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Biopsychosocial Correlates of Binge Eating Disorder in Caucasian and African American Women with Obesity in Primary Care Settings

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Abstract

This study examined racial differences in eating-disorder psychopathology, eating/weight-related histories, and biopsychosocial correlates in women ($n=53$ Caucasian and $n=56$ African American) with comorbid binge-eating disorder (BED) and obesity seeking treatment in primary care settings. Caucasians reported significantly earlier onset of binge eating, dieting, and overweight, and greater number of times dieting than African American. The rate of metabolic syndrome did not differ by race. Caucasians had significantly elevated triglycerides whereas African Americans showed poorer glycemic control (higher glycated hemoglobin A1c [HbA1c]), and significantly higher diastolic blood pressure. There were no significant racial differences in features of eating disorders, depressive symptoms, or mental and physical health functioning. The clinical presentation of eating-disorder psychopathology and associated psychosocial functioning differed little by race among obese women with BED seeking treatment in primary care settings. Clinicians should assess for and institute appropriate interventions for comorbid BED and obesity in both African American and Caucasian patients.

Keywords

Binge eating disorder; Obesity; Race; Women; Metabolic syndrome

Introduction

Binge eating disorder (BED) is defined in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) as recurrent binge eating (eating large quantities of food while experiencing a subjective sense of loss of control), marked distress about the binge eating and the absence of extreme weight compensatory behaviors. BED is the most prevalent eating disorder, affecting 2.6% of U.S. adults (Kessler

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et al., 2013). Unlike other specific eating disorders, the prevalence of BED is quite similar across racial and ethnic groups (Hudson, Hiripi, Pope, & Kessler, 2007; Marques et al., 2011). Relatively little is known about racial differences in the development or clinical manifestation of BED. A few community-based studies have reported some differences in clinical features between Caucasian and African American women with BED although the findings are somewhat mixed. Pike and colleagues (Pike, Dohm, Striegel-Moore, Wilfley, & Fairburn, 2001) found higher body mass index (BMI) and greater frequency of binge eating but lower levels of associated eating disorder psychopathology among African American than Caucasian women with BED. Striegel-Moore and colleagues (Striegel-Moore et al., 2005), in another community-based study, reported that risk factor variables and binge eating frequency differed little by race, although age at binge eating onset was earlier in Caucasian than African American women. Such community-based findings suggest that race may be associated with varied clinical presentations of BED and highlight the need for further work to address the relative dearth (and equivocal nature) of data.

Race does appear to play a role in treatment-seeking behaviors among persons with BED. Although African American and Caucasian adults with eating disorders report similar levels of psychosocial impairment, Caucasians are significantly more likely to access mental-health services (Marques et al., 2011). Little is known, however, about racial differences in clinical presentation of BED among treatment-seeking patients with BED. Grilo, Lozano, and Masheb (2005) reported differences in history and features of eating disorders across race groups that varied according to whether the samples were drawn from community or clinic samples. This suggests that there may be sampling biases by race associated with BED. Two large studies have examined racial differences in demographic and clinical variables of treatment-seeking patients with BED who participated in research studies at university-based specialty programs (Franko et al., 2012; Lydecker & Grilo, in press). Franko and colleagues (Franko et al., 2012), in a study which pooled BED treatment trial data from nine different research sites, reported that African American patients had higher BMI than Caucasian patients but neither binge eating frequency nor levels of associated eating disorder psychopathology differed by race. Lydecker and Grilo (Lydecker & Grilo, in press), in a study of a large treatment-seeking sample of adults with BED at a university-based program, reported that African American participants had higher BMI and more frequent binge eating but lower depression levels than Caucasian participants whereas levels of associated eating disorder pathology did not differ by race. In terms of developmental histories, Caucasian participants with BED reported younger ages at onset for obesity, dieting, and binge eating.

