedures were performed by trained doctoral-level research-clinicians. BED diagnosis was based on the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I/P) [23] and confirmed with the Eating Disorder Examination interview (EDE) [24]. Participants' height and weight were measured at the intake assessment using a high capacity digital scale. Participants' fasting lipid levels, fasting blood glucose level, and glycated hemoglobin (HbA1c) were obtained and analyzed by Quest Diagnostics (Madison, NJ). Participants' blood pressure and heart rate were measured by trained research staff using an upper arm blood pressure machine (A & D Medical, Tokyo, Japan). Participants' measured their waist circumference in inches at the level of their navel. Participants also completed the self-report questionnaires described below.

Eating Disorder Examination (EDE) [24]—The EDE, a well-established investigator-based interview, assesses eating disorder psychopathology [25] with established reliability [26]. Except for diagnostic items, which are rated according to the appropriate duration stipulations, the EDE focuses on the previous 28 days. The EDE assesses the frequency of different forms of overeating, including *objective bulimic episodes* (*OBEs*; i.e., binge eating defined as unusually large quantities of food with a subjective sense of loss of control), *objective overeating episodes* (*OOEs*; i.e., unusually large quantities of food without a subjective sense of loss of control) and subjective bulimic episodes (*SBEs*; i.e., subjective sense of loss of control but a normal or small amount of food). The EDE also assesses participants' meal patterning by asking how many times in the past 28 days they have eaten

specific meals and snacks. Participants were categorized as a *meal skipper* if they skipped more than 50% (more than 14 days) of a meal (breakfast, lunch or dinner) over the past 28 days.

Weight and Eating History Interview (WEH)—The WEH is a structured clinical interview that assesses current and historical obesity-related variables of interest, including lowest and highest (non-pregnant) adult (≥18 years old) weight and age at those respective weights. Age at diet onset was assessed with the following question: "At what age do you remember first going on a diet?" Pre-treatment BMI change refers to the difference between participants' current BMI and BMI 12 months prior. Participants' weight 12 months prior was assessed with the following question: "What was your weight 12 months ago?"[27]. BMI (kg/m^2) was calculated using height measured at intake.

Self-Report Questionnaires—The Questionnaire for Eating and Weight Patterns-Revised (QEWP-R) [28] is a self-report measure that assessed participants' age at first binge, age when first overweight (10 pounds as a child or 15 pounds as an adult), and weight cycling (number of times lost and regained 20+ pounds). The Three Factor Eating Questionnaire (TFEQ) [29] comprises three factors: cognitive restraint, disinhibition, and hunger and has psychometric support. The current study employed the cognitive restraint subscale. The Beck Depression Inventory (BDI) [30] is a 21-item widely used and well-established inventory [31] assessing symptoms of depression and negative affect. The Rosenberg Self-Esteem Scale (RSES) [32] is a 10-item, widely-used, psychometrically well-established measure of global self-esteem.

Metabolic Syndrome (MetSyn)—Participants were categorized as having MetSyn if they met three or more of the five criteria outlined by the National Cholesterol Education Program's Adult Treatment Panel III guidelines [2]: *abdominal obesity*: waist circumference >40 inches (102 cm) in men and >35 inches (88 cm) in women; *hypertension*: ≥130 mm Hg systolic or ≥85 mm Hg diastolic; *hypertrigylceridemia*: ≥150 mg/dl (1.7 mmol/l); *low high density lipoprotein (HDL) cholesterol*: <40 mg/dl (1.03 mmol/l) in men and <50 mg/dl (1.30 mmol/l) in women; or *high fasting glucose*: ≥110 mg/dl (6.1 mmol/l).

Statistical Analyses

Analyses of Variance (ANOVAs) for continuous variables and chi-square analyses for dichotomous variables were conducted to compare individuals with versus without MetSyn on disordered eating and weight history developmental variables. Pearson bivariate correlations were conducted to test for associations between the MetSyn components and disordered eating and weight history developmental variables. Binary logistic regressions assessed whether specific developmental variables significantly increased the likelihood of MetSyn.

