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Metabolic Characterization of Adults with Binge Eating in the General Population: The Framingham Heart Study

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Abstract

OBJECTIVE—To describe the metabolic profile of individuals with objective binge eating (OBE) and to evaluate whether associations between OBE and metabolic risk factors are mediated by body mass index (BMI).

DESIGN AND METHODS—Participants from the Framingham Heart Study, Third Generation and Omni 2 cohorts (n = 3551, 53.1% women, mean age 46.4 years) were screened for binge eating. We used multivariable-adjusted regression models to examine the associations of OBE with metabolic risk factors.

RESULTS—The prevalence of OBE was 4.8% in women and 4.9% in men. Compared to non-binge eating, OBE was associated with higher odds of hypertension (OR 1.85, 95% CI 1.32–2.60), hypertriglyceridemia (OR 1.42, 95% CI 1.01–2.01), low HDL (OR 1.70, 95% CI 1.18–2.44), insulin resistance (OR 3.18, 95% CI 2.25–4.50) and metabolic syndrome (OR 2.75, 95% CI 1.94–3.90). Fasting glucose was 7.2 mg/dl higher in those with OBE (p=0.0001). Individuals with OBE had more visceral, subcutaneous and liver fat. Most of these associations were attenuated with adjustment for BMI, with the exception of fasting glucose.

CONCLUSIONS—Binge eating is associated with a high burden of metabolic risk factors. Much of the associated risk appears to be mediated by BMI, with the exception of fasting glucose.

Keywords

Epidemiology; Population; Prevention; Binge Eating; Obesity

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INTRODUCTION

Obesity is a significant health problem in the United States, affecting more than one-third of U.S. adults and increasing the risk for type 2 diabetes, coronary heart disease and all-cause mortality.[1, 2] Despite awareness of this problem, effective strategies to prevent obesity have been elusive.

"Binge eating" is a behavior marked by consumption of large amounts of food in a short time frame, accompanied by a sense of lack of control over eating. "Binge eating disorder" (BED) is a condition of chronic and recurrent binge eating (at least once-weekly for 3 months) without compensatory behavior such as purging.[3] It is thought to be the most common eating disorder in the US, and is particularly prevalent in obese people seeking treatment, among whom BED prevalence may be 30% or greater.[4] Treatment seeking individuals with BED have high rates of metabolic syndrome (32–60%).[5–7] Estimates of BED prevalence among individuals with type 2 diabetes from small clinic-based samples have varied widely, from 2.5% to over 25%.[8–11] A five-year longitudinal study of 134 individuals with BED compared to controls matched for age, sex and body mass index (BMI) showed that BED was associated with an increased risk for reporting one or more components of the metabolic syndrome, even after adjustment for baseline BMI, suggesting the possibility of non-BMI-mediated mechanisms.[12] If these associations are present in the general population, binge eating may represent an important clinical target for identifying individuals at high risk for obesity and adverse metabolic outcomes.

While prior work has pointed to a strong association between BED and obesity, and a high rate of metabolic syndrome among selected treatment-seeking obese individuals with BED, a detailed metabolic characterization of individuals with binge eating derived from the general adult population has not been carried out. Studies that make use of directly measured risk factors, which are not susceptible to reporting bias, are needed. Thus, the purpose of the present investigation was to examine the association between binge eating behavior and metabolic outcomes in a large population-based cohort with robust measurement of risk factors, and further to examine whether observed associations were mediated by BMI. A formal diagnosis of BED requires associated psychiatric symptoms which we did not assess, thus we used the term objective binge eating (OBE) to describe individuals who fulfilled behavioral criteria for BED.

We also sought to characterize the metabolic risk associated with "subjective binge eating (SBE)," which describes the sensation of loss of control over eating without objective overeating.[13] SBE appears to be a less severe manifestation of binge eating, however it is still associated with obesity and psychological stressors of disordered eating.[13, 14] When grouped together, OBE and SBE are termed "loss of control eating." We hypothesized that binge eating would be associated with an increased burden of metabolic risk factors and that some of this burden would be independent of BMI.

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METHODS

Study Sample

The Framingham Heart Study is a large population-based cohort study of cardiovascular disease and its risk factors, which started in Framingham, Massachusetts in 1948, beginning with the Original Cohort.[15] Participants in the current study were drawn from the Third Generation (Exam 2) and Omni 2 (Exam 2) Cohorts.

Recruitment of the Third Generation began in November 2001. The second examination cycle began in 2008 and continued through 2011. A total of 3411 participants completed the exam, of whom 3375 answered questions about binge eating and provided blood samples. After excluding individuals with missing covariates, 3272 participants from Generation 3 Exam 2 were available for analysis.

