Psychoneuroendocrinology 90 (2018) 141–147

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen

Association of psychosocial stressors with metabolic syndrome severity among African Americans in the Jackson Heart Study

Michelle I. Cardel[a,*], Yuan-I Min[b], Mario Sims[b], Solomon K. Musani[b], Akilah Dulin-Keita[c], Mark D. DeBoer[d], Matthew J. Gurka[a]

[a] Department of Health Outcomes and Biomedical Informatics, University of Florida, PO Box 100177, Gainesville, FL, 32610-0177, USA
[b] Department of Medicine, University of Mississippi Medical Center, 2500 N. State St., Jackson, MS, 39216, USA
[c] Department of Behavioral and Social Sciences, Brown University School of Public Health, Box G-S121-4, 121 S. Main St., Providence, RI, 02912, USA
[d] Department of Pediatrics, PO Box 800386, University of Virginia Health System, Charlottesville, VA, 22908-0386, USA

ARTICLE INFO

Keywords:
Stress
Metabolic syndrome
Jackson heart study
African Americans
Adults
Cardiovascular health

ABSTRACT

Introduction: Using Jackson Heart Study (JHS) data, we assessed the association between perceived psychosocial stressors and metabolic syndrome (MetS) severity in African American adults.
Methods: Participants included 3870 African American JHS participants aged 21–95 years (63.1% women; mean age 53.8 ± 13.0). Psychosocial stressors assessed included: major life events (MLEs); global stress; and weekly stress inventory. Each stress measure was classified into tertiles (low, medium, and high). Associations of psychosocial stressors with a sex- and race/ethnic-specific MetS severity Z-score were examined after adjustment for demographics and MetS risk factors (i.e., nutrition, physical activity, smoking status, and alcohol consumption).
Results: Independent of lifestyle factors, participants who reported high (versus low) perceived global stress and MLEs had significantly greater MetS severity ($p = .0207$ and $p = .0105$, respectively). Weekly stress was not associated with MetS severity. Compared to men, women reported significantly higher global stress and MLEs ($p < 0.0001$). A significant interaction between sex and MLEs ($p = .0456$) demonstrated men significantly increased their MetS severity at medium levels of stress, whereas women's MetS severity was significantly increased at high levels of MLEs.
Conclusions: In the total sample, higher reported global stress and MLEs were associated with increased risk of MetS severity, while weekly stress was not. Men's and women's stress responses to MLEs were differentially associated with MetS severity, with male MetS severity increasing significantly at lower levels of MLEs relative to women's MetS severity. These data may have implications for targeting stress-related factors in interventions to improve cardiometabolic health in African American adults.

1. Introduction

Metabolic syndrome (MetS) is a risk factor for the development of cardiovascular disease (CVD) and type 2 diabetes. Individuals with MetS are twice as likely to develop CVD (Gami et al., 2007; Mottillo et al., 2010) and up to 5 times more likely to develop type 2 diabetes (Ford et al., 2008). Common features of MetS include abdominal adiposity, hypertension, hyperglycemia, and dyslipidemia (Grundy et al., 2005a). In 2012, a nationally representative sample demonstrated the prevalence of MetS in the U.S. was 34.7%, with slightly higher rates among African Americans (AA; 35.5%) than the general population (Aguilar et al., 2015). These rates are consistent with previously published MetS prevalence in the Jackson Heart Study (JHS) with 28.3–36.4% of participants having MetS (Gurka et al., 2016).

While development of MetS is attributed to known risk factors (e.g., poor diet, physical inactivity, smoking, low education) (Cameron et al., 2004; Irwin et al., 2002), adverse psychosocial factors may also contribute to MetS development and severity. Previously published JHS work indicated AA women with depression had worse MetS severity over an 8-year period relative to AA women who were not depressed (Gurka et al., 2016). Additionally, chronic stress has been identified as a possible risk factor for MetS, as it has been posited that psychosocial stress and neuroendocrine activation exert causal effects on MetS development (Björntorp, 1996; Hjemdahl, 2002; Kaur, 2014; Rosmond, 2005). For example, the "Bjorntorp hypothesis" states that chronic stress can activate the HPA axis, which increases cortisol levels leading