RESULTS

Demographic and Metabolic Variables

Forty-four percent (n=65) met criteria for MetSyn; 56.1% (n=83) did not meet criteria for MetSyn. Table 1 depicts the comparison of participants with versus without MetSyn on demographic variables as well as MetSyn components. Participants with MetSyn did not differ from those without MetSyn on gender, ethnicity, education, age, or BMI. Participants with MetSyn had, on average, significantly wider waist circumferences, lower levels of HDL, higher levels of triglycerides, higher systolic and diastolic blood pressure, as well as higher fasting glucose and HbA1c levels. Participants with MetSyn did not significantly differ from those without MetSyn in levels of LDL, total cholesterol, or heart rate.

Developmental Variables

Table 2 presents the comparison of participants with MetSyn to those without MetSyn on developmental variables. Participants with MetSyn reported a significantly older age at BED onset ($F(1, 136)=5.324$, $p=0.023$) as well as a significantly longer time between their first binge and when they met full diagnostic criteria for BED ($F(1,100)=4.088$, $p=0.046$) compared to those without MetSyn. Participants with MetSyn also reported a shorter duration of BED compared to those without MetSyn ($F(1,135)=6.650$, $p=0.011$), after controlling for current age. Participants with MetSyn did not differ from those without MetSyn on age at first binge, age at diet onset, age first overweight, age at lowest or highest adult BMI, weight cycling or BMI change in year prior to seeking treatment.

Current Disordered Eating Pathology and Clinical Variables

Table 3 depicts the comparison of participants with versus without MetSyn on current disordered eating pathology and clinical variables. Participants with MetSyn versus without Metsyn did not differ on global eating disorder pathology, current objective bulimic episode frequency, meal skipping, dietary restraint, depressive symptoms, or self-esteem.

Metabolic Syndrome Components and Developmental Variables

Table 4 depicts the associations between specific MetSyn components and developmental variables. Participants who reported an older age at dieting onset, an older age when first became overweight, as well as an older age at their highest BMI were significantly more likely to have a higher systolic blood pressure. Participants with an older age at dieting onset, an older age at lowest BMI, and more weight cycling were significantly more likely to have lower HDL. Participants with an older age at BED onset were significantly more likely to have higher fasting blood glucose. BMI change in the year prior to seeking treatment was not significantly correlated with any MetSyn components. Developmental variables were not significantly associated with either diastolic blood pressure or triglyceride levels.

Metabolic Syndrome Components and Current Eating Pathology and Clinical Variables

Table 4 also depicts the correlations between specific metabolic syndrome components and current disordered eating pathology and clinical variables. Participants with greater eating pathology and more dietary restraint were significantly more likely to have a smaller waist circumference. Participants with *lower* eating pathology were significantly more likely to have higher blood glucose levels. Current eating pathology and clinical variables were not significantly associated with systolic or diastolic blood pressure, HDL or triglycerides.

Metabolic Syndrome and Developmental Trajectories

Binary logistic regressions revealed several developmental trajectory variables associated with MetSyn. After taking into account BMI and current age, an older age at BED onset (B=0.036, Wald (1,138)=6.243, $p=0.012$) was associated with a significantly increased likelihood of MetSyn. After controlling for BMI and current age, a longer time between when participants had their first binge and when they met full criteria for BED was associated with a significantly increased likelihood of developing MetSyn (B=0.054, Wald (1,102)=4.818, $p=0.028$). After taking into account BMI and current age, a shorter time between when participants' met full diagnostic criteria for BED and when they sought treatment for binge eating disorder was associated with a significantly lower likelihood of developing MetSyn (B=−0.036, Wald (1,138)=6.243, $p=0.012$).

DISCUSSION

To our knowledge, this study is the first to examine associations between the MetSyn and the developmental trajectories of disordered eating and weight-related behaviors in a sample of obese treatment-seeking individuals with BED. Understanding the development of MetSyn and associated medical comorbidities in this high-risk subgroup is critically important given the possible physical consequences and increased health care utilization of BED patients [10, 33]. Primary findings indicate that 44% of our sample met criteria for MetSyn, which appears to be associated with an older age at BED onset, a longer time from first binge to BED onset and a shorter BED duration before seeking treatment.