The Omni cohorts were initiated in 1994 to reflect the increasing ethnic diversity of the community; they include African-American, Hispanic, Asian, Indian, Pacific Islander and Native American participants. Recruitment of the second Omni cohort began in 2003. Omni 2 exam 2 ran from 2009 through 2011 and was contemporaneous with the second examination of the Third Generation cohort. A total of 321 participants completed the exam, of whom 318 answered questions about binge eating and provided blood samples. After excluding individuals with missing covariates, 279 participants from Omni 2 Exam 2 were available for analysis. Thus the total sample size was 3551.

Study protocols and procedures were approved by the institutional review board at Boston University. Written informed consent was obtained from all participants.

Binge Eating Assessment

We assessed patterns of binge eating using questions drawn from a standard questionnaire (Questionnaire on Eating and Weight Patterns-Revised) that is widely used for the diagnosis of BED.[16] The current study was designed using the definition of BED in the prior version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), which differs from the most recently published definition (DSM-5) in duration and frequency of binge eating.[17]

Participants were asked “Do you often feel that you can’t control what or how much you eat?” and “Do you often eat, within any 2-hour period, what most people would regard as an unusually large amount of food?” If participants responded yes to either of these questions, they were then asked to quantify the frequency of this behavior over the previous 6 months. Choices were “Less than once per month,” “Once per month,” “Two to three times per month,” “Once per week,” and “Twice per week or more.” “Clinical OBE” was defined as “Yes” to the first two questions followed by a frequency of “Twice per week or more” for the third question. “Subclinical OBE” was defined as “Yes” to the first two questions followed by any of the lesser frequencies in response to the third question. SBE was defined as “Yes” to the first question about uncontrolled eating but “No” to the second question. Individuals who answered “No” to the first question were deemed non-binge eating (NBE).

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Metabolic Risk Factor Assessment

Key metabolic risk factors were measured at the contemporaneous examination. BMI, defined as weight in kilograms (assessed using a Detecto scale, Webb City, Missouri) divided by the square of the height in meters was measured as part of each examination cycle. Seated systolic and diastolic blood pressures were measured on site manually using a mercury column sphygmomanometer. Waist circumference was measured at the level of the umbilicus and reported to the nearest quarter inch. Plasma glucose, insulin (for HOMA-IR calculation), total cholesterol, HDL cholesterol, triglycerides, AST and ALT were measured from fasting morning samples. Insulin was measured via enzyme-linked immunosorbent assay (Roche e411 immunochemistry analyzer).

Hypertension was defined as systolic blood pressure ≥ 140 mm Hg, diastolic blood pressure ≥ 90mm Hg or on treatment. Hypertriglyceridemia was defined as triglycerides > 150 mg/dL or on lipid-lowering treatment. Low HDL was defined as < 40mg/dL in men or < 50mg/dL in women. Type 2 diabetes was defined as a fasting glucose ≥ 126 mg/dL, casual glucose ≥ 200 mg/dL or on diabetes treatment. Metabolic syndrome was defined using modified ATP III criteria.[18] Obesity was defined as BMI ≥ 30 kg/m2. HOMA-IR was dichotomized at the 75th percentile; individuals with diabetes were excluded for all HOMA-IR analyses.

Finally as a summary measure, we calculated the Framingham risk score (FRS), which estimates the 10-year risk of any cardiovascular event including coronary, cerebrovascular and peripheral arterial disease and heart failure.[19] The score is derived from a composite of age, blood pressure, cholesterol, smoking status, and the presence of diabetes.

Multidetector CT Substudy

A subset (n=1496) of participants underwent multidetector computed tomography of the chest and abdomen using Discovery VCT 64-slice PET/CT scanner (GE Healthcare). Beginning at approximately 2 cm above the S1 vertebrae, 30 contiguous 5-mm thick slices (120kVP; 100–300mA dependent on BMI) were acquired. Subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) volumes were assessed (Aquarius 3D Workstation, TeraRecon Inc., San Mateo, CA). To determine fat quality, an image display window width of −195 to −45 Hounsfield units (HU) and a window center of −120 HU were used. The abdominal muscular wall separating the visceral and subcutaneous compartments was traced manually. Inter-reader reproducibility was assessed by 2 independent readers measuring VAT and SAT on a subset of 46 randomly selected participants. Intra-reader correlations were 0.99 for VAT and 0.99 for SAT.

Liver attenuation, as a marker of liver fat, was measured in HU, as we have previously reported.[20] Additionally, a calibration phantom with a water equivalent compound (CT-Water, Light Speed Ultra; General Electric, Milwaukee, WI) and calcium hydroxyapatite was placed under each participant. This was used to calculate a liver-to-phantom ratio (LPR) of the participant's liver fat divided by the phantom fat in HU. LPR is thus an indexed unitless standard with a lower value indicating higher liver fat content.[21]

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Assessment of Covariates

Covariates were ascertained via interview or measured directly at the contemporaneous examination. These included age, sex, body mass index, height, ethnicity and education status. Current smoking was assessed via physician interview at the time of examination and defined as smoking at least 1 cigarette daily for the previous year. Alcohol intake (dichotomized at > 14 drinks/week in men or > 7 drinks/week in women) was quantified by a series of questions at the physician interview. Depressive symptoms were assessed via Center for Epidemiologic Study Scale (CES-D) with a score of 16 or greater required for diagnosis of depressive symptoms.[22]

Statistical Analysis

The binge eating groups were first characterized by standard demographic, anthropometric and laboratory parameters. The characteristics of patients with clinical and subclinical OBE were generally similar and approximately 90% of individuals designated as subclinical OBE reported a frequency of either “Two to three times per month” or “Once per week” (Supplemental Table 1). Thus clinical and subclinical OBE were merged into a single group called OBE for subsequent analyses. Overall, age-specific and sex-specific prevalence of binge eating behaviors were calculated.

