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The Impact of Race on Metabolic Disease Risk Factors in Women with and without Posttraumatic Stress Disorder

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Abstract

The literature on PTSD and metabolic disease risk factors has been limited by lacking investigation of the potential influence of commonly comorbid disorders and lacking consideration of the role of race. Data were provided by a sample of 134 women (63 PTSD and 71 without PTSD). Separate sets of models examined associations of psychiatric disorder classifications with metabolic disease risk factors. Each model included race (African American or Caucasian), psychiatric disorder, and their interaction. There was an interaction of race and PTSD on body mass index, abdominal obesity, and triglycerides. While PTSD was not generally associated with deleterious health effects in African American participants, PTSD was related to worse metabolic disease risk factors in Caucasians. MDD was associated with metabolic disease risk factors, but there were no interactions with race. Results support the importance of race in the relationship between PTSD and metabolic disease risk factors. Future research would benefit from analysis of cultural factors to explain how race might influence metabolic disease risk factors in PTSD.

Keywords

Posttraumatic stress disorder; metabolic disease; race/ethnicity; obesity; major depressive disorder

Introduction

An increasingly large body of literature and epidemiologic evidence suggest a close relationship between posttraumatic stress disorder (PTSD) and physical health (Schnurr & Green, 2004). Research has shown that individuals with PTSD report more health complaints, suffer from more physician-diagnosed medical conditions (Beckham, et al., 1998; Calhoun, Wiley, Dennis, & Beckham, 2009), and exhibit higher rates of health care utilization (Boscarino, 1997; Dennis, et al., 2009). PTSD has been linked with several health outcomes related to metabolic abnormalities including increased rates of metabolic syndrome (Babic, et al., 2007; Jakovljevic, et al., 2007; Jakovljevic, Saric, Nad, Topic, &

Vuksan-Cusa, 2006; Violanti, et al., 2006), and higher rates of obesity (David, Woodward, Esquenazi, & Mellman, 2004; Dobie, et al., 2004; Perkonig, Owasbi, Stein, Kirschbaum, & Wittchen, 2009; Trief, Ouimette, Wade, Shanahan, & Weinstock, 2006; Vieweg, et al., 2007).

Although the literature on PTSD and metabolic disease risk factors has provided a number of notable findings, this literature is complicated by the frequent comorbidity of major depressive disorder (MDD) and substance use disorders (SUD) (Campbell, et al., 2007). In the National Comorbidity Survey, 49% of women with PTSD also reported a history of a major depressive episode (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). The association of MDD with poor physical health, including metabolic disease risk factors, has been observed in several decades of research (Eaton, 1996; Katon & Ciechanowski, 2002). In addition to MDD, PTSD is often accompanied by comorbid SUD. In the National Comorbidity Survey, 28% of women with PTSD also reported a history of comorbid alcohol abuse or dependence, and 27% reported a history of drug abuse or dependence (Kessler, et al., 1995). Several studies have noted an association between SUD and metabolic disease (Virmani, Binienda, Ali, & Gaetani, 2006). SUD has been linked with poor glycemic control (Ng, Darko, & Hillson, 2004) and more severe diabetes (Glasgow, et al., 1991).

Studies estimate the lifetime prevalence of PTSD in the United States is between 6% and 8% (Kessler, et al., 2005), and is twice as high in women as in men (Breslau, 2001), with estimates ranging from 10% to 13% (Calhoun, et al., 2009). Further, women are twice as likely to develop PTSD following trauma exposure and are more likely to develop chronic symptoms of PTSD (Calhoun, et al., 2009). Unfortunately, much of the research on PTSD and physical health has occurred with samples that are primarily or exclusively male. While much of the research on PTSD and physical health has been conducted in men, some research supports the generalizability of this association to female samples. Studies have shown that women with PTSD exhibit a higher resting heart rate compared to those without PTSD (Beckham, Flood, Dennis, & Calhoun, 2009; Vrana, Hughes, Dennis, Calhoun, & Beckham, 2009), and exaggerated heart rate startle response (Carson, et al., 2007). Reports have also shown that negative affect (symptoms of depression and anxiety) has been linked to hypertension (Davidson, Jonas, Dixon, & Markovitz, 2000; Raikkonen, Matthews, & Kuller, 2001; Raikkonen, Matthews, Kuller, Reiber, & Bunker, 1999), predict the risk for development of metabolic syndrome in women (Raikkonen, Matthews, & Kuller, 2002), and increases the risk of mortality from cardiovascular disease (Wassertheil-Smoller, et al., 2004). The Third National Health and Nutrition Examination Survey, a study that included 3186 men and 3003 women, found that the prevalence of metabolic syndrome was elevated in women with a history of depression, while men with a history of depression were not significantly more likely to have metabolic syndrome (Kinder, Carnethon, Palaniappan, King, & Fortmann, 2004).

