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The Factor Structure of the Metabolic Syndrome in Obese Individuals with Binge Eating Disorder

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Abstract

Objective—Metabolic syndrome (MetS) is strongly linked with cardiovascular disease and type-II diabetes, but there has been debate over which metabolic measures constitute MetS. Obese individuals with binge eating disorders (BED) are one of the high risk populations for developing MetS due to their excess weight and maladaptive eating patterns, yet, the clustering patterns of metabolic measures have not been examined in this patient group.

Methods—347 adults (71.8% women) were recruited for treatment studies for obese individuals with BED. We used the VARCLUS procedure in the Statistical Analysis System (SAS) to investigate the clustering pattern of metabolic risk measures.

Results—The analysis yielded four factors: obesity (body-mass-index [BMI] and waist circumference), lipids (HDL and triglycerides), blood pressure (systolic and diastolic blood pressure), and glucose regulation (fasting serum glucose and Hb1Ac). The four factors accounted for 84% of the total variances, and variances explained by each factor were not substantially different. There was no inter-correlation between the four factors. Subgroup analyses by sex and by race (Caucasian vs. African American) yielded the same four factor structure.

Conclusion—The factor structure of MetS in obese individuals with BED is not different from those found in normative population studies. This factor structure may be applicable to the diverse population.

Keywords

Metabolic syndrome; Factor analysis; Binge eating disorders; Obesity; Treatment sample

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Introduction

Metabolic syndrome (MetS) is a clustering of metabolic risk factors, including central adiposity, high blood pressure, low level of high-density-cholesterol (HDL), elevated level of triglyceride, and high fasting glucose level [1]. Clear evidence suggests that these metabolic risk factors are associated with both cardiovascular disease and type-II diabetes [2, 3], and contribute to a growing health care cost in the U.S. [4]. Obesity, as defined by body-mass-index (BMI) > 30, is a strong risk factor for MetS. While the prevalence of MetS in the U.S. adult population is approximately 24% [5], the rates of MetS are roughly 50% in obese women and 60% in obese men [6].

Binge eating disorder (BED) is defined by recurrent excessive food intake with feeling loss of control in the absence of inappropriate compensatory weight-control behaviors [7]. BED has been strongly associated with the severity of obesity [8, 9]. Obese binge eaters engage in maladaptive eating behaviors and patterns that may place them at greater risk for MetS, compared with non-binge eating obese individuals. For example, a gorging eating pattern has been linked with more liver fat and elevated lipids in severely obese men and women [10]. Eating meals irregularly [11] and meal skipping [12] have also been associated with MetS. A longitudinal study indeed demonstrated that binge eaters were at greater risk for newly diagnosis of MetS components, compared with non-binge eaters [13]. Therefore, obese individuals with BED have been highlighted as an important subgroup with elevated risk for MetS [9].

Currently, there are several different definitions of MetS, which consist of similar metabolic risk factors. However, depending on the definitions, the prevalence of MetS varies even within samples, and the discrepancy could be substantial for some subgroups [14], highlighting the need for a unified MetS definition that reflects underlying pathophysiological mechanisms across different populations. Factor analysis has been used to identify a clustering pattern of metabolic risk factors that may represent the underlying pathophysiological processes. There has been some consensus on the three- to four factor structure, with insulin and glucose measures, blood lipids measures, blood pressure measures, and obesity measures (BMI and waist/hip circumferences) each clustering together [15–24]. Different patterns of clustering of metabolic risk factors, however, have been found in those with dysregulated metabolic function, such as type-II diabetes [25, 26]. Given that abnormal eating patterns appear to have unique impact on the metabolic function, obese individuals with BED may also show a distinctive factor structure of MetS.