We first performed an ANOVA to test whether there was a significant difference among binge eating groups. We then pursued pairwise comparisons (OBE compared to NBE and SBE compared to NBE) to better characterize the nature of the differences. Multivariable-adjusted linear and logistic regression models were used to assess the relationship between binge eating status and metabolic risk factors (continuous and dichotomized). The first multivariable model included adjustment for age, sex, smoking history, alcohol intake, education and depressive symptoms. Model 2 was additionally adjusted for BMI. Finally we tested the interaction between OBE and BMI for each metabolic risk factor (continuous and dichotomized). To characterize the direction of significant interactions, we then performed multivariable adjusted regression analyses stratified by BMI category (normal weight, overweight or obese) as exploratory analyses.

All statistical analyses were conducted using SAS version 9.3. We considered two-tailed p values <0.05 to be statistically significant; there was no adjustment for multiple comparisons.

RESULTS

Study Sample Characteristics

Demographic and descriptive characteristics for the overall cohort and for categories of binge eating are shown in Table 1. The mean age was approximately 46.6 years and slightly more than half were women.

The overall prevalence of OBE was nearly 5% (4.9% among men, 4.8% among women); the overall prevalence of SBE was 7.1% (4.7% among men, 9.2% among women) (Table 2).

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Binge eating prevalence was not significantly different among age groups (p=0.43) and was more common among obese and overweight individuals (p <0.0001).

Metabolic Characterization of Patients with OBE and SBE

There was a significant difference (ANOVA p<0.01) among binge eating groups for nearly all outcomes (continuous and dichotomous). Thus, we pursued multivariable-adjusted regression models with pairwise comparisons. OBE was associated with more adverse levels of several metabolic parameters (Table 3) compared to NBE. For example, systolic blood pressure was 3.6 mm Hg higher (p=0.001) in OBE compared to NBE. HDL-cholesterol was 4.3 mg/dL lower (p=0.0009), fasting glucose was 7.2 mg/dL higher (p<0.0001), and ALT was 4.3 mg/dL higher (p=0.002) in individuals with OBE. Among the participants in the CT substudy, those with OBE had higher VAT volume (+522.5 cm3, p=0.0003), higher SAT volume (+1012.5 cm3, p< 0.0001) and more fatty liver (−5.9 HU, p=0.0001). After BMI adjustment, these associations were no longer significant with the notable exception of fasting glucose. After adjustment for BMI, fasting glucose remained 4.3 mg/dL higher (p = 0.001) among individuals with OBE. For individuals with SBE, several outcomes including fasting glucose were higher compared to NBE (Table 3). However, these differences did not persist when additional adjustment for BMI was performed.

Based on the Framingham risk score, individuals with OBE had a 1.6% higher 10-year risk of a CVD event compared to NBE (p < 0.0001); SBE had a 0.9% higher 10-year risk (p=0.002, Table 3). These results were also attenuated by BMI adjustment.

Results for dichotomized outcomes are shown in Table 4. Individuals with OBE had a higher burden of metabolic risk factors including hypertension (OR 1.85, 95% CI 1.32–2.60), low HDL (OR 1.70, 95% CI 1.18–2.44), type 2 diabetes (OR 2.95, 95% CI 1.75–4.96) and metabolic syndrome (OR 2.75, 95% CI 1.94–3.90) compared to NBE participants. These associations were attenuated when additionally adjusting for BMI. Results were similar for SBE. We found significant interactions between BMI and OBE for several outcomes (Tables 5 and 6). To characterize significant interactions, we performed exploratory analyses examining the associations between OBE and outcomes stratified by BMI category. For the continuous outcomes, the effects were somewhat stronger among obese individuals. For dichotomous outcomes, if there was an interaction, it was that the relationship between OBE and the outcome was stronger among normal weight individuals and was attenuated in obese individuals. For example, the odds ratios of OBE for type 2 diabetes and metabolic syndrome were higher in normal weight individuals than in obese individuals.

After data collection was completed for the present study, the definition of BED was revised in the DSM-5.[3] The duration and frequency required for diagnosis was reduced to “once per week or more for three months.” Because of this new definition, we conducted a secondary analysis of OBE using a frequency of “once per week or more for six months” (the closest approximation to the current definition available from the data we collected). This secondary analysis did not change the findings substantially; these results are included in Supplementary Tables 3 and 4.