The existing literature suggests a significant connection between metabolic disease risk factors and PTSD (Dedert, Calhoun, Watkins, Sherwood, & Beckham, 2010), but mechanisms underlying the relationship of PTSD to metabolic disease risk factors are not well understood. Although few data are available on the influence of race on metabolic disease risk factors in PTSD, there is reason to expect that sociodemographic factors play a role in metabolic disease risk factors. Recent data from the National Health and Nutrition Examination Survey revealed a sex difference in the rates of metabolic disease risk factors by race. While data from men indicated that metabolic syndrome was more prevalent in Caucasians, data from women indicated that metabolic syndrome was more prevalent in African Americans (38.5%), compared to Caucasians (31.5%) (Ervin, 2009). Looking deeper into the pattern of results, several studies have found that African Americans tend to have elevations in several measures of metabolic disease risk, including weight elevations,

abdominal obesity, hyperglycemia, and high blood pressure, but that Caucasians exhibit dyslipidemia (e.g. high triglycerides) (Ervin, 2009; Kurella, Lo, & Chertow, 2005; Zeno, et al., 2010). Differences in metabolic disease risk factors across race have lead some to suggest establishing different metabolic disease criteria depending on race (Zeno, et al., 2010).

A study using data from the National Epidemiologic Survey on Alcohol and Related Conditions found that the lifetime prevalence of PTSD in African Americans was 8.7%, in a sample of 34,653 adults (Roberts, Gilman, Breslau, Breslau, & Koenen, 2011). This was significantly higher than what was found both in Caucasians (7.4%) and the general population (7.3%). Higher rates of PTSD in racial minorities could be due in part to differences in trauma severity and access to care. Available research on responses to trauma have found that, while Caucasians report higher rates of trauma, African Americans tend to experience more severe traumatic events for which they are less likely to receive treatment (Kessler, et al., 1995; Perilla, Norris, & Lavizzo, 2002; Roberts, et al., 2011).

Research on the relationship between PTSD and metabolic disease risk factors has suffered from complexity in interpretation due to the high comorbidity of PTSD with MDD and SUD. In addition, while it is clear that sex and race play a role in metabolic disease risk factors, these sociodemographic variables have received little consideration in the relationship between PTSD and metabolic disease risk factors. Thus, the current research has three aims: 1) investigate the associations of PTSD with metabolic disease risk factors among a sample of Caucasian and African American women; 2) explore the role of race in the PTSD and metabolic disease risk factors relationship; and 3) evaluate the associations of psychiatric disorders commonly comorbid with PTSD (current major depressive disorder and history of substance use disorder) and total number of axis I psychiatric disorders with metabolic disease risk factors. Because African Americans have a higher prevalence than Caucasians in obesity, hypertension, and diabetes (Flegal, Carroll, Ogden, & Curtin, 2010; Sumner, 2009), we hypothesized that, relative to Caucasian women, African American women would demonstrate a stronger association between PTSD and the likelihood of metabolic disease risk factors. In addition, since comorbid psychiatric disorders (i.e., MDD, SUD, and number of psychiatric disorders) are also associated with significant medical illness, we hypothesized that measures of physical illness would also be elevated in groups with non-PTSD psychiatric disorders.