Most studies have used principal components analysis (PCA) as a factor extraction method. However, use of PCA has been criticized for its exploratory and subjective nature, especially the secondary application of rotation methods [15, 16, 24]. To extract independent, interpretable solutions, previous studies primarily applied orthogonal rotation (e.g., varimax), which forces factors to be statistically uncorrelated. This may be particularly questionable given MetS is often thought to represent a single disease construct with related risk components [15]. Further, even after orthogonal rotation is applied, there is often overlapping of factor loading [17–20], precluding clear interpretation of the identified factor structure. Woolston et al. [16] recently suggested an application of the VARCLUS procedure in the Statistical Analysis System (SAS) (release 9.3, 2002–2010, SAS Institute, Cary, NC) [27] as an alternative method of factor extraction. The VARCLUS is a type of oblique component analysis, and aims to identify one-dimensional clusters of mutually correlated variables [16]. The study applied different factor extraction methods to the same data, and demonstrated that without increasing the complexity of the statistical procedure, the VARCLUS procedure produced non-overlapping factor components, which is a simpler

model to interpret than the results of PCA with an oblique rotation where a few metabolic risk measures loaded on more than one factor.

To our knowledge, the factor analysis of metabolic risk factors in obese individuals with BED has not been studied. This is important to evaluate whether currently available definitions of MetS are suitable in diagnosis of metabolic abnormalities in obese individuals with BED. In addition, the relationships among factor components and contribution of each metabolic risk factor are also valuable to understand risk for MetS that is specific to this population. These research questions have significant clinical implications as they may inform ways to develop targeted screening and prevention strategies for cardiovascular diseases and type-II diabetes. Therefore, the present study aimed to explore the factor structure of MetS in obese individuals with BED who participated in treatment studies for BED, using the VARCLUS procedure.

Methods

Participants

Participants were 347 adults (249 women, 98 men; mean age = 46.4 ± 10.7 years old) who were interested in participating in treatment studies for obese individuals with BED. All participants were obese ($BMI \geq 30 \text{ kg/m}^2$), and met or exceeded BED criteria based on DSM-5. We used the duration criterion of 6 months from DSM-IV-TR (APA) because it is more stringent than the 3-month duration criterion of DSM-5. Exclusion criteria were: current anti-depressant therapy, severe psychiatric problems (lifetime bipolar disorders and schizophrenia, and current substance dependence), severe medical problems (e.g., cardiac and liver diseases), and uncontrolled hypertension, thyroid conditions, or diabetes. Ethnic composition of the sample was 62.4% Caucasian, 23.4% African American, 8.7% Hispanic, and 5.5% Other. 94.8% of the sample reported that they completed high school education or greater. Written informed consent was obtained from all participants and the research protocol was approved by the Yale Human Investigation Committee for the Protection of Human Subjects Involved in Research.

Assessment and Measures

Features of Eating Disorders—The Eating Disorder Examination (EDE) [28] interview assesses eating disorders and their features. The EDE focuses on the frequency of different forms of overeating in the past 28 days, including objective bulimic episodes (OBE; i.e., binge eating defined as unusually large quantities of food coupled with a subjective sense of loss of control), which corresponds to the DSM-based definition of binge eating. The EDE also comprises four subscales: Restraint, Eating Concern, Shape Concern, and Weight Concern. The items assessing these four scales are rated on a 7-point scale (0–6 range), with higher scores reflecting greater severity or frequency. An EDE global score was calculated as the mean of the four scales. The EDE interview is a well-established measure [29] with good inter-rater and test-retest reliability in studies with BED [30].

2.2.2. Metabolic measures—Participants' weights were measured using a high-capacity digital scale. Height, waist circumference, heart rate, and blood pressure were measured by trained research staff. Fasting lipid profile (total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides), glucose levels, and glycated hemoglobin 1Ac (HbA1c) were obtained through serum samples and were analyzed by Quest Diagnostics (Madison, New Jersey).

