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Short Communication: Metabolic Characteristics of Youth with Loss of Control Eating

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Abstract

Purpose—Preliminary data in adults suggest that binge eating is associated with greater prevalence of metabolic syndrome (MetS) components. However, there are limited data in youth, and little is known of the role of binge episode size in these relationships.

Methods—We examined the relationship between loss of control eating and metabolic characteristics in a convenience sample of 329 treatment-seeking and non-treatment-seeking

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adolescent boys and girls. The sample was enriched by design with adolescents who were overweight or obese and with individuals who reported episodes of loss of control over their eating (either objectively large binge episodes, OBEs or subjectively large binge episodes, SBEs in the past month), as assessed by clinical interview. MetS components (blood pressure, lipids, glucose, and waist circumference) were the primary variables of interest.

Results—46% of the cohort reported loss of control eating; among those, 53% reported SBEs only and 47% reported OBEs. Youth with loss of control eating had higher systolic blood pressure (p=.001) and higher low-density lipoprotein cholesterol (LDL-c) (p=.002) compared to those without loss of control eating, in analyses adjusted for intervention-seeking status, fat mass and sociodemographic characteristics. Youth reporting OBEs had higher LDL-c (p=.013) compared to those reporting only SBEs.

Conclusions—Adolescents reporting loss of control episodes had greater dysfunction in some components of the MetS compared to youth without loss of control; episode size may contribute to metabolic dysfunction.

Keywords

Binge Eating; Loss of Control Eating; Metabolic Dysfunction; Adolescence

1. INTRODUCTION

Metabolic syndrome (MetS) is characterized by a cluster of abnormalities including abdominal obesity, dysglycemia, and abnormal cholesterol and triglyceride concentratoins, which in combination, magnify risk for development of heart disease and type-2 diabetes in adults (Zimmet, et al., 2007). Although obesity is considered a primary contributor to MetS, emerging evidence suggests that psychological factors may play a unique role in metabolic dysfunction.

Binge eating disorder (BED), characterized by recurrent consumption of large amounts of food while experiencing a loss of control over eating (*The Diagnostic and Statistical Manual of Mental Disorders*, 2013), is associated with obesity and may place individuals at risk for MetS. Cross-sectional (Epel, et al., 2004; Taylor, Hubbard, & Anderson, 1999) and prospective (Hudson, et al., 2010) data among adults suggests links between binge eating behavior and metabolic dysfunction, even after accounting for body weight. Moreover, we previously reported prospective links between children's report of binge eating and components of MetS (worsening triglycerides, increased visceral adipose tissue) (Tanofsky-Kraff, et al., 2012).

Although BED is uncommon among youth, loss of control (LOC) eating episodes are frequently reported (Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). LOC is characterized by a subjective experience of lack of control over eating, irrespective of the amount of food consumed, and can be comprised of classic, objectively large binge episodes (OBE) and/or subjectively large binge episodes (SBE) (Tanofsky-Kraff, et al., 2012). LOC episode size may have important implications in terms of metabolic function; given the greater energy intake involved, it is plausible that those with OBEs may have worse

metabolic function compared to youth with SBEs, however this notion remains unexplored. Therefore, we examined the relationship between LOC episode size and metabolic function in adolescents. We hypothesized that youth who reported who reported LOC, specifically OBEs would exhibit greater metabolic dysfunction compared to youth who reported SBEs or no episodes.

2. MATERIALS AND METHODS

2.1. Sample

A convenience sample was assembled from participants in several non-treatment (ClinicalTrials.gov ID: NCT00631644, NCT00001195, and NCT00001522, n=109), treatment (NCT00001723, n=68), and prevention (NCT00263536, n=17; NCT00680979, n=90, NCT01425905, n=45) studies of eating behavior and obesity conducted at the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD). NCT00680979 was also conducted at the Uniformed Services University of the Health Sciences (USUHS). For all studies, individuals were excluded for major medical or psychiatric issues, pregnancy, or recent significant weight loss. For the intervention studies, youth currently involved in psychotherapy were excluded. Metabolic data from 5% of the current sample were previously published with the results of a questionnaire assessment of binge eating (Tanofsky-Kraff, et al., 2012). For the present analysis, youth between the ages of 12 and 18 years who were interviewed using the Eating Disorder Examination (EDE) at baseline visits were included. All studies were approved by the NICHD Institutional Review Board (IRB). The one prevention study (NCT00680979) conducted jointly at two sites, was also approved by the USUHS IRB. Written consent and assent were provided by parents and children, respectively.

2.2. Procedure

Participants were seen at the NIH Hatfield Clinical Research Center. Data for participants were included if metabolic parameters were collected within 3–4 months of the EDE.