The proportion of participants in our study with MetSyn (44%) is lower than the 60% prevalence rate reported in a previous study of BED in a specialty care clinic [18], but remarkably similar to estimates of MetSyn among BED patients recruited through primary care settings (43.2%) [19]. With respect to demographic variables, we found that patients with MetSyn did not differ from those without MetSyn on BMI, gender, ethnicity, age, or education. Thus, despite a larger sample, we failed to replicate results from previous population-based studies [7, 8] and clinic-based trials [18, 19] that suggested MetSyn is more prevalent among men than women. For example, 66% of men and 33% of women met criteria for MetSyn in a primary care settings sample [19], while 90.2% of men and 50.7% of women met criteria for MetSyn in a specialty care clinic sample [18]. The gender composition of the study samples may partially explain this discrepancy, as the current study included twice as many men than these previous trials [18, 19]. Notably, the overall distribution of MetSyn did not differ significantly by race/ethnicity or by age. These findings parallel those of Barnes et al. [19], but differ from studies of BED in specialty care clinics [18] and population-based trials [7, 8] that have demonstrated higher prevalence rates of MetSyn among Caucasians than African-Americans. Consistent with two previous studies [18, 19], we found no significant age differences between patients with and without MetSyn, although at least one previous population-based study has illustrated a link between older age and MetSyn [8]. Limited sample sizes have likely contributed to these inconsistencies across trials with respect to demographic variables (age, gender, and racial/ethnic group) and prevalence rates of MetSyn. Discrepant exclusion criteria across studies may also partially explain differences in prevalence rates of MetSyn, with this study's exclusionary criteria excluding participants at high risk for MetSyn.

Dieting behaviors and weight cycling remain areas of controversy in obesity research. Participants in this study with and without MetSyn did not differ on a number of developmental variables (age at dieting and overweight onset, and weight cycling) and disordered eating pathology variables (meal skipping and dietary restraint). Although some dieting behaviors (i.e., dieting at a younger age and greater dietary restraint) have emerged as protective factors against the development of MetSyn among patients with BED in two previous studies [18, 19], we failed to replicate these findings. One potential explanation is that patients who present for weight loss to primary care clinics (particularly those with exhibit metabolic abnormalities) may receive more intensive treatment than patients assessed in other settings. Of note, regularly skipping more than 50% of meals was not associated with MetSyn in this sample. Although the 50% cut off is arbitrary, it is interesting that this significant dietary restriction did not have an effect on these metabolic variables. These results are consistent with a primary care sample [19] but inconsistent with a clinical sample that found MetSyn to be associated with more meal skipping [18]. It is possible that skipping any proportion of meals has adverse consequences on some metabolic variables and that our 50% cut off may not reflect the best way to define problematic levels of meal skipping. Given the wide range of eating patterns and behavioral strategies that likely fall under the umbrella term of "dieting behaviors," further research is necessary to more clearly

understand the role of specific dieting behaviors and weight cycling in the developmental trajectory of MetSyn.

To our knowledge, this study is the first to examine the developmental trajectories of BED and its relation to MetSyn. Given our finding that a longer delay between first binge and meeting BED criteria significantly increases the likelihood of developing MetSyn, even sub-threshold BED behavior over a sustained amount of time may have a negative impact on metabolic functioning. It may be that low frequency binge eating disrupts metabolic processes more significantly than higher frequency BED behaviors, or that other factors (e.g., binge size, timing) may have a greater impact on the development of MetSyn. Importantly, a shorter time between when participants met full diagnostic criteria for BED and when they sought treatment for the disorder significantly decreases the likelihood of developing MetSyn. It is possible that early intervention for binge eating and/or excess weight may help prevent the onset of physical complications associated with BED/obesity and this should be the focus of future research.

We found that participants who develop BED at an older age are more likely to meet criteria for metabolic syndrome (i.e., mean age at onset of BED was 31 years for those with MetSyn versus 25.8 years for those without MetSyn). Such findings are of uncertain significance and require replication in future studies; we can only speculate that it is possible that as individuals age, they may be less able to effectively adapt to the consumption of a large amount of calorie-dense foods, placing them at higher risk for metabolic problems. For example, previous studies have demonstrated that eating large amounts of food in a discreet period of time and eating rapidly are associated with exaggerated insulin secretion, increased fasting glucose levels, decreased glucose tolerance, and elevated serum lipids [12, 13]. We note that research with older adults has suggested that aging is associated with disruptions in the metabolic processes that regulate energy balance, which are more pronounced among obese older adults as compared to their overweight counterparts [34]. Future research should explore the timing and sequence of BED onset and various metabolic disruptions across the lifespan in order to disentangle these complex relationships.