Methods

Participants and Procedure

This study employed a secondary analysis of data from an investigation of psychiatric disorders and physical health outcomes in women. Previous publications from this data set have reported on relationships between hostility and ambulatory cardiovascular monitoring (Beckham, et al., 2009)) and intervening effects of psychiatric symptoms in the relationship between childhood trauma and weight outcomes (E. A. Dedert, et al., 2010). Participants were recruited *via* advertising for a study on trauma and health posted at two local hospitals and a more general flyer to recruit participants for a non-PTSD comparison group. From the 193 participants screened for the study, 11 participants were excluded because they met diagnostic criteria for current alcohol or other substance dependence/abuse or psychotic disorders, including schizophrenia and bipolar with active manic symptoms. Two participants were excluded during the screening process due to medication usage (amitriptyline and methadone). Participants taking second generation antipsychotics were not excluded, but only two participants were taking second generation antipsychotic medications during the study. Although both of these participants were diagnosed with

PTSD, one was African American and the other was Caucasian. Due to the low frequency of these medications in our dataset, they likely do not account for the observed results.

Further, 32 participants were recruited for the comparison group but excluded because they met criteria for lifetime (but not current) PTSD or lifetime MDD. Due to an insufficient number of participants in other racial groups to conduct comparisons, the sample was restricted to Caucasians ($n = 66$) and African Americans ($n = 68$) for a total sample of 134 women used for analysis. Of the 14 participants who were excluded from analysis five self-identified as having more than one race, four as Asian Americans, three as Hispanic Americans, one as Native American, and one as Pacific Islander.

The study was approved by both the University and VA institutional review boards. Participants were recruited from among outpatients at the Duke University Primary Health Care and PTSD Clinics, and the Durham Veterans Affairs Medical Center, and also from the community. Participants provided self-report data on racial/ethnicity status, income, years of education, age, and job status. Participants were excluded if there was a current or previous diagnosis of organic mental disorder, schizophrenia, or paranoid disorders; depression severe enough to require immediate psychiatric treatment (i.e. inpatient psychiatric hospitalization), bipolar depression, or depression accompanied by delusions, hallucinations or bizarre behavior; current SUD (including both alcohol and/or drugs); or illiteracy in English.

We administered the Clinician Administered PTSD Scale to determine PTSD diagnostic status (Blake, et al., 1995), and the Structured Clinical Interview for DSM-IV (First, Spitzer, Gibbon, & Williams, 1997) was used to diagnose other possible Axis I disorders. Current diagnoses were determined using a one-month time frame with the exception of substance abuse in which a three-month time frame was used to define current SUD. Given the high comorbidity of SUD in general and the possible effects on the variables of interest, it was decided that longer period of lack of substance abuse (i.e., greater than the usual one month) was needed. Both of these structured interviews have demonstrated excellent reliability and validity in clinical settings (Weathers, Keane, & Davidson, 2001; Weathers, Ruscio, & Keane, 1999; Zanarini, et al., 2000). Eight diagnostic raters were utilized, and interrater reliability for diagnoses based on videotapes of patient interviews was high (Fleiss' $K = .94$). Participants also listed their current medications and provided demographic information, including socioeconomic status based on the Hollingshead index (Hollingshead & Redlich, 1958). Study participants were compensated a total of \$150 for two sessions of questionnaires and diagnostic assessment.

Physical Health Measures

Body Mass Index and Abdominal Obesity—Participants provided self-reported height, and study personnel measured weight for calculation of body mass index (BMI), as well as measuring waist circumference (in centimeters) to assess abdominal obesity. Waist circumference was measured once for each participant, so no reliability data were calculated for these measurements. Participants with a BMI score of 30 or more were classified as obese.

Lipid Panel Profiles—Prior to blood draws for lipid assessment, all participants reported fasting for 12 hours. The laboratory technician drew blood into tubes without stasis for the lipid determinations. Serum samples were separated by centrifugation and then analyzed. Lipid assessment consisted of determination of high density lipoprotein (HDL), low density lipoprotein (LDL), and plasma triglycerides. Triglycerides were determined enzymatically. HDL assays used a precipitation procedure (Warnick, Benderson, & Albers, 1983). LDL

was arithmetically calculated from HDL, total cholesterol, and triglycerides using the Friedewald equation (Friedewald, Levy, & Fredrickson, 1972).