Thus, the available data regarding race and BED suggest that there may be some important differences in developmental histories (e.g., earlier onset for Caucasian patients) and current clinical manifestations (e.g., higher BMI and more frequent binge eating in African American patients) although there are only few differences in associated eating disorder psychopathology. The generalizability of such emerging data on race differences associated with treatment-seeking adults with BED, however, is uncertain for at least two reasons. First, data that come from university-based specialty clinic settings have generally had underrepresentation of racial and ethnic minority group participation (Franko et al., 2012). Second, research has indicated that racial minorities with eating disorders are more likely to

seek health care at generalist primary care settings than at specialist mental health programs (Crow, Peterson, Levine, Thuras, & Mitchell, 2004; Marques et al., 2011). Indeed, Grilo and colleagues (Grilo, White, Barnes, & Masheb, 2013) previously reported greater racial representation in BED research primary care settings, and increased likelihood of comorbid psychiatric disorders in African American patients, compared with Caucasian patients. Therefore, the aim of this study was to evaluate racial differences in the clinical presentation of treatment-seeking adults with BED and co-occurring obesity in primary care. In addition to evaluating the clinical domains (BMI, binge eating, associated eating disorder psychopathology, depression, and developmental onset of obesity, dieting, and binge eating) reported by Lydecker and Grilo (in press) with a university-based program, the present study provides new information on psychosocial, physical, and metabolic functioning by race.

Methods

Participants

Participants were 109 women (mean age 44.3 ± 11.8 years) who responded to advertisements for treatment studies for adults with BED and co-occurring obesity being conducted in primary care settings. All participants were obese ($\text{BMI} \geq 30 \text{ kg/m}^2$), and met *DSM-5* criteria thresholds for BED (we required a more stringent duration criterion of 6 months of at least once-weekly binge eating). Exclusion criteria for this treatment-seeking group were: $\text{BMI} > 50 \text{ kg/m}^2$, current anti-depressant therapy, severe psychiatric problems (lifetime bipolar disorders and schizophrenia, and current substance use dependence, assessed by the Structured Clinical Interview for DSM-IV [SCID-P; First, Spitzer, Gibbon, & Williams, 1995]), severe medical problems (e.g., cardiac and liver diseases), and uncontrolled hypertension, thyroid conditions, or diabetes. The study had IRB approval and written informed consent was obtained from all participants.

Assessment and measures

BED and eating disorder characteristics

The Eating Disorder Examination (EDE) (Fairburn & Cooper, 1993) 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 (binge eating defined as unusually large quantities of food coupled with a subjective sense of loss of control), objective overeating episodes (eating defined as unusually large quantities of food without a subjective sense of loss of control), and subjective bulimic episodes (subjective sense of loss of control while eating a quantity of food not regarded to be large given the context). The EDE also comprises four subscales (Restraint, Eating Concern, Shape Concern, and Weight Concern) and a Global Score; higher scores reflect greater severity or frequency.

The Questionnaire for Eating and Weight Patterns-Revised (QEWP-R) (Yanovski, 1993) was used to assess the age of first becoming overweight, age of binge eating onset, and age of dieting onset. Additional questions assessed a total number of attempted diets and supervised diets.

Psychological and physical functioning

The Beck Depression Inventory (BDI) (Beck & Steer, 1987; Beck & Steer, 1993) is a widely-used measure of the features of depression with established psychometric properties (Beck, Steer, & Garbin, 1988) that consists of 21 questions regarding levels and symptoms of depression in the past week. For each question, participants were asked to select one of four statements that best described their feeling (scored 0-3). A total score was calculated, and higher scores reflect higher levels of depression and negative affect (score range = 0-63). Scores 0-13 indicates minimal depression, 14-19 indicates mild depression, 20-28 indicates moderate depression, and 29-63 indicates severe depression.

The 36-Item Short Form Survey (SF-36) consists of 36 questions regarding health-related quality of life (Ware & Sherbourne, 1992), with well-established reliability and validity (McHorney, Ware, Lu, & Sherbourne, 1994; McHorney, Ware, & Raczek, 1993). Physical and mental component summaries were calculated. Scores less than 50 indicate poorer functioning than the average.

Metabolic measures

Participants' height was measured and weight was measured using a high-capacity digital scale. Waist circumference 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 A1c (HbA1c) were obtained through serum sample, and were analyzed by Quest Diagnostics (Madison, New Jersey). Individuals were categorized as having metabolic syndrome (MetS) if they met three or more of the five criteria outlined by the National Cholesterol Education Program's Adult Treatment Panel III guidelines (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001): a) Central or abdominal obesity (> 40 inches for men, > 35 inches for women); b) Triglycerides ≥ 150 mg/dL; c) Reduced HDL cholesterol (< 40 mg/dL for men, < 50 mg/dL for women); d) Systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg; and e) Fasting glucose ≥ 110 mg/dL.