2.3. Measures

2.3.1. LOC Eating—The *Eating Disorder Examination version 12OD/C.2* (EDE) (Fairburn & Cooper, 1993)was administered by trained interviewers to categorize participants as those who: endorsed at least one episode of unambiguous overeating with a sense of LOC (OBE); reported LOC, but the amount consumed was ambiguously large (SBE); reported overeating without LOC; or endorsed no episodes of overeating or LOC. LOC eating was defined by the presence of one or more OBE or SBE in the previous month. The EDE has good inter-rater reliability for all episode types for adolescents (Glasofer, et al., 2007).

2.3.2. Body Composition—Height and fasting weight, as well as BMI and BMI-z were calculated as previously described (Nicholson, et al., 2001). Body fat mass (kg) was measured either by dual-energy x-ray absorptiometry (DXA) (Hologic, Bedford, MA, USA) or air displacement plethysmography (Bod Pod; Life Measurement Inc., Concord, CA,

USA). Measurements of adiposity were considered equivalent for the two different assessment techniques (Robotham, et al., 2006).

2.3.3. Metabolic Function—Systolic (SBP) and diastolic blood pressures (DBP) were measured once after 5 minutes rest at the right brachial artery via an automated blood pressure monitor (Dynamap, GE Heathcare). Waist circumference (WC) measurements were taken twice, with a flexible, non-elastic tape measure (Country Technology, Inc, Gay Mills, WI) at the midpoint between the bottom of the rib cage and above the top of the iliac crest. Fasting triglycerides (TGL), glucose and cholesterol were measured from blood samples using a Hitachi 917 analyzer using reagents from Roche Diagnostics (Indianapolis, IN). A Cobas FARA analyzer was used to directly measure HDL-c using reagents from Sigma chemical (St. Louis, MO). LDL-c was calculated using the following formula: Total Cholesterol - HDL - (TGL/5). MetS was defined using an age- and sex-specific percentile-based cut-off definition commonly used in previous reports (Gustafson, et al., 2009), and was considered present when an adolescent met the cut-points for at least 3 factors.

2.4. Analytic Approach

All analyses were performed with IBM SPSS 22.0. Data were screened for outliers, skew, and kurtosis and outliers were Winsorized to fall 1.5 times the interquartile range below or above the 25th or 75th percentile. Two MANCOVA models were used to compare differences in MetS components; independent variables were (1) LOC vs. no LOC and (2) OBEs vs. SBEs. A binary logistic regression was performed to determine the impact of LOC, as well as episode size, on the presence of MetS, or any elevated component of MetS. Covariates in all models included age, race, sex, fat mass, height, and intervention-seeking status. Follow-up analyses were conducted separately with intervention and non-intervention youth. Differences between groups were considered significant when p values were <.05. All tests were two-tailed.

3. RESULTS

3.1. Preliminary Analyses

3.1.1. Descriptive Statistics—Participants were 329 youth (41 males) 12 to 18 years old (M±SD 14.79±1.6). The majority of participants were non-Hispanic Black (35%) or non-Hispanic White (32%), and 45.6% endorsed at least one episode of LOC in the past month. Among youth with LOC, 53.3% reported SBEs only, 25.3% reported OBEs, and 21.3% reported both SBEs and OBEs. The number of reported LOC episodes ranged from 1 to 24 (M±SD 4.0 ±4.8). Approximately 2% of the sample met full DSM-IV-TR criteria for BED, and 8% of all met full criteria for MetS. Sample demographics are presented in Table 1.

3.2. Presence vs. Absence of LOC and metabolic function

There was a main effect of LOC, such that the multivariate test was significant for LOC presence, Pillai's Trace= 0.074, F=3.61 df= (7,315), p=0.001, indicating a difference in overall metabolic factors between youth with and without LOC. The univariate F tests showed differences between youth with and without LOC: youth reporting LOC had

significant higher SBP (F=10.36, p=0.001). and higher LDL-c (F=9.90, p=0.002) compared to youth without LOC (Table 2).

When running the same analyses, without weight-loss treatment-seekers, the multivariate test remained significant for LOC, Pillai's Trace= 0.095, F=3.71, df= (7,248), p=0.001, indicating a difference in overall metabolic factors between youth with and without LOC. The univariate F tests showed a significant difference between youth with and without LOC for higher SBP (F=6.06, p=0.014), lower HDL-c (F=4.64, p=0.032), and higher LDL-c (F=12.46, p<0.001). When running the same analyses, excluding both weight-loss treatment-seekers and youth enrolled in the type 2 diabetes prevention study, the multivariate test remained significant for LOC, Pillai's Trace= 0.126, F=4.17, df= (7,203), p<0.001, indicating that youth with LOC displayed significantly higher SBP (F=5.55, p=0.019) and LDL-c (F=16.98, p<0.001).