Blood Pressure—Blood pressure was measured with ambulatory blood pressure monitors. Participants were fitted with an ambulatory recorder: the Accutracker II (Suntech Medical Instruments, Raleigh, NC), or the Spacelabs 90207. Participants were instructed to carry on with their usual daily activities, to keep their nondominant arm (where the cuff was attached) at their side whenever the recorder operated, and to wear the monitor for 24 hours. The recorder was programmed to operate every 30 minutes $\pm$ 5 minutes during waking hours based on self-reported sleep/wake times. On each measurement occasion, the device obtained single readings systolic and diastolic blood pressure. Both monitors were programmed to retake any blood pressure readings that appeared out of range. Criteria for detection and elimination of final values in the dataset included the following: systolic blood pressure less than 50 mmHg or greater than 245 mmHg; diastolic blood pressure less than 40 mmHg or greater than 140 mmHg; and systolic/diastolic ratio of greater than three. Whenever the systolic or diastolic blood pressure was removed, the corresponding measurement was also removed. Participants rated their level of physical activity each time the recorder operated while they were awake. Analyses for this report used only ratings from inactive periods (e.g., Sitting, Reclining, Sleeping). Each measurement occasion was rated as asleep or awake based on report of sleep and wake times completed during the 24-hour monitoring period.

Statistical Analysis

We tested for demographic differences by current PTSD, current MDD, lifetime SUD, and racial group (Caucasian vs. African Americans) using general linear models for continuous variables and chi-square tests for categorical variables. To evaluate associations of PTSD with metabolic symptoms, we first split the sample into the following two groups: 1) all participants with PTSD, and 2) all participants without PTSD. Because analysis of a PTSD group with all common comorbidities removed would not be representative of PTSD, we included all participants with PTSD in analyses of the association of PTSD with health variables. However, comorbidity is a critical issue to consider in the association of PTSD with medical problems, so we then evaluated comorbidity by conducting separate analyses comparing presence vs. absence of the following: current PTSD, current MDD, and lifetime SUD. Participants who met criteria for more than one of these disorders were included in more than one analysis, in the interest of accurately representing the psychiatric groups being evaluated. These analyses were conducted using general linear models. A final analysis used regression to evaluate the association of the number of axis I disorders with our dependent measures.

The sample had a mean age of 40.4 ($SD = 12.9$) and generally resided in the lower middle social class (Hollingshead $M = 42.1$). As age did not differ between groups, age was not included as a covariate in the models. To examine the relationships of PTSD and race with disease risk factors, we used general linear models to evaluate associations of PTSD and race with triglycerides, BMI, waist-to-hip ratio, HDL, and LDL. We then entered the interaction between PTSD and race. We then conducted similar analyses to evaluate the associations of MDD and race in disease risk factors.

Results

Demographic Differences Across Psychiatric Disorders

Table 1 presents summary statistics for relevant demographic, psychiatric, and medical variables for the PTSD group, the non-PTSD group, and the sample as a whole. Evaluation

of age, SES (from Hollingshead categories), and race differences between psychiatric groups revealed no differences between PTSD groups. Participants with current MDD had a higher mean age ($F(1,132) = 5.13, p = .025$, semipartial $\eta^2 = .037$) and lower SES ($X^2_1 = 24.02, p < .001$), but no between-group difference in race. Participants with a lifetime SUD had a lower SES ($X^2_1 = 14.50, p = .006$), but no significant differences in age or race. A greater number of Axis I disorders was generally associated with lower SES ($F(4,129) = 4.57, p = .002$, semipartial $\eta^2 = .124$), but there was no significant relationship of Axis I disorders with age or race. However, models were not adjusted for SES differences due to problems associated with using covariates to correct for differences in psychiatric groups (Miller & Chapman, 2001).