Strengths of the current trial include a larger subset of men than previous investigations of MetSyn among individuals with BED, assessments administered by doctoral-level research clinicians utilizing state-of-the-art and well-validated measures, and MetSyn determined based on measured lab findings. Several limitations, however, should be noted. This sample comprised primarily Caucasians, who have significantly lower rates of MetSyn than African Americans and Hispanic Americans [7]. Importantly, since this study group was one of convenience recruited for a clinical trial, a number of our exclusionary criteria may have resulted in lower rates of MetSyn than we would otherwise have observed. For example, patients currently utilizing anti depressants were excluded from the trial. Further, individuals with uncontrolled hypertension (> 160 systolic or > 95 diastolic), uncontrolled diabetes (HbA1c > 8.0) and/or significant cardiovascular disease were also deemed ineligible to participate in the larger treatment study and were therefore excluded from assessments. Thus, these findings may not generalize to individuals with more severe cardiovascular and metabolic abnormalities or psychiatric symptoms who may seek alternative treatments at different medical settings.

In summary, 44% of obese patients with BED in our sample met criteria for MetSyn, although this estimated frequency is likely to be a conservative one as our exclusionary criteria may have excluded potential cases at high risk for MetSyn. Patients with MetSyn did not differ from those without MetSyn on BMI, gender, ethnicity, age, or education. MetSyn was associated with a distinct developmental trajectory, which, surprisingly, is associated with a later onset and shorter BED duration. Taken collectively, these results may suggest

the potential positive impact of early intervention on the metabolic functioning of patients with BED and suggest a greater need for screening of obese individuals to identify higher-risk patients. The current study also underscores the multiple gaps that exist in the research on MetSyn among patients with BED. Future studies should prospectively examine causal connections in the development of MetSyn in relation to disordered eating in addition to excess weight.

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Table 1

Participants with versus without metabolic syndrome on demographic and metabolic variables.

	No MetSyn*n*=83	MetSyn *n*=65		
	***n* (%)**	***n* (%)**	**χ^2**	***p*-value**
Gender			2.799	0.094
Male	19 (22.9)	23 (35.4)		
Female	64 (77.1)	42 (64.6)		
Race/Ethnicity			0.250	0.617
African-American	13 (16.7)	8 (13.6)		
Caucasian	65 (83.3)	51 (86.4)		
Education			3.633	0.163
HS or GED	10 (12.5)	13 (21.0)		
Some College	27 (33.8)	25 (40.3)		
College	43 (53.8)	24 (38.7)		
	Mean (SD)	**Mean (SD)**	**F**	***p*-value**
Age	48.5 (8.1)	47.9 (9.6)	0.166	0.684
BMI(kg/m^2)	38.9 (6.2)	40.5 (5.7)	2.461	0.119
Waist Circumference	45.6 (5.5)	48.3 (5.4)	9.188	0.003 **
LDL (mg/dl)	119.3 (28.0)	114.6 (37.6)	0.751	0.388
HDL (mg/dl)	59.2 (13.5)	43.8 (11.1)	55.542	0.000 ***
Triglycerides	99.8 (38.2)	198.6 (74.6)	108.720	0.000 ***
Total Cholesterol	198.4 (30.7)	199.2 (46.6)	0.015	0.903
Systolic BP (mm Hg)	125.6 (14.2)	134.2 (14.3)	13.053	0.000 ***
Diastolic BP (mm Hg)	79.2 (8.6)	83.7 (11.5)	7.379	0.007 **
Heart Rate	75.1 (9.4)	77.5 (13.0)	1.750	0.188
Glucose (mg/dl)	94.3 (10.1)	106.8 (29.7)	17.839	0.000 ***
HbA1c	5.7 (0.29)	6.1 (0.96)	11.137	0.001 **

Note. MetSyn=metabolic syndrome. HS=high school. GED=General Equivalency Diploma. BMI=body mass index. LDL=low density lipoprotein. HDL=high density lipoprotein. BP=blood pressure. HbA1c=glycated hemoglobin.

* $p \leq 0.05$,

** $p \leq 0.01$,

*** $p \leq 0.001$.

Table 2

Participants with versus without metabolic syndrome on developmental variables.