* Corresponding author at: 2197 Mowry Road, 132, PO Box 100177, Gainesville, Florida, 32610, USA.
E-mail addresses: mcardel@ufl.edu (M.I. Cardel), ymin@umc.edu (Y.-I. Min), msims2@umc.edu (M. Sims), smusani@umc.edu (S.K. Musani), akilah_keita@brown.edu (A. Dulin-Keita), MDD5Z@hscmail.mcc.virginia.edu (M.D. DeBoer), matthewgurka@ufl.edu (M.J. Gurka).

https://doi.org/10.1016/j.psyneuen.2018.02.014
Received 16 November 2017; Received in revised form 8 February 2018; Accepted 14 February 2018

to an increase in visceral fat deposition, which in turn promotes development of MetS (Björntorp, 1996,2001). Yet, our group and others have demonstrated that AA tend to have less visceral adiposity relative to their non-Hispanic white counterparts (Cardel et al., 2011; Wagner and Heyward, 2000). Thus, there may be additional factors driving development of MetS in AA beyond that of excess visceral adiposity. The "Weathering Hypothesis" suggests that the health of AA deteriorates prematurely relative to non-Hispanic whites as a result of chronic exposure to social and environmental risk factors (Das, 2013; Thorpe et al., 2016). However, limited research has been conducted investigating the influence of psychosocial stressors on MetS outcomes in large samples of AA. Additionally, most research has focused on development or incidence, rather than severity, of MetS resulting from psychosocial stressors.

Thus, this study used JHS data to examine the associations of psychosocial stressors with severity of MetS among AA adults and whether relationships differed by sex. The hypothesis was that AA adults, particularly women, would have a higher MetS severity and that these relationships would be associated with psychosocial stressors.

2. Methods

2.1. Study population

The JHS is a large, population-based cohort that investigates the etiology of CVD and related risk factors in AA. Between 2000–2004, 5306 participants between the ages of 21–95 were recruited from the Jackson, Mississippi metropolitan area (Taylor et al., 2005). Three clinical examinations were conducted between 2000 and 2013 (exam 1 (baseline): 2000–2004; exam 2: 2005–2008; exam 3: 2009–2013). The Institutional Review Board (IRB) of the University of Mississippi Medical Center, Tougaloo College, and Jackson State University approved the study and all participants provided informed consent. In this cross-sectional study, data were utilized from exam 1 (2000–2004). Exam 1 included collection of baseline information through interviews in home and clinic settings. Questionnaires included inquiries about education, income, and lifestyle elements including physical activity, smoking status, alcohol consumption, and diet (Taylor et al., 2005). Exclusion criteria were presence of diabetes at baseline (n = 1233) and missing MetS severity score at exam 1 (n = 203), resulting in a sample size of 3870 for this study.

2.2. Psychosocial stress measures

Three psychosocial stress measures were included in this study: the Global Perceived Stress Scale (GPSS), Major Life Events (MLEs), and the Weekly Stress Inventory (WSI). The GPSS was given to participants in the baseline clinic examination, MLEs were determined through a telephone interview at the first annual follow-up, and the WSI was given to participants at the end of the baseline clinic examination (Gebreab et al., 2012).

The GPSS, a scale created specifically for the JHS cohort, consists of 8 domains assessing chronic stressors (i.e., employment, relationships, legal and medical issues, racism and discrimination, basic needs, etc.) throughout a 12-month period (Payne et al., 2005). Each domain is assessed using a 4-point scale of options ranging from "not stressful" to "very stressful" (0–3) with total scores varying between 0 and 24 (Payne et al., 2005). This scale was adapted from standardized stress scales including the Survey of Recent Life Experiences (Kohn and Macdonald, 1992), Perceived Stress Scale (Cohen et al., 1983), and Life Events Scale (Sarason et al., 1978). Inter-item reliability was satisfactory (Cronbach's $\alpha = 0.72$).