When full criteria MetS was considered categorically as presence or absence, there was no significant difference between youth with and without LOC (χ^2=0.028, p=0.87; 7.3% of youth with LOC had MetS, 95% CI [4.14, 12.65] vs. 7.8% of youth without LOC, 95% CI [4.53, 13.11]). When individual components of the MetS were considered categorically, youth with and without LOC did not differ in prevalence of elevated BP (χ^2=2.34, p=0.21), elevated WC (χ^2=0.10, p=0.42), elevated TGL (χ^2=1.05, p=0.38) or low HDL-c (χ^2=2.81, p=0.12).

3.3. OBE vs. SBE and metabolic function

Among those with LOC, there was a main effect of episode size, such that the multivariate test for metabolic function analyses was significant for OBE presence, Pillai's Trace= 0.12, F=2.57, df= (7,136), p=0.016, indicating a difference in overall metabolic function between youth with OBEs vs. SBEs. Youth with OBEs had significantly higher LDL-c compared to SBEs (F=6.30, p=0.013). Results remained the same when accounting for episode frequency.

Among the cohort of non-intervention youth, the multivariate test was no longer significant, Pillai's Trace= 0.076, F=1.46, df= (7,125), p=0.19, indicating no difference in overall metabolic factors between youth SBEs and OBEs.

There was no significant difference between OBE compared to SBE with regard to MetS presence (χ^2=1.79, p=0.18; 4.3% with OBE had MetS, 95% CI [1.12, 12.84] vs. 10% with SBE, 95% CI [5.15, 18.51). When individual components of the MetS were considered categorically, youth with SBEs and OBEs differed on HDL-c (χ^2=6.23, p=0.013), with 71.3% of SBE compared to 51.4% of OBE having low HDL cholesterol. However, after applying a Bonferroni correction for the multiple comparisons made, this difference was not considered significant.

4. DISCUSSION AND CONCLUSIONS

In a convenience sample of adolescents, we found LOC was associated with some elements of metabolic dysfunction, even after adjusting for adiposity and other potentially

confounding variables. Adolescents with LOC had higher SBP and LDL-c compared to those without LOC. Furthermore, youth with OBEs had higher LDL-c compared to those with SBEs. Higher LDL-c and elevated blood pressure are both associated with heightened risk of cardiovascular disease (Wilson, et al., 1998). Our findings are consistent with adult (Epel, et al., 2004; Hudson, et al., 2010; Taylor, et al., 1999) and child (Tanofsky-Kraff, et al., 2012) research demonstrating relationships between LOC and components of metabolic dysfunction, independent of adiposity.

LOC may be associated with metabolic dysfunction based on macronutrient selection. Although youth with LOC do not necessarily consume more energy at meals than those without LOC, LOC episodes are characterized by greater consumption of carbohydrates, including snacks and desserts, and less from protein (Tanofsky-Kraff, et al., 2009). High intake of sweets and snacks could potentially contribute to worsened metabolic factors in youth, including higher LDL-cholesterol and blood pressure.

LOC episode size was independently associated with LDL-c. It is plausible that those with OBEs may have elevated LDL-c compared to those with SBEs, given the great amount of food consumed in a typical episode. Further, OBEs may be more indicative of greater eating pathology compared to SBE, which may in turn promote the consumption of more foods higher in cholesterol.

Strengths include the large sample size, inclusion of both non-overweight and overweight youth, and use of a well-validated interview measure to assess LOC. The direct estimation of fat mass and metabolic dysfunction using criterion methods as opposed to relying on BMI and self-reported metabolic function are strengths. The cross-sectional design, however, does not allow for understanding directionality of the relationship between LOC and metabolic function. Our sample included a combination of treatment-seeking, prevention-seeking, and non-treatment-seeking youth, thus results may not be generalizable to all children. Additionally, given the limited number of boys, we were unable to run separate analyses by sex.

LOC eating may represent a behavioral marker upon which to focus early intervention to prevent MetS, regardless of episode size. Youth with LOC who experience SBEs and OBEs may potentially benefit from interventions to reduce LOC to improve metabolic health.