Statistical Analysis—We used the VARCLUS procedure in the Statistical Analysis System (SAS) (release 9.3, 2002–2010, SAS Institute, Cary, NC) [27]. The following

metabolic risk factors were included: BMI, waist circumference, systolic and diastolic blood pressure, HDL cholesterol, triglycerides, fasting serum glucose levels, and HbA1c. To correct skewness and kurtosis, triglycerides, fasting glucose levels, and HbA1c were log-transformed. The VARCLUS procedure attempts to divide a set of variables into non-overlapping clusters (i.e., no variable loads on more than one factor) by identifying the first principle component, and then iteratively splits them into two separate clusters to maximize the sum across clusters of the variance accounted for by the cluster components [27]. Each variable is assigned to a cluster component with which it has the higher squared correlation. In the VARCLUS, the quality of each cluster is measured by the ratio between R^2 of the variables when regressed on the remaining variables in the cluster to which it is assigned (R^2_{own}), and R^2 of the variable when regressed on any other cluster produced in the analysis ($R^2_{nearest}$). A variable has a strong fit to the cluster which is assigned when it has high R^2_{own} and low $R^2_{nearest}$ values, which results in a low R^2 ratio (i.e., $[1-R^2_{own}]/[1-R^2_{nearest}]$). Given the exploratory nature of this study, following Woolston et al. [16], we did not specify the number of clusters to extract (i.e., MAXCLUSTERS) or the proportion of variance explained by each cluster (i.e., PROPORTION). SAS also uses a maximum eigenvalue > 1 by default to identify latent clusters. We again did not specify eigenvalue as it is the most commonly used threshold to determine the sufficiency of a single underlying factor dimension [31]. To explore the relationship between the features of BED and identified clusters, correlation coefficients between the cluster component scores and eating and weight-related characteristics were also calculated. Furthermore, the factor structure or the patterns of factor loadings could differ by sex and race [24, 32]. Therefore, in addition, we completed the VARCLUS procedure separately for men and women, and for non-Hispanic Caucasian and African American. Except for comparison between races, the analyses included all participants.

Results

Sample characteristics

Table 1 summarizes basic eating/weight-related characteristics. Table 2 summarizes the metabolic characteristics of the participants. Based on the guideline by the National Cholesterol Education Program's Adult Treatment Panel III guidelines [33], 38.9% of individuals met clinical criteria for the MetS. Among individual metabolic components, the most prevalent metabolic clinical problem was central or abdominal obesity (~98%), followed by high blood pressure (~50%).

Results of the VARCLUS

We identified four clusters: 1) *Obesity* (BMI and waist circumference), 2) *Glucose regulation* (fasting serum glucose level and Hb1Ac), 3) *Blood pressure* (systolic and diastolic BP), and 4) *Lipids* (HDL and glycerides) (Figure 1). The low R^2 ratio indicates that all variables are strongly fit to the assigned cluster component, and all cluster components are well formed (Table 3). Inter-cluster correlations were small (i.e., $r < .30$; Table 4). Each variable loads highly only on its own cluster component, and shows low loadings on other cluster components (Table 5). The identified four-cluster structure explains 84% of a total variance (Table 6). The variance explained was similar across four cluster components. As indicated by 'the proportion explained', all cluster components appear to be well explained by assigned variables.

The VARCLUS by sex and race

In both men and women, the same four-factor structure as the overall sample was identified. Similar to the factor structure in the overall sample, variables showed low R^2 ratio (range = 0.110–0.303 [women], 0.089–0.305 [men]), and high loading only on its own cluster

component (range = 0.847–0.946 [women], 0.822–0.960 [men]). The four clusters showed small inter-correlations in both sex, except for medium correlation ($r = -0.332$) between *obesity* and *lipids* in men (range = 0.085–0.250 [women], -0.215–0.097 [men]). For both men and women, the amount of variances explained was not substantially different by cluster components (range = 21.4%–26.7% [women], 21.6%–27.5% [men]) (Table 6).

Similarly, we compared the factor structure between non-Hispanic Caucasian and African American participants. Again, the same four-factor structure was identified for both Caucasian and African American samples. All variables showed low R^2 ratio (range = 0.107–0.251 [Caucasian], 0.073–0.326 [African American]), and high loading only on its own cluster component (range = 0.880–0.947 [Caucasian], 0.855–0.967 [men]). In both Caucasian and African American samples, *obesity* and *lipids* showed medium correlations ($r = -0.341$ and -0.374 , respectively). Small correlations were found for other inter-correlations among cluster components (range = -0.065–0.260 [Caucasian], 0.083–0.219 [African American]). For both Caucasian and African American samples, the amount of variances explained was not substantially different by cluster components (range = 23.1%–26.8% [Caucasian], 21.4%–27.4% [African American]) (Table 6).