MLEs were assessed for the past 12 months through the use of binary responses (yes/no) to an 11-item questionnaire (Holmes and Rahe, 1967). Items assessed include: 1) serious personal illness; 2) being a victim of physical assault; 3) being a victim of robbery/home burglary; 4) losing a loved one to violent behavior; 5) experience of gunfire at home/neighborhood; 6) death of a close friend/relative; 7) major illness/injury of a close friend/relative; 8) moving to a worse residence/neighborhood; 9) losing a job; 10) being forced into retirement when you did not want to; 11) experience of divorce/separation from a spouse. Total points possible range from 0 to 11 with 0 indicating no MLEs were experienced in the past 12 months and 11 indicating that all MLEs listed were experienced in the past 12 months. Cronbach's alpha was not calculated as this measure is considered an index and not a true scale.

The WSI includes 87 items and was developed to report minor, daily stressful encounters and the magnitude of those encounters over the past week using an 8-point response scale ranging from "not stressful" to "extremely stressful" (0–7) (Brantley et al., 1987). Examples of these minor stressors include financial challenges, work-related tasks, transportation issues, relationships, household responsibilities, and leisurely activities. The score was determined by the number of minor stressors encountered (0–87). Cronbach's alpha was 0.97.

2.3. MetS classification and Z-score

At exam 1, MetS was defined using the adult ATP-III criteria (Grundy et al., 2005b). To be classified as having MetS, participants had to meet ≥3 of the following 5 criteria: concentration of triglycerides ≥1.69 mmol/L (150 mg/dL), HDL-C < 1.04 mmol/L (40 mg/dL) for men and < 1.3 mmol/L (50 mg/dL) for women, waist circumference ≥102 cm for men and ≥88 cm for women, glucose concentration ≥5.55 mmol/L (100 mg/dL), and systolic BP ≥130 mmHg or diastolic BP ≥85 mmHg (Grundy et al., 2005b).

MetS severity Z-score was calculated using formulas previously published (Gurka et al., 2012; Gurka et al., 2014). Briefly, these scores were determined with a confirmatory factor analysis (CFA) of the 5 standard components of MetS (described above (Grundy et al., 2005b)) to determine the weighted contribution of each of these components to a latent MetS "factor" on a sex- and race/ethnicity-specific basis. CFA was conducted on data from the National Health and Nutrition Examination Survey for adults ages 20–64 years (Gurka et al., 2014) divided into 6 sub-groups based on sex and self-identified race/ethnicity including: non-Hispanic white, non-Hispanic black, and Hispanic. For each of these 6 population sub-groups, loading coefficients for the 5 MetS components were determined to load on a single MetS factor and used to generate equations to calculate a standardized MetS severity score for each sub-group (found at http://mets.health-outcomes-policy.ufl.edu/calculator/). These MetS severity scores are Z-scores (with 99.75% of values ranging from −3 to 3) of relative MetS severity on a sex- and race/ethnicity-specific basis, with higher scores indicating worse MetS severity.

2.4. Covariates

Self-reported covariate measures included baseline age (continuous), sex, education, and income. Education was categorized by years of schooling completed: less than high school (< HS), high school graduate or GED equivalency through 1–3 years of college (HS4-C 1-3), and college graduate or more (C4+ years). Income was divided into 4 categories based on family size, US Census poverty levels, and year of baseline clinic visit (2000–2004): poor (less than federal poverty level), lower-middle (1–1.5 times the federal poverty level), upper-middle (more than 1.5, but less than 3.5 times the federal poverty level), and affluent (3.5 or more times the federal poverty level). Risk factors for development of MetS, including nutrition, physical activity, smoking status, and alcohol consumption were included as covariates. Both physical activity and nutrition status of each participant were classified into tertiles of ideal, intermediate, or poor based on recommendations of the American Heart Association's Life's Simple 7 cardiovascular health status metrics (Folsom et al., 2011). With consideration that

Table 1
Participant characteristics overall, by sex and by age.