PTSD and Metabolic Disease Risk Factors

Examination of associations between PTSD and metabolic disease risk factors revealed that individuals with PTSD in our sample had higher BMI ($F(1,132) = 4.39, p = .038$, semipartial $\eta^2 = .032$) and rate of obesity ($F(1,132) = 9.12, p = .003$, semipartial $\eta^2 = .065$), larger abdominal obesity ($F(1,131) = 9.28, p = .003$, semipartial $\eta^2 = .066$), and higher systolic blood pressure ($F(1,105) = 4.51, p = .036$, semipartial $\eta^2 = .041$).

Race and Metabolic Disease Risk Factors

Table 2 presents summary statistics for demographic, psychiatric, and medical variables by race. Examination of associations between race and metabolic disease risk factors revealed that African American participants had lower SES ($F(1,132) = 10.66, p = .001$, semipartial $\eta^2 = .075$), larger abdominal obesity ($F(1,131) = 5.81, p = .017$, semipartial $\eta^2 = .043$), and lower triglycerides ($F(1,129) = 10.99, p = .001$, semipartial $\eta^2 = .079$).

Influences of Race and Comorbidities in PTSD and Metabolic Disease Risk Factors

Results from general linear models examining associations of race and several psychiatric disorders, as well as the interactions of race with these disorders, with dependent variables are summarized in Table 3. As illustrated in Figure 1, there was a race by PTSD interaction in BMI, such that Caucasians without PTSD had a lower BMI than Caucasians with PTSD ($t(1) = 3.27, p = .001, d = 4.70$). Caucasians without PTSD also had a lower BMI than racial minorities regardless of PTSD status ($t(1) = 2.76, p = .007, d = 3.97$ for PTSD; $t(1) = 3.05, p = .003, d = 4.38$ for non-PTSD). Similarly, there was an interaction of PTSD and race on obesity, such that Caucasians without PTSD had lower rates of obesity than each other group ($t(1) = 3.82, p < .001, d = 5.49$ compared to PTSD Caucasians; $t(1) = 3.67, p < .001, d = 5.27$ compared to PTSD African Americans; $t(1) = 3.17, p = .002, d = 4.54$ compared to non-PTSD African Americans). There was an interaction of PTSD and race on abdominal obesity, such that Caucasians without PTSD had lower abdominal obesity than each other group ($t(1) = 3.82, p < .001, d = 5.50$ compared to PTSD Caucasians; $t(1) = 3.93, p < .001, d = 5.64$ compared to PTSD African Americans; $t(1) = 3.38, p = .001, d = 4.85$ compared to non-PTSD African Americans). As illustrated in Figure 2, there was an interaction for triglycerides, such that Caucasians with PTSD had higher triglycerides than each other group ($t(1) = 4.00, p < .001, d = 5.76$ for non-PTSD Caucasians; $t(1) = 4.98, p < .001, d = 7.16$ for PTSD African Americans; $t(1) = 4.21, p < .001, d = 6.08$ for non-PTSD African Americans).

Models testing the association of MDD and race with disease risk factors revealed that MDD was associated with elevations in BMI, obesity rate, waist-hip ratio, LDL level, and triglyceride level. In contrast to models with PTSD, the models with MDD resulted in no interactions of MDD with race. A history of SUD was associated with higher systolic blood pressure, but no other health risk measures. There were no interactions of SUD history with race in models of health risk measures. The number of total axis I disorders was associated

with an increased rate of obesity ($F(1,130) = 11.02, p = .001$; semipartial $\eta^2 = .074$), larger waist-hip ratio ($F(1,129) = 11.11, p = .001$; semipartial $\eta^2 = .075$), higher LDL levels ($F(1,127) = 11.03, p = .001$; semipartial $\eta^2 = .077$), and higher triglyceride levels ($F(1,127) = 14.79, p < .001$; semipartial $\eta^2 = .095$).