Relationship between clusters and features of BED

Table 7 summarizes the correlation between each cluster component score and features of BED. Greater *obesity* component score was correlated with earlier first time overweight. Greater *glucose regulation* component score was correlated with later dieting onset and less EDE eating concern score. Greater *blood pressure* component score was correlated with later dieting onset and binge eating onset, and less EDE shape and eating concern scores. Greater *lipid* component score was correlated with earlier dieting onset, and greater EDE scores, except for eating concern.

Discussion

Using the VARCLUS procedure, the present study found a four-factor structure of MetS, consisting of obesity (BMI and waist circumference), glucose regulation (fasting serum glucose level and Hb1Ac), blood pressure (systolic and diastolic blood pressure), and lipids (HDL and triglycerides) in a sample of obese individuals with BED. Although there are some differences in metabolic risk measures included in the model between studies, the four-factor structure observed for this study group of obese patients with BED is generally consistent with previous studies with normative population samples [15–17, 22, 24]. Obese individuals with BED thus do not appear to show a distinctive pattern of clustering of metabolic risk measures. In addition, the amount of variance explained was not substantially different between each cluster component, suggesting that the identified four factors seem to make equivalent contribution to forming MetS in our sample. This finding is different from the studies that applied non-PCA factor analytic approaches to understand factor structures of MetS in a normative adult population. Using the VARCLUS procedure, Woolston et al. [16] reported that the factor was predominantly explained by insulin resistance component, while Shen et al. [15] that reported that insulin resistance and obesity factors primarily represented MetS through confirmatory factor analysis. Therefore, while the factor structure may be shared, whether one or more factor(s) play a more vital role than others in MetS may vary by populations.

Further correlation analysis revealed distinctive relationship of each cluster component with features of BED. Approximately half of our sample met the clinical criteria for elevated blood pressure, and later onset of binge eating may increase risk for high blood pressure. Earlier dieting onset was associated with greater abnormality in glucose regulation, blood pressure, and lipid levels (note the directionality of factor loadings in Table 5). Thus, even

among obese individuals, early attempts to reduce their weight may help reduce the risk for developing metabolic abnormalities. Disordered eating pathology was also associated with less abnormality in glucose regulation (eating concern), blood pressure (eating and shape concerns), and lipid levels (total, restraint eating, and shape and weight concerns). Our sample did not report severe symptoms of eating disorders. Thus, moderate attempts to regulate food intake may reduce the negative impact of obesity and binge eating pathology on metabolic function.

Subsequent subgroup analyses yielded the same four-factor structure across sex and race, suggesting the stability of the identified factor structure. Consistent with the whole sample analysis, the variance explained by each factor was not substantially different. Again, this suggests that among obese individuals with BED, all metabolic factors may equally represent MetS, regardless of sex or race. The subgroup analyses, however, revealed that obesity and lipids factors showed medium correlation in men, and in both Caucasian and African American. Thus, while the factor structure may be consistent, the degree of association between factors may differ by subgroups of individuals.

Some limitations in this study are noteworthy and may suggest the need for further investigations of the factor structure of MetS in obese individuals with BED. Unlike some previous studies, the current study did not include fasting insulin level, but instead included Hb1Ac, a measure of glycemic regulation. Given that insulin resistance has been suggested as a unifying underlying mechanism of MetS, inclusion of fasting insulin level seems to be an important future research venue. However, recent research has highlighted that Hb1Ac be considered as part of MetS risk measures [34]. In the current study, Hb1Ac showed high and specific loading on the glucose regulation factor along with fasting serum glucose level. Thus, Hb1Ac may signal an important biological process that constitutes MetS. Our sample consisted of treatment-seeking obese individuals without major or uncontrolled health issues and our findings may not generalize to non-treatment-seeking groups or to similar groups with advanced problems such as diabetes [25, 26]. Given that this was the first study to examine the factor structure of MetS, we employed an exploratory factor analytic approach, which is useful for hypothesis generation but not for hypothesis testing [35]. Future research with confirmatory factor analysis should help understand the pathophysiology of MetS.