	Overall	By Sex			By Age			
		Women	Men	p-value[b]	21–44	45–64	65+	p-value[c]
N	3870	2441	1429		1087	1965	818	
Demographics								
Age, Mean (SD)	53.8 (13.0)	54.2 (13.0)	53.1 (13.0)	**0.0136**	38.2 (5.7)	55.0 (5.9)	71.6 (5.1)	**< .0001**
Sex (% Women)	63.1	–	–	–	61.7	62.8	65.5	0.2211
SES/Lifestyle factors								
Education (%)								
< High School	15.4	14.9	16.2	0.5026	3.7	11.7	39.8	**< .0001**
High School 4–College 3	49.8	50.4	48.8		60.7	49.3	36.4	
College 4+	34.8	34.7	35.0		35.6	39.0	23.8	
Income (%)								
Poor	14.0	16.6	9.6	**< .0001**	16.4	10.3	19.5	**< .0001**
Lower-middle	23.3	24.9	20.6		19.3	19.7	37.0	
Upper-middle	29.9	30.8	28.2		36.4	29.5	21.9	
Affluent	32.9	27.7	41.7		27.9	40.5	21.6	
Cigarette smoking (AHA categorization) (%)								
Never smoked/quit ≥ 12 month ago	85.5	88.4	80.6	**< .0001**	84.6	84.0	90.4	**< .0001**
Quit < 12 month ago	1.2	1.2	1.2		1.9	1.0	0.8	
Current smoker	13.3	10.5	18.2		13.6	15.0	8.8	
Alcohol consumption								
Alcohol drinking in the past 12 months (%)	49.1	41.9	61.5	**< .0001**	63.7	49.4	29.3	**< .0001**
Average number of drinks per week, Mean (SD)	1.8 (5.8)	0.7 (3.4)	3.5 (8.2)	**< .0001**	2.4 (7.1)	1.8 (5.7)	0.8 (3.4)	**< .0001**
Nutrition (AHA categorization) (%)								
Ideal health	0.8	0.9	0.6	**0.0051**	0.2	0.8	1.5	**< .0001**
Intermediate health	38.9	40.8	35.5		28.8	40.9	46.6	
Poor health	60.3	58.3	63.8		71.0	58.2	51.9	
Physical Activity (AHA categorization) (%)								
Ideal health	21.0	18.2	25.8	**< .0001**	26.2	20.8	14.6	**< .0001**
Intermediate health	32.8	34.3	30.1		38.2	32.3	26.7	
Poor health	46.2	47.5	44.1		35.6	46.9	58.8	
Stressor Scores, Mean (SD)								
Global Stress	5.2 (4.4)	5.6 (4.5)	4.6 (4.2)	**< .0001**	6.6 (4.6)	5.3 (4.3)	3.1 (3.5)	**< .0001**
Weekly stress	83.7 (81.2)	86.1 (81.3)	79.5 (80.9)	0.0634[d]	105.0 (89.4)	79.8 (77.1)	55.1 (64.7)	**< .0001**
Median (IQR)	56 (28, 111.5)	59 (28.5, 113)	51 (25, 108)		75 (41, 143)	53 (27, 106)	32 (18, 65)	
Major Life Events[a]	1.3 (1.2)	1.4 (1.2)	1.2 (1.1)	**< .0001**	1.3 (1.3)	1.4 (1.2)	1.2 (1.1)	0.0904
Cumulative Stress Score	6.1 (1.7)	6.3 (1.7)	5.9 (1.6)	**< .0001**	6.6 (1.6)	6.1 (1.7)	5.4 (1.5)	**< .0001**
Health Measures, Mean (SD)								
BMI	31.1 (7.1)	32.1 (7.5)	29.3 (6.0)	**< .0001**	31.8 (8.0)	31.2 (6.9)	29.8 (6.1)	**< .0001**
Waist Circumference	98.5 (15.6)	98.0 (16.1)	99.4 (14.8)	**0.0057**	97.3 (17.2)	98.9 (15.2)	99.0 (14.4)	**0.0138**
SBP	126.4 (16.8)	125.7 (17.0)	127.6 (16.3)	**0.0006**	118.5 (13.2)	127.3 (15.8)	134.7 (18.6)	**< .0001**
DBP	76.2 (8.7)	74.9 (8.5)	78.5 (8.6)	**< .0001**	75.7 (8.6)	77.5 (8.3)	73.7 (9.0)	**< .0001**
Triglycerides	99.8 (66.4)	94.2 (51.7)	109.4 (85.0)	**< .0001**	89.4 (60.4)	104.3 (74.0)	102.9 (51.4)	**< .0001**
HDL	52.4 (14.8)	55.8 (14.8)	46.5 (12.8)	**< .0001**	49.8 (13.1)	52.5 (14.9)	55.3 (16.0)	**< .0001**
Glucose	90.4 (8.9)	89.8 (9.1)	91.5 (8.6)	**< .0001**	86.9 (7.7)	91.2 (9.0)	93.2 (8.9)	**< .0001**
ATP-III MetS Status (%)	21.5	22.7	19.5	0.0222	13.2	23.7	27.4	**< .0001**
MetS severity score Z-score	−0.070 (0.72)	−0.072 (0.73)	−0.064 (0.70)	0.7292	−0.253 (0.76)	−0.016 (0.70)	0.046 (0.67)	**< .0001**