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Abbreviations

HDL	high-density lipoprotein
LDL	low-density lipoprotein
LOC	loss of control eating
BMI	body mass index
BMI-z	body mass index z-score
EDE	eating disorder examination
DXA	dual-energy x-ray absorptiometry

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Table 1

Baseline demographic and anthropometric characteristics of youth with and without loss of control eating

	Total Sample				LOC-only Sample			
Variable	**Loss of Control Eating**[a]	**No Loss of Control Eating**	***t* or χ^2**	***P*-Value**	**OBE ± SBE**	**SBE only**	***t* or χ^2**	***P*-Value**
N	150	179			70	80		
Age (y)[b]	14.8±1.6	14.8±1.5	0.35	0.73	15.2±1.5	14.5±1.7	2.49	0.015
Sex			10.56	0.001			3.72	0.05
%Female	94%	82%			90%	98%		
Race (%)			30.72	<0.001			5.99	0.31
Non-Hispanic White	43%	23%			36%	49%		
Non-Hispanic Black	33%	37%			34%	31%		
Asian	7%	9%			9%	5%		
Multiple or Other race/ethnicities	18%	31%			21%	15%		
Socioeconomic Status (median)	2	3	7.86	0.17	3	2	4.75	0.32
BMI (kg/m^2)[b,c]	28.0±5.3	26.2±7.9	2.05	0.04	27.7±6.5	28.2±4.2	0.54	0.59
BMI-z[b,d]	1.63±0.85	1.44±1.15	1.43	0.16	1.57±0.94	1.72±0.69	0.77	0.44
Height (cm)[b]	163.4±7.9	164.2±7.9	0.40	0.37	163.7±7.7	163.0±8.1	0.59	0.56
Fat Mass (kg) [b, e]	31.1±13.0	31.2±17.4	0.06	0.96	31.9±16.2	30.4±9.3	0.65	0.51
Lean Mass (kg) [b, f]	48.8±8.1	50.3±10.3	1.51	0.13	50.3±8.5	47.5±7.6	2.09	0.04

[a] LOC= Loss of Control Eating, defined as the presence of at least 1 episode, irrespective of size, in the month prior to assessment on the Eating Disorder Examination.

[b] M ± SD

[c] BMI= Body Mass Index

[d] BMI-z= Body mass index standard deviation score for age and sex, calculated according to the Centers for Disease Control and Prevention 2000 growth charts.[22]

[e] Fat Mass, as measured by either by dual-energy x-ray absorptiometry (DXA) or air displacement plethysmography (Bod Pod). Measures of adiposity were adjusted to account for known differences between the different assessments.[23]

[f] Lean Mass, as measured by either by dual-energy x-ray absorptiometry (DXA) or air displacement plethysmography (Bod Pod). Measures of lean mass were adjusted to account for known differences between the different assessments.[23]

Table 2

Comparison of youth with and without loss of control eating on metabolic components and comparison of youth with loss of control eating on episode size and metabolic components. Data from MANCOVA adjusted for age, race, sex, fat mass (kg), height (cm), and treatment-seeking status.

	All Participants				Participants with Loss of Control eating			
Variable	**Loss of Control Eating**	**No Loss of Control Eating**	**F or χ^2**	***P*-Value**	**OBEs ± SBEs**	**SBEs only**	**F or χ^2**	***P*-Value**
n	150	179			70	80		
Systolic blood pressure (mm Hg); z-score [a]	119.2±0.9 0.78±0.09	115.5±0.8 0.41±0.08	10.36	0.001	118.2±1.1 0.76±0.10	119.5±1.0 0.92±0.10	1.27	0.262
Diastolic blood pressure (mm Hg); z-score [a]	66.5±0.7 0.13±0.06	65.6±0.6 0.05±0.05	0.75	0.39	65.6±0.9 0.03±0.08	67.3±0.85 0.21±0.08	1.96	0.163
Waist circumference (cm)[a]	89.5±0.7	88.5±0.6	0.98	0.32	88.9±0.9	89.2±0.8	0.17	0.686
Triglycerides (mg/dL) [a]	87.4±3.9	88.3±3.6	0.03	0.87	81.1±5.2	91.3±4.8	1.60	0.208
HDL-cholesterol (mg/dL) [a]	47.0±1.0	49.2±0.9	2.76	0.10	50.3±1.3	45.8±1.2	4.03	0.05
LDL-cholesterol (mg/dL) [a]	88.3±3.0	75.4±2.7	9.90	0.002	94.1±3.9	80.0±3.7	6.30	0.013
Plasma glucose (mg/dL) [a]	86.7±0.6	87.0±0.6	0.14	0.71	87.4±0.9	86.0±0.8	0.83	0.37
Metabolic syndrome (%)[b]	7.3%	7.8%	0.001	0.99	4.3%	10%	1.85	0.17

[a] m±SE

[b] Metabolic syndrome defined using age- and sex-specific percentile-based cut-off definition commonly used in previous reports (23, 24) values of at least 90th percentile for waist circumference, systolic or diastolic blood pressure, and triglycerides (25) and no higher than 10th percentile for HDL cholesterol, and a fasting glucose value of at least 100 mg/dL was used to indicate impaired fasting glucose. Metabolic syndrome was considered present when a child met the cut-points for at least three of these factors.