In summary, the present study demonstrated that the factor structure of MetS was not different in obese individuals with BED from normative population studies. In addition, metabolic dysfunction of this sample was not explained by one or two specific metabolic component. The consistency of the factor structure between the current study and previous studies, despite difference in metabolic variables included in the analyses, suggests that this four factor structure may be universally applicable to diverse populations. Finally, the current findings provide additional evidence to the strengths of the VARCLUS procedure. As Woolston et al. [16] demonstrated, the strength of VARCLUS procedure is the simplicity of the factor interpretation by extracting non-overlapping factors (i.e., no variable loads on more than one factor). As factors are not overlapping, interpretation of variances explained by each factor and inter-correlations between factors are also simpler than factor analysis, which is particularly useful in comparison of the results between populations or subgroups when the same factor solutions are identified. This has significant clinical implication as identifying differences in contribution by factor components or the degree of inter-correlations among factor components can provide insights into differences in the underlying mechanism of MetS by subgroups, which ultimately can inform screening and prevention strategies to a specific population. Our findings suggest that monitoring metabolic measures related with obesity, blood pressure, glucose regulation, and lipids profile should be generally good indicators of metabolic dysfunction, but emphasis may need to be placed on different metabolic components for each population. To understand the

role of MetS as risk for cardiovascular diseases and type-II diabetes, future studies should also investigate whether the four metabolic cluster components have different predictive validity for these diseases in various populations.

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Figure 1.
A dendrogram of the cluster structure produced by VARCLUS.

Table 1**Eating/Weight-Related Characteristics**

	Mean (SD)
BED developmental variables	
Age of first time overweight	19.3 (11.1)
Age of binge eating onset	25.4 (13.0)
Age of dieting onset	22.8 (10.3)
Features of eating disorders	
Binge Eating Frequency (episodes in the past 28 days)	18.9 (14.9)
EDE Global	2.60 (0.9)
EDE Restraint	1.70 (1.3)
EDE Eating concern	2.06 (1.3)
EDE Shape concern	3.52 (1.2)
EDE Weight concern	3.14 (1.1)

Notes. EDE = Eating Disorder Examination.

Table 2**Metabolic measures**

	Mean (SD)
BMI	38.7 (5.7)
Waist (inches)	46.3 (5.8)
Systolic BP (mmHg)	128.2 (14.8)
Diastolic BP (mmHg)	79.9 (10.2)
Fasting glucose level (mg/dL)	104.8 (32.0)
HbA1c (%)	5.9 (0.9)
Total cholesterol	193.3 (37.4)
200 mg/dL (%)	39.8
HDL (mg/dL)	52.9 (14.5)
LDL (mg/dL)	114.1 (32.3)
Triglycerides (mg/dL)	130.8 (66.9)
% meeting criteria for symptoms of metabolic syndrome (MetS) ^I	
Central or abdominal obesity (> 40 inches for men, > 35 inches for women)	97.7
Triglycerides 150 mg/dL	28.2
Reduced HDL cholesterol (< 40 mg/dL for men, < 50 mg/dL for women)	32.9
Systolic BP 130 mmHg or Diastolic BP 85 mmHg	50.4
Fasting glucose 110 mg/dL	21.6
Meeting criteria for MetS (%)	38.9

Notes. BMI = Body mass index; BP = blood pressure; HbA1c = glycated hemoglobin 1Ac.

^I = Criteria for MetS was based on the National Cholesterol Education Program's Adult Treatment Panel III guidelines [31]. Participants were categorized as having metabolic syndrome if they report 3 or more of the 5 criteria.

Table 3

R^2 measures demonstrating the ‘quality’ of each cluster component.