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DISCUSSION

Principal Findings

Our principal findings are threefold. First, our study demonstrates that binge eating is common, with approximately 5% of our sample reporting OBE; this was equal across gender and age groups. An additional 7% of our sample (9.2% of woman, 4.7% of men) reported SBE. Second, individuals with OBE have a high burden of metabolic risk factors including obesity, hypertension, hypertriglyceridemia, and type 2 diabetes compared to NBE. They had more visceral, subcutaneous and hepatic fat. SBE, which consists only of the feeling of a lack of control over eating without reported objective overeating episodes, also carried a higher risk of obesity and type 2 diabetes. Third, the increased metabolic risk factors associated with OBE and SBE are largely attenuated after adjusting for BMI, suggesting that much of the risk factor burden is mediated by excess body weight. A notable exception was fasting glucose, which was higher in those with OBE even after BMI adjustment.

There were several significant interactions between OBE and BMI. The analysis stratified by BMI category found that the association of OBE with continuous outcomes was stronger among obese, while the association with dichotomous outcomes was stronger in normal weight individuals and was less pronounced in obese individuals. This pattern is likely due to the lower baseline risk among normal weight individuals.

In the Context of the Current Literature

Our findings expand upon previous work that demonstrated associations with metabolic syndrome in treatment-seeking overweight and obese samples with BED.[5, 6, 23] In our large community-based sample that included individuals across the body weight spectrum, we observed higher odds ratios for obesity and metabolic syndrome in those with OBE compared to NBE. Our results demonstrate a higher burden of risk factors among binge eaters across a wide range of metabolic function. Our findings show that binge eating is a relevant problem in the general population and that individuals who binge eat are at high risk for cardiovascular disease.

Our findings differ from a prior longitudinal study of 268 individuals,[12] which showed a higher risk for components of metabolic syndrome in BED that persisted after adjustment for BMI. There are some important differences which may explain our disparate findings. First, the sample was drawn from treatment-seeking overweight and obese people with mean baseline BMI ~35 kg/m^2. Second, the baseline prevalence of metabolic syndrome was low (2.5%), suggesting the possibility of under-ascertainment. Metabolic risk factors were self-reported, which may be less accurate and thus quantification of associations less reliable.

Our findings also demonstrated that individuals with OBE and SBE had higher volumes of VAT and SAT; the fat quality assessments showed lower Hounsfield unit attenuation, which we have previously shown to be associated with more adverse metabolic risk factor profiles.[24] These results add to the clinical picture of higher metabolic burden in individuals with binge eating.

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Potential Mechanisms

Our results demonstrate that binge eating is associated with a host of adverse metabolic factors including type 2 diabetes, hypertension and measures of ectopic fat including liver fat and visceral fat. The association with obesity explains much of this metabolic burden. A notable exception was fasting glucose, which was higher among individuals with OBE even after BMI adjustment. We considered the possibility that some individuals with OBE in our sample may have binge eaten overnight prior to their blood draw and thus their samples were truly non-fasting. If this had been the case however, one might have expected that triglycerides would similarly have been elevated independent of BMI, but this was not observed. Therefore, this is unlikely to fully explain our findings.

Further research is needed to confirm and explore the finding of BMI-independent higher fasting glucose among OBE individuals, however the published literature suggests several potential mechanisms that may explain the association.

Leptin is a hormone produced by adipose tissue that reduces hepatic glucose production via suppression of glucagon.[25] In normal physiology, the majority of fasting glucose production is derived from the liver due to a combination of glycogenolysis and gluconeogenesis.[26] One prior study found that ingestion of an entire day's caloric intake in a single meal caused higher fasting glucose and impaired the normal post-prandial leptin response.[27] Thus, the observed BMI-independent fasting glucose elevation among OBE individuals in the present study may be due to binge eating causing insufficient leptin production.

Another possibility is that binge eating may disrupt adipocytokines, which in turn have important mediatory effects on metabolic pathways. For example, adiponectin levels are reduced in women with BED compared to healthy women.[28] Adiponectin mediates both gluconeogenesis and fatty acid oxidation, and promotes insulin sensitivity.[28, 29] Deficiency of adiponectin may lead to higher fasting glucose among binge eaters.

Finally it is possible that concentration of caloric intake over a short period causes more sustained peaks in glucose and insulin related to rapid absorption. One study found that ingestion of daily food intake over 3 larger meals compared to over 13 smaller snacks, with calories held constant, resulted in higher mean glucose and insulin levels.[30] The authors attributed these differences to the rate at which calories were presented to the body.

Implications

Our findings suggest that binge eating is particularly prevalent among obese individuals. It is associated with a significant burden of metabolic factors that are known to increase the risk for cardiovascular disease and mortality. In the present study, individuals with OBE and SBE had a higher estimated 10-year risk of CVD based on Framingham risk score. This result suggests that metabolic risk factors and cardiovascular disease may represent significant co-morbidities in individuals with binge eating. Furthermore, the observation that not all of the higher fasting glucose associated with binge eating is mediated by BMI merits further investigation to elucidate hormonal pathways and other mechanisms at play in conferring this risk.