Boldface indicates statistical significance ($p < 0.05$).
[a] Collected at the first annual follow-up interview.
[b] t-test for continuous variables, chi-square tests for categorical variables.
[c] ANOVA for continuous variables, chi-square tests for categorical variables.
[d] p = .0083 by Wilcoxon rank sum test.

some of these covariates could mediate psychosocial stress levels, results are shown before and after adjustment for the covariates using linear modeling in 3 different models.

2.5. Statistical analysis

Descriptive statistics are presented by age and sex. Chi-square tests and *t*-tests (by sex)/ANOVA (by age category) were used to compare groups by categorical and continuous variables, respectively. Each stress measure was classified into tertiles indicating low, medium, and high stress of each participant, and mean MetS z-scores were compared across the 3 stress tertile groups. Multivariable linear regression was used to compare mean MetS z-scores across the stress tertiles with predetermined sets of sequential adjustments. Model 1 adjusted for age and sex; model 2 adjusted for model 1 covariates plus education (< HS/HS-some college/college graduate); model 3 adjusted for model 2 covariates + nutrition (poor heath/other), physical activity (poor/intermediate/ideal health), smoking status (current smoker/other), and alcohol consumption (number of drinks per week). These models were used to estimate the main effect of stress on MetS severity; additional Model 3 was separately fit to explore interactions between sex and age category. Prevalence of ATP-III MetS was also estimated and compared across tertiles of the 3 stress measures. Each analysis was performed based on all available data, excluding observations with missing data.

Table 2
Linear Models of MetS Severity at Visit 1, by Sex (Model 3).

		Men			Women		
Stressor	N	Mean Z-score (95% CI)[b]	p-value[c]	N	Mean Z-score (95% CI)	p-value	p-value for Sex*Stressor Interaction
Global Stress							0.0836
Low	598	−0.065 (-0.126, −0.048)	–	824	−0.11 (-0.158, −0.059)	–	
Medium	341	−0.0026 (-0.079, 0.074)	0.1993	656	−0.10 (-0.155, −0.046)	0.8307	
High	257	−0.0067 (-0.095, 0.082)	0.2798	655	−0.036 (-0.091, 0.019)	0.0580	
Weekly Stress							**0.0020**
Low	242	−0.048 (-0.140, 0.044)	–	401	−0.12 (-0.185, −0.048)	–	
Medium	225	0.030 (−0.063, 0.122)	0.2289	444	−0.16 (-0.227, −0.097)	0.3493	
High	196	−0.153 (-0.253, −0.052)	0.1284	415	−0.064 (-0.135, 0.0036)	0.2912	
Major Life Events[a]							**0.0456**
Low	719	−0.084 (-0.137, −0.030)	–	1180	−0.095 (-0.135, −0.054)	–	
Medium	235	0.061 (−0.030, 0.152)	**0.0057**	526	−0.102 (-0.162, −0.042)	0.8349	
High	143	0.030 (−0.086, 0.146)	0.0754	325	−0.014 (-0.091, 0.062)	0.0676	

Low = first tertile; medium = s tertile; high = third tertile.
Model 3 covariates: Individual stress score (by tertile), age, education (< hs/hs-some college/college graduate), nutrition (poor heath/other), physical activity (poor/intermediate/ideal health), smoking status (current smoker/other), alcohol consumption (number of drinks per week).
Boldface indicates statistical significance ($p < 0.05$).
[a] Collected at the first annual follow-up interview.
[b] Calculated at median values of continuous covariates and observed marginal distribution of categorical covariates.
[c] Comparing to the "low" stress group.