Analysis

Caucasian and African American women were compared on eating-related characteristics, psychosocial functioning, and metabolic measures. Chi-square tests were used for categorical variables. Analysis of covariance (ANCOVA) was used for continuous variables with BMI as covariates, and partial eta-squared (partial η^2) was calculated to indicate effect size (small = 0.01, medium = 0.06, large = 0.14). All analyses were conducted with the Statistical Analysis System (SAS) (release 9.4, 2002-2010, SAS Institute, Cary, NC).

Results

Caucasian and African American women did not differ significantly in sociodemographic characteristics. Mean age was 46.8 ± 13.3 years for Caucasian women and 43.6 ± 10.7 years for African American women ($t[107] = 1.40, p > .05$). Among Caucasian women, 77.4% reported completing college education, 13.2% reported some college education, and 9.4% reported completing high school education; among African American women, 69.6%

reported completing college education, 21.4% reported some college education, and 9.0% reported completing high school education ($\chi^2[2] = 1.28, p > .05$).

Table 1 summarizes eating-related characteristics by race. There was no significant difference in BMI by race. Analyses revealed significant differences in the developmental histories of eating/weight concerns. Caucasian women reported significantly earlier onset of becoming overweight, beginning binge eating and dieting, and a greater number of times on a diet; the observed differences reflected medium-to-large effect sizes. Caucasian and African American women were not significantly different in their eating disorder psychopathology.

Table 2 summarizes psychosocial and physical functioning, biological measures, and metabolic measures. There were no significant differences in BDI score, the physiological health component of the SF-36, or the mental health component of the SF-36. Analyses revealed some significant differences in metabolic functioning by race. Diastolic blood pressure was significantly higher in African American women than Caucasian women. African American women also had significantly greater HbA1c than Caucasian women. On the other hand, triglycerides were higher in Caucasian women than African American women. The observed significant differences on the metabolic variables generally reflected medium effect sizes. There were no other significant differences in metabolic measures, including the proportion of those who met MetS criteria.

We also compared the proportion of women meeting clinical criteria for each metabolic component and the patterns of significant results were similar to dimensional measures. A greater proportion of Caucasian women showed clinically elevated triglycerides (25.5% [Caucasian] vs. 6.5% [African American]; $\chi^2[1] = 6.09, p < .05$), whereas a greater proportion of African American women showed clinically elevated blood pressure (36.2% [Caucasian] vs. 60.4% [African American]; $\chi^2[1] = 5.84, p < .05$). No significant differences were found by race for central or abnormal obesity (100.0% [Caucasian] vs. 94.1% [African American]; $\chi^2[1] = 2.91, p > .05$), reduced HDL cholesterol (39.5% [Caucasian] vs. 34.8% [African American]; $\chi^2[1] = 0.22, p > .05$), and fasting glucose level (19.1% [Caucasian] vs. 20.0% [African American]; $\chi^2[1] = 0.01, p > .05$).

Discussion

The present study compared history and features of BED, psychosocial functioning, and metabolic measures between Caucasian and African American women with comorbid BED and obesity seeking treatment in primary care. Compared with African American women, Caucasian women reported significantly younger age of first overweight, binge eating onset, dieting onset, as well as more frequent dieting. Earlier onsets of binge eating, overweight, and dieting among Caucasian patients observed here (generally reflecting medium-to-large effect sizes) is consistent with previous studies examining BED in a community sample (Striegel-Moore et al., 2005) and a large sample of treatment-seeking individuals who were recruited for a university-based program (Lydecker & Grilo, in press). Further research is needed to understand why Caucasian women exhibit various eating/weight-related problems earlier than African American women. Clinically, such findings might represent useful early

signs to assist clinicians in primary care settings in their screening to enhance earlier identification of BED.