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Strengths and Limitations

There are several strengths to our study. First, our cohort was drawn from the community-based Framingham Heart Study. Metabolic risk factors were well-characterized and we did not depend on self-report. In addition, our sample was large, lending more power to detect differences. Finally, we were able to look across a broad spectrum of binge eating behaviors. Some limitations warrant mention. Our population is primarily Caucasian, limiting generalizability to other races/ethnicities. The cross-sectional design precludes inference of temporality. Because our study is observational, we cannot infer causality between binge eating and associated risk factors. Another limitation is that we did not exclude the possibility that some individuals classified as binge eating may have met criteria for bulimia nervosa. Bulimia nervosa is also characterized by binge eating, but unlike BED includes inappropriate compensatory behaviors (such as purging, excessive exercise or fasting), which we did not assess.[31] However, because bulimia nervosa is considerably less common than BED and tends to affect a younger subset of the population than that represented in our sample, this is unlikely to have significantly altered the results.[32, 33] Finally, the use of questionnaire methods to assess binge eating may have led to an overestimation of its prevalence relative to that found by interview methods.[34]

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WHAT IS ALREADY KNOWN ABOUT THIS SUBJECT?

Binge eating disorder is known to be prevalent among individuals seeking treatment for obesity.

Prior work has suggested that some associations between binge eating and metabolic risk factors may be independent of BMI.

WHAT DOES THIS STUDY ADD?

A detailed metabolic characterization of binge eating disorder from a large generally healthy adult population.

Most of the metabolic risk associated with binge eating disorder is mediated by BMI. Our results found that binge eating was associated with impaired fasting glucose after accounting for BMI, suggesting that binge eating may be associated with glucose metabolism independent of its associations with weight.

Our study found that binge eating is a common condition affecting men and women across age groups and carries a high burden of metabolic risk factors; thus it is an important contributor to metabolic and cardiovascular risk in the general population.

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Table 1

Characteristics of binge eating

Continuous Characteristics: mean (SD) except Triglycerides, Drinks/Week and Liver Phantom Ratio shown as Median (25^{th}–75^{th} percentile)
Categorical/Dichotomous Characteristics: n (%)

	Objective binge eating (n=172)	Subjective binge eating (n=252)	Non-binge eating (n=3127)
Age, years	47.0 (9.3)	47.5 (9.4)	46.5 (9.1)
Women (%)	91 (52.9)	173 (68.7)	1620 (51.8)
Race/Ethnicity† (%)			
White	167 (97.1)	245 (97.2)	2980 (95.3)
Hispanic	5 (2.9)	12 (4.8)	102 (3.3)
Black	5 (2.9)	5 (2.0)	79 (2.5)
Asian	1 (0.6)	5 (2.0)	82 (2.6)
Pacific Islander	1 (0.6)	0 (0.0)	2 (0.1)
Native American or Alaskan	2 (1.2)	3 (1.2)	40 (1.3)
Education (%)			
< High School	0 (0)	1 (0.4)	34 (0)
High School Graduate	22 (12.8)	34 (13.5)	403 (12.9)
Some College	63 (36.6)	87 (34.5)	886 (28.3)
College Graduate	87 (50.6)	130 (51.6)	1804 (57.7)
BMI (kg/m^2)	33.0 (7.0)	30.9 (6.9)	27.5 (5.4)
Waist Circumference (cm)	109 (17)	103 (17)	96 (15)
BMI>= 30 kg/m2 (%)	120 (69.8)	118 (46.8)	838 (26.8)
Physical Activity Index	35.8 (7.4)	35.6 (6.4)	36.5 (6.5)
Current Smokers (%)	27 (15.7)	17 (6.8)	333 (10.7)
VAT, cm^3	2706 (1430)	2422 (1520)	2171 (1360)
SAT, cm^3	4339 (1793)	4480 (1858)	3203 (1578)
VAT/SAT Ratio	0.70 (0.54)	0.58 (0.44)	0.72 (0.44)
VAT, HU	−96 (7)	−95 (6)	−94 (7)
SAT, HU	−107 (4)	−108 (3)	−106 (4)
ALT, mg/dL	29 (17)	23 (13)	25 (17)
AST, mg/dL	22 (9)	21 (8)	22 (10)
Liver Phantom Ratio (Median (25^{th}–75^{th} %ile)	0.35 (0.30–0.38)	0.35 (0.28–0.38)	0.36 (0.33–0.38)