	No MetSyn Mean (SD)	MetSyn Mean (SD)	F	*p*-value
Age at first binge	23.7 (12.2)	24.2 (12.6)	0.049	0.825
Age at BED onset	25.8 (12.6)	31.0 (14.0)	5.324	0.023*
1st binge to BED time lag	5.2 (7.6)	8.8 (1.1)	4.088	0.046*
Duration of BED[a]	22.6 (13.3)	16.7 (13.2)	6.650	0.011*
Age at diet onset	20.7 (9.5)	21.1 (10.8)	0.047	0.829
Age first overweight	18.2 (10.0)	17.7 (10.1)	0.076	0.783
Age at lowest adult BMI	24.0 (6.9)	22.0 (5.4)	3.379	0.068
Age at highest adult BMI	45.2 (9.4)	44.2 (11.4)	0.312	0.577
Weight cycling	3.0 (1.0)	3.1 (1.0)	0.019	0.892
Pre-treatment BMI change	2.87 (3.7)	2.6 (3.1)	0.154	0.696

Note. MetSyn=metabolic syndrome. BED=binge eating disorder.

[a]Duration of BED calculated as age at BED onset subtracted from current age with current age as a covariate in this ANOVA. Weight cycling=number of times lost and regained 20 pounds. Pre-treatment BMI change=weight change in year before seeking treatment.

*$p \leq 0.05$.

Table 3

Participants with versus without metabolic syndrome on disordered eating pathology and clinical variables.

	No MetSyn Mean (SD)	MetSyn Mean (SD)	F	*p*-value
Global EDE	2.9 (0.9)	2.7 (1.0)	1.291	0.258
OBEs Month 1 Episodes	18.8 (11.3)	19.0 (13.6)	0.015	0.904
Meal Skipping[a]	***n* (%)**	***n* (%)**	0.124	0.725
No Skip	50 (33.8)	41 (27.7)		
Skip	33 (22.3)	24 (16.2)		
TFEQ Dietary Restraint	7.1 (4.2)	6.7 (3.6)	0.375	0.541
BDI	16.1 (8.5)	15.2 (9.2)	0.416	0.520
Self-Esteem	29.9 (6.2)	28.7 (6.7)	1.226	0.270

Note. OBE=objective binge episode.

[a]No skip=consumed ≥50% of all meals (breakfast, lunch, and dinner) during the past 28 days; chi-square analysis reported for this dichotomous variable. TFEQ=Three Factor Eating Questionnaire. BDI=Beck Depressive Inventory.

Table 4

Correlations among metabolic syndrome and developmental and clinical variables.

	Waist	Syst BP	Diast BP	HDL	Trig	Glucose
Age at First Binge	0.118	0.079	0.103	−0.068	−0.004	−0.038
Age at BED Onset	0.099	0.128	0.047	−0.117	0.158	**0.196** *
Age at Dieting Onset	0.124	**0.178** *	0.125	**−0.252** **	0.040	−0.035
Age First Overweight	0.052	**0.176** *	0.085	−0.067	−0.116	0.052
Age at Lowest BMI	−0.161	−0.044	−0.079	**0.187** *	−0.133	−0.008
Age at Highest BMI	−0.028	**0.207** *	0.060	0.039	0.068	0.173
Weight Cycling	0.065	0.013	−0.009	**0.170** *	0.094	0.101
Pre-Treatment BMI Change	−0.012	−0.002	0.040	0.005	−0.016	−0.133
Global EDE	**−0.199** *	−0.096	−0.125	0.095	−0.145	**−0.180** *
OBE Month 1 episodes	0.010	−0.073	−0.021	−0.072	−0.016	−0.048
TFEQ Restraint	**−0.288** ***	−0.076	0.024	0.108	−0.031	0.025
BDI	−0.042	0.034	−0.010	−0.052	0.017	−0.098
RSES	0.057	−0.002	0.023	0.139	−0.079	0.089

Note. Waist=waist circumference. Syst BP=systolic blood pressure. Diast BP=diastolic blood pressure. HDL=high density lipoproteins. Trig=triglycerides. Weight Cycling=Number of times lost and regained ≥20 lbs. BMI = body mass index. EDE=Eating Disorder Examination Interview; mean subscales employed. OBE=objective bulimic episode. Pre-treatment BMI change=weight change in year before seeking treatment. TFEQ=Three Factor Eating Questionnaire. BDI=Beck Depression Inventory. RSES=Rosenberg Self-Esteem Scale.

* $p \leq 0.05$,

** $p \leq 0.01$,

*** $p \leq 0.001$.