Discussion

This investigation found that race played significant role in the association of PTSD with metabolic disease. Of the metabolic disease measures that were associated with PTSD, only systolic blood pressure did not also have a significant interaction between PTSD and race. Further, the pattern of interactions was relatively consistent. In our dataset, PTSD did not typically have an impact on metabolic disease risk factors in African Americans, but PTSD was associated with a notable worsening of metabolic disease risk factors in Caucasians. This pattern was noted in BMI, obesity rates, abdominal obesity, and triglycerides.

Across race, results generally indicated that race was not consistently related to metabolic disease risk factors, as African American women had higher rates of obesity, but Caucasian women had higher triglycerides. These findings are consistent with recent epidemiologic data on women (Ervin, 2009).

Because we found no significant increase in rates of obesity, triglyceride levels, waist to hip ratio, or systolic blood pressure in racial minorities with PTSD, it raises the question of whether fasting glucose levels are a key component of increased risk of metabolic syndrome in racial minorities with PTSD, particularly African American women. Our findings are consistent with previous data that suggest that a lower prevalence of metabolic syndrome in racial minorities may be due to lower frequency of problems with triglycerides and HDL levels when compared to Caucasians (Gaillard, Schuster, & Osei, 2009; Osei, 2010) and suggest a stronger relationship between insulin resistance and metabolic risk.

Another important future research question involves the possibility of a ceiling effect for weight problems in racial minorities that prevents further worsening through the presence of PTSD. In contrast, since Caucasian women have a lower rate of obesity, more variance in weight problems is available to be influenced by PTSD.

Further research is needed to determine which mechanisms might lead to especially deleterious effects of PTSD on metabolic disease risk factors in Caucasians. Metabolic abnormalities in PTSD could be influenced by different reactions to atypical anti-psychotic medications. These medications are associated with glucose dysregulation (Jin, Meyer, & Jeste, 2004; Wirshing, et al., 2002) and are often prescribed off-label for treatment of PTSD and might interact with race. Potential psychophysiological mechanisms include differential effects by race on hypothalamic-pituitary-adrenal axis function, inflammation, immune function, and behavioral health. For example, one study that observed racial differences in metabolic syndrome found that these differences were eliminated when metabolic syndrome criteria included physical fitness, c-reactive protein, percent body fat, and insulin resistance (Zeno, et al., 2010). Research on race differences in physical illness will likely benefit from progress in genetic research. It will be important to consider the influence of race in genetic studies with participants with PTSD. Finally, future research on the influence of race in physical illness would benefit from incorporating cultural variables that provide the potential to explain the process underlying effects of race on physical illness. Cultural elements such as social norms, values, beliefs, and interpersonal styles will need to be examined to provide depth to findings on race effects (Betancourt & Lopez, 1993; Perilla, et al., 2002). This depth will likely provide insights into intervention targets and strategies.

Limitations

Because this is a secondary analysis of an existing data set, data on interesting risk factors such as fasting glucose data and microalbuminuria were not available. As mentioned above, fasting glucose data can be used as criteria for classification of metabolic syndrome (Grundy, Brewer, Cleeman, Smith, & Lenfant, 2004). This is a particularly interesting question for future research in light of previous data indicating that African American women have elevated fasting glucose levels, and higher rates of and mortality from diabetes (Goodwin & Davidson, 2005; Trief, et al., 2006; Weisberg, et al., 2002). However, due to the evolving definition of metabolic syndrome and modified versions used across studies (Zeno, et al., 2010), we believe the current report adds to the literature on metabolic disease risk factors and presents novel data on the role of race in the relationship between PTSD and metabolic disease risk factors. Because our sample was fairly small, these results should be interpreted cautiously. In particular, we explored associations of PTSD with metabolic disease risk factors broadly, with no correction for multiple comparisons. Because this study represents preliminary research into race effects that have not been previously characterized, this exploratory approach is a useful method for research advances. However, it will be important to replicate these results and examine larger, nationally representative data sets that include fasting glucose data before firm conclusions can be reached regarding the role of race in the relationship between PTSD and metabolic disease.