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	Objective binge eating (n=172)	Subjective binge eating (n=252)	Non-binge eating (n=3127)
Alcoholic Drinks/Week	1.0 (0.0–4.5)	1.5 (0.0–4.0)	3.0 (0.5–7.3)
High Alcohol Intake (%)(> 14/wk men or > 7/wk women)	15 (8.7)	22 (8.7)	466 (14.9)
Depressive symptoms (%)(CES-D ≥ 16)	44 (26.6)	37 (14.7)	255 (8.2)
Total CES-D	10.8 (9.4)	8.6 (8.7)	5.7 (6.9)
Diabetes (%)	21 (12.2)	28 (11.1)	138 (4.4)
Fasting glucose (mg/dL)	105 (31)	99 (22)	96 (17)
HOMA-IR£ > 75th %ile (%)	71 (47.0)	75 (33.5)	695 (23.3)
HOMA-IR£	3.40 (2.54)	2.68 (1.97)	2.23 (1.72)
Low HDL Cholesterol‡ (%)	46 (26.7)	47 (18.7)	514 (16.5)
Hypertriglyceridemia†† (%)	65 (37.8)	87 (34.5)	929 (29.7)
Total Cholesterol, mg/dL	187 (44)	187 (36)	186 (35)
HDL Cholesterol, mg/dL	54 (17)	59 (17)	60 (18)
Triglycerides (mg/dL)	114 (79–152)	101 (74–143)	92 (67–132)
Hypertension (%)	55 (32.0)	67 (26.6)	669 (21.4)
Systolic Blood Pressure (mm Hg)	120 (13)	117 (14)	116 (14)
Diastolic Blood Pressure (mm Hg)	76 (9)	74 (9)	74 (9)
Metabolic Syndrome (%)	65 (37.8)	71 (28.2)	586 (18.7)
Lipid-lowering medication (%)	38 (22.1)	50 (19.8)	509 (16.3)
Anti-hypertension medication (%)	43 (25.0)	58 (23.0)	519 (16.6)
Framingham Risk Score, %	7.5 (8.5)	5.8 (6.0)	5.5 (5.9)

† Participants permitted to self-identify as more than 1 race thus percentages add to > 100%

£ HOMA-IR analyses exclude participants with diabetes (n=150 for OBE, 223 for SBE, 2976 for NBE)

‡ Low HDL cholesterol is defined as < 40mg/dL in men or < 50mg/dL in women

†† Hypertriglyceridemia is defined as ≥ 150mg/dL

BMI=body mass index; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue; HU = Hounsfield units; ALT = alanine aminotransferase; AST = aspartate aminotransferase; CES-D = Center for Epidemiologic Studies Depression Scale; HOMA-IR = homeostatic model assessment for insulin resistance

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Table 2

Overall, Sex-Specific and Age-Specific Prevalence of binge eating

	Objective binge eating	Subjective binge eating	Non-binge eating
Overall	4.8 (172)	7.1 (252)	88.1 (3127)
Gender			
Women	4.8 (91)	9.2 (173)	86.0 (1620)
Men	4.9 (81)	4.7 (79)	90.4 (1507)
Age Strata:			
Age < 35	4.3 (15)	6.3 (22)	89.5 (314)
35 ≤ Age < 45	4.7 (50)	6.3 (67)	89.0 (950)
45 ≤ Age < 55	4.9 (71)	7.5 (109)	87.6 (1266)
55 ≤ Age < 65	4.1 (31)	7.4 (45)	87.5 (531)
Age ≥ 65	6.3 (5)	11.3 (9)	82.5 (66)
BMI Category			
Normal weight	1.5 (18)	4.1 (49)	94.4 (1124)
Overweight	2.7 (34)	6.6 (85)	90.7 (1165)
Obese	11.2 (120)	11.0 (118)	77.9 (838)

Data shown are % (n)

BMI = body mass index

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Table 3

Multivariable-adjusted association of Binge Eating and Metabolic Risk Factors (Continuous outcomes)

	OBE vs. NBE		SBE vs. NBE	
Outcome*	**Mean difference (95% CI)**	**p-value**	**Mean difference (95% CI)**	**p-value**
Systolic Blood Pressure (mm Hg)				
MV Model**	3.6 (1.3–6.0)	0.001	1.6 (−0.3–3.6)	0.13
MV Model + BMI	0.1 (−2.4–2.2)	1.00	−0.7 (−2.6–1.2)	0.63
Diastolic Blood Pressure (mm Hg)				
MV Model**	2.2 (0.5–3.8)	0.006	0.2 (−1.1–1.6)	0.91
MV Model + BMI	−0.19 (−1.79–1.41)	0.96	−1.25 (−2.58–0.07)	0.07
Total Cholesterol (mg/dL)				
MV Model**	2.2 (−4.1–8.6)	0.69	1.9 (−3.4–7.2)	0.68
MV Model + BMI	1.4 (−5.2–7.9)	0.88	1.3 (−4.1–6.7)	0.83
HDL-Cholesterol (mg/dL)				
MV Model**	−4.3 (−7.1–1.5)	0.0009	−2.3 (−4.6–0.1)	0.06
MV Model + BMI	0.2 (−2.5–2.9)	0.98	0.7 (−1.6–2.9)	0.77
Log Triglycerides				
MV Model**	0.1 (0.0–0.2)	0.002	0.1 (0.0–0.2)	0.02
MV Model + BMI	0.0 (−0.1–0.1)	0.95	0.0 (−0.1–0.1)	0.98
Fasting glucose (mg/dL)				
MV Model**	7.2 (4.4–10.1)	<0.0001	2.8 (0.5–5.2)	0.01
MV Model + BMI	4.3 (1.5–7.2)	0.001	1.0 (−1.4–3.3)	0.58
Log HOMA-IR				
MV Model**	0.4 (0.3–0.5)	<0.0001	0.2 (0.1–0.3)	<0.0001
MV Model + BMI	0.0 (−0.1–0.1)	0.71	0.0 (−0.1–0.1)	0.90
BMI (kg/m2)				