The lack of significant racial differences in the cognitive/behavioral features of eating disorders is also similar to Lydecker and Grilo (Lydecker & Grilo, In press). Such findings might suggest that existing specialist treatments for BED may be appropriate across racial groups without much modification. Aggregated analysis of specialty-clinic based studies reported marked distress and suffering related with eating disorder and obesity among racial/ethnic minorities (Franko et al., 2012). Lydecker and Grilo (Lydecker & Grilo, in press) reported higher BMI, more frequent binge eating, and lower depression levels in African American women, compared with Caucasian women. The current study with primary care patients, however, observed racial similarities in levels of depressive symptoms, physical and mental health functioning. Both Caucasian and African American patients reported, on average, mild depressive symptoms and less than ideal physical and mental health functioning. Our findings on cognitive/behavioral features of eating disorders and psychosocial functioning thus suggest that physical and mental health of African American women may suffer equally from BED, and would benefit from referral to a special treatment for BED. The current study also found no significant differences in sociodemographic characteristics and BMI by race. We do note, however, that although we failed to observe a statistical significant difference in BMI by race, the observed BMIs for African American and Caucasian participants in our study (39.3 vs 37.2 kg/m², respectively; reflecting a small-to-medium effect size) are quite similar to those reported by Lydecker and Grilo (Lydecker & Grilo, in press) in their larger and better powered study (40.1 vs 37.9 kg/m², respectively). In addition, while the two clinical studies included both men and women, the current study only focused on women as the smaller number of men would have precluded meaningful analyses by race.

While the proportion of individuals with MetS was similar among African American and Caucasian women, we found a few significant differences in individual components of metabolic measures. Levels of triglycerides were significantly higher in Caucasian women than African American women, while blood pressure was significantly higher in African American women than Caucasian women. Compared with Caucasian women, African American women also had significantly higher Hb1Ac, indicating poorer control of blood glucose levels; although the observed difference reflected a medium effect-size, we note that the average Hb1Ac values for both Caucasian and African American patients satisfied the clinical criteria for poor glycemic control (HbA1c value ≥ 5.45 ; Sung & Rhee, 2007). These findings are consistent with racial differences reported in U.S. adolescent and adult population-based studies (Lin, Carnethon, Szklo, & Bertoni, 2011; Sentell, He, Gregg, & Schillinger, 2012; Sumner, 2009). We previously reported higher frequency of metabolic abnormalities in men with comorbid BED and obesity, compared with their female counterparts (Udo et al., 2013). Such findings suggest that clinicians in primary care settings should assess for metabolic disturbances and institute appropriate interventions in patients with comorbid BED and obesity. Some longitudinal research has reported that BED is associated with heightened risk for components of the metabolic syndrome (Hudson et al., 2010).

In specialty clinic-based treatment studies for eating disorders, racial minorities are under-represented and while we do not know the exact reasons for this disparity, it may perhaps be due partly to either actual or perceived lack of access to specialty services (Crow et al., 2004). Studying BED in primary care settings resulted in increased representation of African Americans in our sample and thus possibly increased generalizability of the study findings. The study also has some limitations. While our subjects were recruited in primary care settings, they were included because they showed interest in treatment and research for BED and obesity. Our findings may not be generalizable to individuals in the community who are not in treatment, to individuals who present for medical care but are not seeking treatment for eating/weight issues, or to individuals not interested in participating in research. While individuals with uncontrolled diabetes/hypertension were excluded, participants for whom these conditions (or MetS) were medically managed were enrolled. Thus, the frequency of metabolic abnormalities in our sample might reflect under-estimates of actual rates. Our study was not designed to identify or determine actual rates of untreated or uncontrolled metabolic problems; rather, we were interested in examining metabolic correlates in African American and Caucasian women who seek treatment for BED/obesity in primary care. Other selection biases are possible limitations; studies have, for example, reported poorer management of diabetes and hypertension in African American patients (Biello, Rawlings, Carroll-Scott, Browne, & Ickovics, 2010; Chou et al., 2007; Will & Yoon, 2013), and thus it is possible that a greater proportion of African American women were excluded from the study than Caucasian women. Similarly, we excluded individuals under antidepressant treatment. This might have also led to underestimation of mood and anxiety disorders, and depressive symptoms. We were also unable to also include other racial/ethnic groups or men due to their under representation in our sample.