Conclusions

Findings provide additional support for an association of PTSD with metabolic disease that is independent of commonly comorbid disorders that could also influence metabolic disease. In addition, the role of race in the PTSD and health relationship is understudied, especially in light of the findings of the current report. To understand the influence of PTSD on metabolic disease, it is important to analyze and consider the roles of race and culture.

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Figure 1.
Race by PTSD Interactions for Body Mass Index.

Figure 2.
Race by PTSD Interactions for Triglycerides in milligrams per deciliter.

Figure 3.
Race by PTSD Interactions for Obesity Proportions.

Figure 4.
Race by PTSD Interactions for Abdominal Obesity in centimeters.

Table 1

Participant Characteristics and Comparisons by PTSD Status.

	PTSD (<i>n</i> = 63)	Non-PTSD (<i>n</i> = 71)	Total sample (<i>n</i> = 134)	PTSD Effect Size
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	
Age	41.3 (11.6)	39.6 (14.0)	40.4 (12.9)	$\eta^2 = .004$
SES (Hollingshead)	3.7 (1.2)	3.2 (1.2)	42.1 (17.9)	$\eta^2 = .046^*$
# of Axis I Disorders	2.3 (1.1)	0.7 (1.0)	1.5 (1.3)	$\eta^2 = .390^{***}$
Body mass index (BMI)	33.2 (10.1)	29.9 (7.8)	31.4 (9.1)	$\eta^2 = .032^*$
Abdominal Obesity (cm)	97.0 (17.3)	88.0 (16.9)	92.2 (17.7)	$\eta^2 = .066^{**}$
High Density Lipoproteins (HDL)	54.6 (52.1)	50.5 (12.5)	52.4 (36.4)	$\eta^2 = .003$
Low Density Lipoproteins (LDL)	128.0 (37.7)	116.2 (35.4)	121.6 (36.8)	$\eta^2 = .026$
Triglycerides	108.6 (68.4)	89.0 (48.7)	97.9 (59.1)	$\eta^2 = .028$
Systolic Blood Pressure	123.0 (13.7)	117.4 (13.5)	120.1 (13.8)	$\eta^2 = .041^*$
Diastolic Blood Pressure	73.4 (10.3)	71.2 (9.4)	72.2 (9.9)	$\eta^2 = .012$
	Frequency (%)	Frequency (%)	Frequency (%)	
Major Depressive Disorder	35 (56%)	23 (32%)	58 (43%)	$\phi = .233^{**}$
Lifetime Substance Use Disorder	26 (41%)	12 (17%)	38 (28%)	$\phi = .270^{**}$
Obesity (BMI > 30)	40 (63%)	27 (38%)	67 (50%)	$\phi = .254^{**}$

*
p < .05.**
p < .01.***
p < .001. η^2 = semi-partial η^2 , measure of effect size for general linear models

PTSD = posttraumatic stress disorder. SES = Socioeconomic status. A higher Hollingshead score indicates lower SES. Due to missing data on one or more of the four components that made up the determination of metabolic syndrome, a total of 12 participants had missing data for the metabolic syndrome variable.

Table 2

Participant Characteristics and Comparisons by Race.

	African American (<i>n</i> = 68)	Caucasian (<i>n</i> = 66)	Total sample (<i>n</i> = 134)	Race Effect Size
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	
Age	40.6 (12.9)	40.1 (13.1)	40.4 (12.9)	$\eta^2 < .001$
SES (Hollingshead)	3.8 (1.0)	3.1 (1.3)	42.1 (17.9)	$\eta^2 = .075^{**}$
# of Axis I Disorders	1.5 (1.2)	1.5 (1.4)	1.5 (1.3)	$\eta^2 < .001$
Body mass index (BMI)	32.8 (7.4)	30.0 (10.4)	31.4 (9.1)	$\eta^2 = .024$
Abdominal Obesity (cm)	95.7 (15.8)	88.5 (18.9)	92.2 (17.7)	$\eta^2 = .043^*$
High Density Lipoproteins (HDL)	55.3 (49.5)	49.3 (11.7)	52.4 (36.4)	$\eta^2 = .007$
Low Density Lipoproteins (LDL)	116.3 (36.0)	127.2 (37.0)	121.6 (36.8)	$\eta^2 = .022$
Triglycerides	81.8 (47.1)	114.8 (65.7)	97.9 (59.1)	$\eta^2 = .079^{**}$
Systolic Blood Pressure	121.7 (15.0)	118.6 (12.6)	120.1 (13.8)	$\eta^2 = .012$
Diastolic Blood Pressure	73.5 (10.6)	71.1 (9.1)	72.2 (9.9)	$\eta^2 = .014$
	Frequency (%)	Frequency (%)	Frequency (%)	
Major Depressive Disorder	30 (44%)	28 (42%)	58 (43%)	$\phi = .017$
Lifetime Substance Use Disorder	26 (41%)	12 (17%)	38 (28%)	$\phi = -.142$
Obesity (BMI > 30)	40 (63%)	27 (38%)	67 (50%)	$\phi = .179^*$