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	OBE vs. NBE		SBE vs. NBE	
Outcome*	**Mean difference (95% CI)**	**p-value**	**Mean difference (95% CI)**	**p-value**
MV Model**	5.4 (4.4–6.4)	<0.0001	3.5 (2.6–4.3)	<0.0001
MV Model + BMI	---	---	---	---
VAT (cm3)				
MV Model**	522.5 (205.2–839.8)	0.0003	487.2 (219.7–754.8)	<0.0001
MV Model + BMI	−128.0 (−341.0–84.9)	0.34	−126.8 (−307.0–53.4)	0.22
SAT (cm3)				
MV Model**	1012.5 (587.0–1438.0)	<0.0001	1083.9 (725.8–1442.0)	<0.0001
MV Model + BMI	18.4 (−203.5–240.3)	0.98	149.4 (−38.0–336.7)	0.15
VAT/SAT Ratio				
MV Model**	0.0 (−0.1, 0.1)	0.99	0.0 (−0.1, 0.1)	0.84
MV Model + BMI	0.0 (−0.1, 0.1)	0.68	0.0 (0.0, 0.1)	0.32
VAT (HU)				
MV Model**	−2.4 (−4.1 to −0.6)	0.004	−2.1 (−3.6 to −0.6)	0.002
MV Model + BMI	0.7 (−0.6–2.1)	0.38	0.8 (−0.29–1.94)	0.20
SAT (HU)				
MV Model**	−1.3 (−2.4 to −0.2)	0.02	−1.5 (−2.5 to −0.6)	0.0005
MV Model + BMI	0.1 (−1.0–1.1)	0.99	0.2 (−1.1–0.6)	0.80
ALT (mg/dL)				
MV Model**	4.3 (1.3–7.2)	0.002	0.3 (−2.2–2.7)	0.96
MV Model + BMI	1.0 (−2.0–3.9)	0.72	−1.9 (−4.3–0.6)	0.17
AST (mg/dL)				
MV Model**	0.6 (−1.2–2.4)	0.69	−0.4 (−1.9–1.1)	0.77
MV Model + BMI	0.1 (−1.7–1.9)	0.99	−0.8 (−2.3–0.7)	0.44
Liver Attenuation (HU)				

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	OBE vs. NBE		SBE vs. NBE	
Outcome*	**Mean difference (95% CI)**	**p-value**	**Mean difference (95% CI)**	**p-value**
MV Model**	−5.9 (−9.3 to −2.5)	0.0001	−4.6 (−7.4 to −1.8)	0.0004
MV Model + BMI	−0.4 (−3.1–2.3)	0.93	−0.6 (−1.7–2.9)	0.82
Liver Phantom Ratio				
MV Model**	−0.02 (−0.04–0.00)	0.01	−0.02 (−0.03–0.00)	0.01
MV Model + BMI	0.00 (−0.02–0.02)	0.99	0.00 (−0.01–0.02)	0.83
Framingham Risk Score (%)				
MV Model**	1.6 (0.9–2.4)	<0.0001	0.9 (0.3–1.5)	0.002
MV Model + BMI	0.6 (−0.1–1.4)	0.12	0.3 (−0.4–0.9)	0.60

*Continuous outcomes adjusted for treatment: SBP and DBP adjusted for anti-HTN rx; total cholesterol, HDL and TG adjusted for lipid-lowering rx; fasting glucose and HOMA-IR adjusted for diabetes rx

**Multivariable model adjusts for Age, Sex, Smoking Status, Alcohol Intake, Depressive symptoms and Education

OBE = objective binge eating; SBE = subjective binge eating; NBE = non-binge eating; BMI = body mass index; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue; HU = Hounsfield units; ALT = alanine aminotransferase; AST = aspartate aminotransferase

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Table 4

Multivariable-adjusted association of binge eating and Metabolic Risk Factors (Dichotomous Outcomes)