In conclusion, among samples recruited at primary care settings for the treatment of comorbid BED and obesity, the present study found substantially earlier manifestations of eating/weight-related symptoms in Caucasian women than African American women, and significant differences in specific metabolic measures by race. We found no significant differences, however, in eating disorder features or psychosocial functioning. Our findings suggest that the implications of BED and obesity in African American individuals are as serious as in Caucasian individuals. Given that African Americans are less likely to present with BED and obesity at specialty-clinics (Crow et al., 2004), primary care practices should actively screen and assess for BED in primary care settings and provide access or referral to appropriate interventions for comorbid BED and obesity in addition to the metabolic correlates.

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References

American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th. Arlington, VA: American Psychiatric Publishing; 2013.

- Beck, AT.; Steer, R. Manual for revised Beck Depression Inventory. New York: Psychological Corporation; 1987.
- Beck, AT.; Steer, RA. Manual for the Beck Depression Inventory. San Antonio, TX: Psychological Corporation; 1993.
- Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review*. 1988; 8:77–100.
- Biello KB, Rawlings J, Carroll-Scott A, Browne R, Ickovics JR. Racial disparities in age at preventable hospitalization among U.S. Adults. *American Journal of Preventive Medicine*. 2010; 38(1):54–60. DOI: 10.1016/j.amepre.2009.08.027 [PubMed: 20117557]
- Chou AF, Brown AF, Jensen RE, Shih S, Pawlson G, Scholle SH. Gender and racial disparities in the management of diabetes mellitus among Medicare patients. *Womens Health Issues*. 2007; 17(3): 150–161. DOI: 10.1016/j.whi.2007.03.003 [PubMed: 17475506]
- Crow SJ, Peterson CB, Levine AS, Thuras P, Mitchell JE. A survey of binge eating and obesity treatment practices among primary care providers. *International Journal of Eating Disorders*. 2004; 35(3):348–353. DOI: 10.1002/eat.10266 [PubMed: 15048950]
- Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA*. 2001; 285(19):2486–2497. [PubMed: 11368702]
- Fairburn, CG.; Cooper, Z., editors. *The Eating Disorder Examination*. 12th. New York, NY: Guilford Press; 1993.
- First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. *Structured Clinical Interview for DSM-IV, Patient Edition*. Washington, D C.: American Psychiatric Press; 1995.
- Franko DL, Thompson-Brenner H, Thompson DR, Boisseau CL, Davis A, Forbush KT, et al. Wilson GT. Racial/ethnic differences in adults in randomized clinical trials of binge eating disorder. *Journal of Consulting and Clinical Psychology*. 2012; 80(2):186–195. DOI: 10.1037/a0026700 [PubMed: 22201327]
- Grilo CM, Lozano C, Masheb RM. Ethnicity and sampling bias in binge eating disorder: Black women who seek treatment have different characteristics than those who do not. *International Journal of Eating Disorders*. 2005; 38(3):257–262. DOI: 10.1002/eat.20183 [PubMed: 16211631]
- Grilo CM, White MA, Barnes RD, Masheb RM. Psychiatric disorder co-morbidity and correlates in an ethnically diverse sample of obese patients with binge eating disorder in primary care settings. *Comprehensive Psychiatry*. 2013; 54:209–216. DOI: 10.1016/j.comppsy.2012.07.012 [PubMed: 22943959]
- Hudson JI, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*. 2007; 61(3):348–358. DOI: 10.1016/j.biopsych.2006.03.040 [PubMed: 16815322]
- Hudson JI, Lalonde JK, Coit CE, Tsuang MT, McElroy SL, Crow SJ, et al. Pope HG Jr. Longitudinal study of the diagnosis of components of the metabolic syndrome in individuals with binge-eating disorder. *American Journal of Clinical Nutrition*. 2010; 91(6):1568–1573. DOI: 10.3945/ajcn.2010.29203 [PubMed: 20427731]
- Kessler RC, Berglund PA, Chiu WT, Deitz AC, Hudson JI, Shahly V, et al. Xavier M. The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. *Biological Psychiatry*. 2013; 73(9):904–914. DOI: 10.1016/j.biopsych.2012.11.020 [PubMed: 23290497]
- Lin SX, Carnethon M, Szklo M, Bertoni A. Racial/ethnic differences in the association of triglycerides with other metabolic syndrome components: the Multi-Ethnic Study of Atherosclerosis. *Metabolic Syndrome and Related Disorders*. 2011; 9(1):35–40. DOI: 10.1089/met.2010.0050 [PubMed: 20958206]
- Lydecker JA, Grilo CM. Different yet similar: examining race and ethnicity in treatment-seeking adults with binge eating disorder. *Journal of Consulting and Clinical Psychology*. in press.
- Marques L, Alegria M, Becker AE, Chen CN, Fang A, Chosak A, Diniz JB. Comparative prevalence, correlates of impairment, and service utilization for eating disorders across US ethnic groups:

- Implications for reducing ethnic disparities in health care access for eating disorders. *International Journal of Eating Disorders*. 2011; 44(5):412–420. DOI: 10.1002/eat.20787 [PubMed: 20665700]
- McHorney CA, Ware JE Jr, Lu JF, Sherbourne CD. The MOS 36-item Short-Form Health Survey (SF-36): III. Tests of data quality, scaling assumptions, and reliability across diverse patient groups. *Medical Care*. 1994; 32:40–66. [PubMed: 8277801]
- McHorney CA, Ware JE Jr, Raczek AE. The MOS 36-Item Short-Form Health Survey (SF-36): II. Psychometric and clinical tests of validity in measuring physical and mental health constructs. *Medical Care*. 1993; 31:247–263. [PubMed: 8450681]
- Pike KM, Dohm FA, Striegel-Moore RH, Wilfley DE, Fairburn CG. A comparison of black and white women with binge eating disorder. *American Journal of Psychiatry*. 2001; 158(9):1455–1460. [PubMed: 11532731]
- Sentell TL, He G, Gregg EW, Schillinger D. Racial/ethnic variation in prevalence estimates for United States prediabetes under alternative 2010 American Diabetes Association criteria: 1988–2008. *Ethnicity & Disease*. 2012; 22(4):451–458. [PubMed: 23140076]
- Striegel-Moore RH, Fairburn CG, Wilfley DE, Pike KM, Dohm FA, Kraemer HC. Toward an understanding of risk factors for binge-eating disorder in black and white women: A community-based case-control study. *Psychological Medicine*. 2005; 35(6):907–917. [PubMed: 15997611]
- Sumner AE. Ethnic differences in triglyceride levels and high-density lipoprotein lead to underdiagnosis of the metabolic syndrome in black children and adults. *Journal of Pediatrics*. 2009; 155(3):S7.e7–11. DOI: 10.1016/j.jpeds.2009.04.049
- Sung KC, Rhee EJ. Glycated haemoglobin as a predictor for metabolic syndrome in non-diabetic Korean adults. *Diabetic Medicine*. 2007; 24:848–854. [PubMed: 17650158]
- Udo T, McKee SA, White MA, Masheb RM, Barnes RD, Grilo CM. Sex differences in biopsychosocial correlates of binge eating disorder: a study of treatment-seeking obese adults in primary care setting. *General Hospital Psychiatry*. 2013; 35(6):587–591. DOI: 10.1016/j.genhosppsych.2013.07.010 [PubMed: 23969142]
- Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Medical Care*. 1992; 30(6):473–483. [PubMed: 1593914]
- Will JC, Yoon PW. Preventable hospitalizations for hypertension: establishing a baseline for monitoring racial differences in rates. *Preventing Chronic Disease*. 2013; 10:120165.doi: 10.5888/pcd10.120165 [PubMed: 23411035]
- Yanovski SZ. Binge eating disorder: current knowledge and future directions. *Obesity Research*. 1993; 1(4):306–324. [PubMed: 16350580]