Cluster	Variable	R^2 own	R^2 nearest	$(1-R^2 \text{ own})/(1-R^2 \text{ nearest})$
1. Obesity	Waist	.8669	.0794	.1446
	BMI	.8669	.0357	.1380
2. Glucose regulation	Glucose	.9079	.0508	.0970
	HbA1c	.9079	.0732	.0994
3. Blood pressure	Systolic BP	.8537	.0602	.1557
	Diastolic BP	.8537	.0330	.1513
4. Lipids	HDL	.7458	.0511	.2679
	Triglycerides	.7458	.0809	.2766

Notes. BMI = Body mass index; BP = blood pressure; HbA1c = glycated hemoglobin 1Ac. A strong fit to the assigned cluster is demonstrated when a variable has a high R^2 own and low R^2 nearest.

Table 4

Correlations between cluster components

	Cluster 1	Cluster 2	Cluster 3	Cluster 4
1. Obesity	---	---	---	---
2. Glucose regulation	.149	---	---	---
3. Blood pressure	.175	.231	---	---
4. Lipids	-.252	-.260	-.05	---

Notes. Effect size of correlation coefficients are interpreted as: small = $.10 \leq r < .30$, medium = $.30 \leq r < .50$, and large = $.50 \leq r$ [36].

Table 5

Correlation between each covariate and the cluster components (i.e., factor loadings)

	Cluster 1	Cluster 2	Cluster 3	Cluster 4
Waist	.931	.184	.137	–.282
BMI	.931	.094	.189	–.187
Glucose	.137	.953	.203	–.225
HbA1c	.147	.953	.237	–.271
Systolic BP	.201	.245	.924	–.051
Diastolic BP	.122	.182	.924	–.032
HDL	–.226	–.165	–.002	.863
Triglycerides	.209	.284	.076	–.863

Table 6

A summary of the variance explained by each cluster component.

Overall (N = 347)		Women (n = 249)		Men (n = 98)		Caucasian (n = 216)		African American (n = 81)	
Variance Explained	Proportion Explained	Variance Explained	Proportion Explained	Variance Explained	Proportion Explained	Variance Explained	Proportion Explained	Variance Explained	Proportion Explained
Cluster 1	1.734 (25.7%)	1.738 (25.9%)	.869	1.830 (27.3%)	.915	1.691 (25.2%)	.846	1.789 (26.2%)	.895
Cluster 2	1.816 (26.9%)	1.790 (26.7%)	.895	1.842 (27.5%)	.921	1.793 (26.8%)	.897	1.871 (27.4%)	.936
Cluster 3	1.707 (25.3%)	1.742 (26.0%)	.871	1.585 (23.6%)	.793	1.666 (24.9%)	.833	1.710 (25.0%)	.855
Cluster 4	1.492 (22.1%)	1.435 (21.4%)	.716	1.450 (21.6%)	.725	1.549 (23.1%)	.775	1.462 (21.4%)	.731
Total variance explained									
	6.749	6.705	.838	6.708	.839	6.699	.837	6.832	.854

Notes. The variance explained = a variance of the factor structure explained by each cluster component. The proportion explained = a total variance of the variables in the cluster divided by the variance explained.

Table 7

Correlation between cluster component scores and features of binge eating disorder.

	Obesity	Glucose regulation	Blood pressure	Lipids
Age of first time overweight	-.153 [‡]	.055	.078	-.013
Age of binge eating onset	-.095	.089	.121 [†]	-.002
Age of dieting onset	-.095	.114 [†]	.137 [†]	-.164 [‡]
Binge Eating Frequency	.091	.071	-.001	-.022
EDE Global	-.042	-.087	-.075	.168 [‡]
EDE Restraint	-.096	-.016	.046	.130 [†]
EDE Eating concern	.025	-.112 [†]	-.134 [†]	.100
EDE Shape concern	-.026	-.057	-.115 [†]	.108 [†]
EDE Weight concern	-.028	-.072	-.017	.163 [‡]

Notes. EDE = Eating Disorder Examination.

[†] $p < .05$;

[‡] $p < .01$.