	OBE vs. NBE		SBE vs. NBE	
Outcome	**Odds Ratios (95% CI)**	**p-value**	**Odds Ratios (95% CI)**	**p-value**
Hypertension				
MV Model*	1.85 (1.32–2.60)	0.0004	1.23 (0.91–1.65)	0.17
MV Model +BMI	0.93 (0.64–1.35)	0.69	0.74 (0.53–1.02)	0.07
Hypertriglyceridemia				
MV Model*	1.42 (1.01–2.01)	0.04	1.42 (1.05–1.90)	0.02
MV Model +BMI	0.82 (0.57–1.19)	0.29	0.99 (0.72–1.35)	0.93
Low HDL				
MV Model*	1.70 (1.18–2.44)	0.004	1.18 (0.84–1.65)	0.34
MV Model +BMI	0.99 (0.67–1.46)	0.97	0.80 (0.56–1.14)	0.22
Type 2 Diabetes				
MV Model*	2.95 (1.75–4.96)	<0.0001	2.67 (1.69–4.21)	<0.0001
MV Model +BMI	1.39 (0.78–2.47)	0.26	1.64 (1.00–2.69)	0.048
HOMA-IR > 75th %ile				
MV Model*	3.18 (2.25–4.50)	<0.0001	1.86 (1.38–2.52)	<0.0001
MV Model +BMI	1.19 (0.79–1.80)	0.41	0.97 (0.67–1.41)	0.87
Metabolic Syndrome†				
MV Model*	2.75 (1.94–3.90)	<0.0001	1.84 (1.34–2.51)	.0001
MV Model + BMI	1.20 (0.80–1.79)	0.37	0.98 (0.68–1.41)	0.92
Obesity (BMI ≥ 30 kg/m2)				
MV Model*	6.33 (4.49–8.93)	<0.0001	2.50 (1.91–3.27)	<0.0001
MV Model +BMI	N/A	N/A	N/A	N/A

*Multivariable model adjusts for Age, Sex, Smoking Status, Alcohol Intake, Depressive symptoms and Education

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OBE = objective binge eating; SBE = subjective binge eating; NBE = non-binge eating; BMI = body mass index; HOMA-IR = Homeostatic model assessment for insulin resistance

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Table 5

Interactions between OBE and BMI (Continuous outcomes)

	OBE-BMI Interaction	Normal weight with OBE (n=18)	Normal weight NBE (n=1124)	Overweight with OBE (n=34)	Overweight NBE (n=1165)	Obese with OBE (n=120)	Obese NBE (n=838)
Metabolic Risk Factor	**p**	**Adjusted mean**	**Adjusted mean**	**Adjusted mean**	**Adjusted mean**	**Adjusted mean**	**Adjusted mean**
HDL Cholesterol	0.01	71.5 mg/dL	68.6 mg/dL	55.1 mg/dL	57.3 mg/dL	51.4 mg/dL	51.9 mg/dL
Log Triglycerides	0.04	4.2 mg/dL	4.3 mg/dL	4.6 mg/dL	4.6 mg/dL	4.8 mg/dL	4.8 mg/dL
VAT	0.01	863 cm3	1015 cm3	2209 cm3	2142 cm3	3312 cm3	3367 cm3
SAT	0.02	2226 cm3	2030 cm3	3353 cm3	3000 cm3	4996 cm3	4865 cm3
VAT HU	0.001	−85.5 HU	−87.5 HU	−94.2 HU	−94.1 HU	−99.0 HU	−99.1 HU
SAT HU	<0.0001	−103.5 HU	−103.6 HU	−106.2 HU	−106.5 HU	−108.7 HU	−108.1 HU
ALT	0.05	19.5 mg/dL	19.1 mg/dL	28.8 mg/dL	25.7 mg/dL	32.3 mg/dL	31.7 mg/dL
Liver Phantom Ratio	0.04	0.38	0.38	0.33	0.35	0.30	0.30

Only significant interactions shown. Interactions were not significant for systolic or diastolic blood pressure, total cholesterol, triglycerides, glucose, log HOMA-IR, VAT/SAT Ratio, AST or Framingham Risk Score

OBE = objective binge eating; NBE = non-binge eating; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue; HU = Hounsfield units; ALT = alanine aminotransferase; AST = aspartate aminotransferase; HOMA-IR = homeostatic model assessment for insulin resistance

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Table 6

Interaction between OBE and BMI (Dichotomous Outcomes)

	OBE-BMI Interaction	Normal Weight: OBE (n=18) vs. NBE (n=1124)	Overweight: OBE (n=34) vs. NBE (n=1165)	Obese: OBE (n=120) vs. NBE (n=838)
Metabolic Risk Factor	**p**	**Odds Ratio**	**Odds Ratio**	**Odds Ratio**
Hypertriglyceridemia	0.004	1.4	1.0	0.9
Type 2 Diabetes	0.04	5.1	3.3	1.6
Metabolic Syndrome	0.002	3.2	1.1	1.3

Only significant interactions shown. Interactions were not significant for hypertension, low HDL cholesterol or HOMA-IR >75th percentile

OBE = objective binge eating; NBE = non-binge eating; BMI = body mass index; HOMA-IR = homeostatic model assessment for insulin resistance