*
p < .05.**
p < .01.***
p < .001. η^2 = semi-partial η^2 , measure of effect size for general linear models

PTSD = posttraumatic stress disorder. SES = Socioeconomic status. A higher Hollingshead score indicates lower SES. Due to missing data on one or more of the four components that made up the determination of metabolic syndrome, a total of 12 participants had missing data for the metabolic syndrome variable.

Table 3
Summary of Models of Race and Psychiatric Disorder Associations with Metabolic Disease Risk Factors.

	Current PTSD		Current MDD		History of Substance Use Disorder	
	<i>F</i>	Semipartial η^2	<i>F</i>	Semipartial η^2	<i>F</i>	Semipartial η^2
Body Mass Index						
Race	2.94	.020	2.96	.020	2.78	.021
Psychiatric D/O	4.79 [*]	.033	16.21 ^{***}	.108	.56	.004
Race x	6.10 [*]	.042	.55	.004	.00	<.001
Psychiatric D/O						
Obesity (BMI > 30)						
Race	4.19 [*]	.028	4.38 [*]	.029	2.64	.019
Psychiatric D/O	9.88 ^{**}	.066	16.32 ^{***}	.108	.94	.007
Race x	5.26 [*]	.035	.02	.000	.66	.005
Psychiatric D/O						
Abdominal Obesity (Waist Circumference)						
Race	5.47 [*]	.036	5.51 [*]	.035	5.81 [*]	.043
Psychiatric D/O	10.14 ^{**}	.067	20.00 ^{***}	.128	1.86	.014
Race x	5.19 [*]	.035	.33	.002	.05	<.001
Psychiatric D/O						
High Density Lipoproteins						
Race	1.18	.009	.68	.005	2.92	.022
Psychiatric D/O	.36	.003	2.05	.016	1.77	.014
Race x	3.21	.024	.81	.006	2.46	.019
Psychiatric D/O						
Low Density Lipoproteins						

	Current PTSD		Current MDD		History of Substance Use Disorder	
	<i>F</i>	Semipartial η^2	<i>F</i>	Semipartial η^2	<i>F</i>	Semipartial η^2
Race	3.22	.024	3.79	.027	2.39	.018
Psychiatric D/O	3.53	.026	7.41**	.054	.07	.001
Race x	.44	.003	.79	.006	.04	<.001
Psychiatric D/O						
Triglycerides						
Race	14.48***	.093	12.05**	.078	9.62**	.070
Psychiatric D/O	4.80*	.031	14.99***	.097	.10	.001
Race x	12.45**	.080	.16	.001	.34	.003
Psychiatric D/O						
Systolic Blood Pressure						
Race	1.13	.010	1.87	.017	2.88	.026
Psychiatric D/O	4.03*	.037	.99	.009	6.71*	.060
Psychiatric D/O	1.06	.010	2.85	.026	.40	.004
Diastolic Blood Pressure						
Race	1.44	.014	1.69	.016	3.15	.029
Psychiatric D/O	1.22	.012	.02	.000	3.29	.031
Race x	.00	.000	.47	.005	.90	.008
Psychiatric D/O						

*
p < .05.
**
p < .01.

p < .001.