Table 1**Eating-Related Characteristics by Race.**

	Caucasian (<i>n</i> = 53)	African American (<i>n</i> = 56)	<i>F</i> -test	Effect size (partial η^2)
BMI	37.2 (4.6)	39.3 (6.0)	$F(1, 99) = 3.65, ns$	0.04
Age of first time overweight	15.7 (9.3)	22.7 (11.0)	$F(1, 93) = 11.44, p < 0.01$	0.11
Age of binge eating onset	22.7 (12.2)	30.0 (13.3)	$F(1, 94) = 7.94, p < 0.01$	0.08
Age of dieting onset	16.9 (7.2)	26.8 (11.2)	$F(1, 88) = 24.82, p < 0.01$	0.22
Number of times on a diet ^a	43.0 (64.6)	21.1 (59.7)	$F(1, 89) = 8.28, p < 0.01$	0.09
Eating Disorder Psychopathology (EDE interview)				
Global Score	2.43 (0.10)	2.32 (0.12)	$F(1, 101) = 0.65, ns$	0.01
Restraint	1.88 (1.26)	1.69 (1.24)	$F(1, 101) = 0.62, ns$	0.01
Eating concern	1.87 (1.18)	1.72 (1.24)	$F(1, 101) = 0.40, ns$	0.00
Shape concern	3.47 (1.19)	3.39 (1.34)	$F(1, 98) = 0.09, ns$	0.00
Weight concern	3.26 (1.02)	3.15 (0.98)	$F(1, 98) = 0.31, ns$	0.00
Objective bulimic episodes <i>I, a</i>	14.80 (10.40)	17.41 (13.68)	$F(1, 101) = 0.83, ns$	0.01
Subjective bulimic episodes <i>I</i>	11.42 (13.10)	9.93 (13.80)	$F(1, 100) = 0.31, ns$	0.00
Objective overeating episodes <i>I, a</i>	4.98 (11.29)	4.37 (9.09)	$F(1, 101) = 0.01, ns$	0.00

Notes. BMI = body mass index. EDE = Eating Disorder Examination.

I = frequency over the past 28 days.

^a = statistics are based on log-transformed values, but presented means and standard deviations are raw values.

Partial η^2 : small = 0.01; medium = 0.06; large = 0.14.

Table 2

Psychosocial Functioning, Physical Functioning, and Biological and Metabolic Measures by Race.

	Caucasian (<i>n</i> = 53)	African American (<i>n</i> = 56)	<i>F</i> -test/ χ^2 -test	Effect size (partial η^2)
Depression (BDI score)	14.4 (8.7)	15.0 (9.4)	<i>F</i> (1, 105) = 0.09, <i>ns</i>	0.00
Health Functioning (SF-36)				
Physical health component (t-score)	44.1 (10.5)	43.6 (10.8)	<i>F</i> (1, 103) = 0.05, <i>ns</i>	0.00
Mental health component (t-score)	43.1 (12.0)	43.9 (10.1)	<i>F</i> (1, 103) = 0.12, <i>ns</i>	0.00
Waist Circumference	44.6 (4.2)	45.8 (6.5)	<i>F</i> (1, 97) = 1.15, <i>ns</i>	0.01
Systolic BP	125.3 (16.2)	129.9 (15.9)	<i>F</i> (1, 98) = 2.02, <i>ns</i>	0.02
Diastolic BP	76.4 (8.1)	83.5 (10.9)	<i>F</i> (1, 98) = 13.30, <i>p</i> < .01	0.12
Fasting glucose level ^a	100.0 (15.6)	100.2 (33.0)	<i>F</i> (1, 85) = 0.30, <i>ns</i>	0.00
HbA1c ^a	5.7 (0.6)	6.0 (0.7)	<i>F</i> (1, 86) = 5.64, <i>p</i> < .05	0.06
Total cholesterol	195.2 (35.6)	188.2 (35.2)	<i>F</i> (1, 87) = 0.87, <i>ns</i>	0.01
HDL	56.5 (17.5)	56.3 (14.3)	<i>F</i> (1, 87) = 0.00, <i>ns</i>	0.00
LDL	112.0 (30.8)	111.7 (30.2)	<i>F</i> (1, 87) = 0.00, <i>ns</i>	0.00
Triglycerides ^a	133.4 (70.6)	100.6 (50.6)	<i>F</i> (1, 87) = 8.23, <i>p</i> < .01	0.09
% meeting criteria for metabolic syndrome (MetS) ¹	33.3	35.6	χ^2 (1) = 0.05, <i>ns</i>	----

Notes. BDI = Beck Depression Inventory; SF-36 = 36-Item Short Form Survey. BP = blood pressure; HbA1c = glycated hemoglobin 1Ac.

¹ = Criteria for MetS was based on the National Cholesterol Education Program's Adult Treatment Panel III guidelines (20).

^a = statistics are based on log-transformed values, but presented means and standard deviations are raw values.

Partial η^2 : small = 0.01; medium = 0.06; large = 0.14.