Table 3
Prevalence of ATP-III MetS at Visit 1, by Sex (Unadjusted).

		Men			Women		
	n/N	% (95% CI)[b]	p-value[c]	n/N	% (95% CI)	p-value	p-value for Sex*Stressor Interaction[d]
Global Stress			0.8196			0.0670	0.2084
Low	131/694	18.9 (16.0, 22.0)		232/939	24.7 (22.0, 27.6)		
Med	81/399	20.3 (16.5, 24.6)		165/735	22.5 (19.5, 25.6)		
High	66/329	20.1 (15.9, 24.8)		149/747	20.0 (17.1, 23.0)		
Weekly Stress			0.3045			0.2067	0.1835
Low	55/295	18.6 (14.4, 23.6)		102/446	22.9 (19.1, 27.1)		
Med	52/255	20.4 (15.6, 25.9)		89/489	18.2 (14.9, 21.9)		
High	38/250	15.2 (11.0, 20.3)		97/481	20.2 (16.7, 24.0)		
Major Life Events[a]			0.2340			0.4282	0.2037
Low	157/852	18.4 (15.9, 21.2)		301/1332	22.6 (20.4, 24.9)		
Med	64/277	23.1 (18.3, 28.5)		126/584	21.6 (18.3, 25.1)		
High	35/178	19.7 (14.1, 26.3)		95/378	25.1 (20.8, 29.8)		

Low = first tertile; medium = s tertile; high = third tertile.
[a] Collected at the first annual follow-up interview.
[b] Clopper-Pearson (Exact) confidence limits.
[c] Chi-square test.
[d] Calculated based on logistic regression models log-likelihood statistics for type 3 analysis.

3. Results

3.1. Sample characteristics

Table 1 includes participant characteristics for the overall sample, stratified by sex and age. Data were evaluated from 3870 participants (63.1% women; mean age 53.8 years) in the JHS. A large proportion of participants were identified as having poor health in the nutritional (60.3%) and physical activity (46.2%) categories. Further, 13.3% of participants reported being current cigarette smokers.

When stratifying by age, young people (ages 21–44) reported significantly more daily stress as measured by the WSI with median scores of 75 (interquartile range (IQR) 41, 143) compared to median scores of 53 (IQR 27, 106) for those ages 45–64 and 32 (IQR 18, 65) for those ages 65+ ($p < 0.0001$). Young people also reported significantly more global stress ($p < 0.0001$) as measured by the GPSS, where the mean score for ages 21–44 was 6.6 (± 4.6) while the mean score for ages 65+ was 3.1 (± 3.5).

When stratified by sex, women reported having significantly more global stress and MLE than men ($p < 0.0001$). Global stress for women averaged 5.6 (± 4.5) while the average global stress score for men was 4.6 (± 4.2). MLEs were reported at 1.4 (± 1.2) for women and 1.2 (± 1.1) for men. The WSI median scores were 59 (IQR 28.5, 113) for women and 51 (IQR 25, 108) for men.

Prevalence of ATP-III MetS was 21.5% for the entire sample. Women (22.7%) and those in the older age group (27.4%) exhibited a higher prevalence of ATP-III MetS than men (19.5%) and those in the lowest age group (13.2%), respectively, with a significant increase by age ($p < 0.0001$). MetS severity Z-scores also showed significant increases by age ($p < 0.0001$).

Table 4
Linear Models of MetS Severity at Visit 1 (Overall).

Stressor	Model 1		Model 2		Model 3	
	Mean Z-score (95% CI)[b]	p-value[c]	Mean Z-score (95% CI)	p-value	Mean Z-score (95% CI)	p-value
Global Stress						
Low (n = 1633, 1.31 ± 1.18)	−0.089 (-0.125, −0.053)	–	−0.091 (-0.127, −0.056)	–	−0.095 (-0.134, −0.056)	–
Medium (n = 1134, 5.36 ± 1.09)	−0.092 (-0.133, −0.050)	0.9278	−0.085 (-0.126, −0.044)	0.8115	−0.061 (-0.106, −0.017)	0.2530
High (n = 1076, 11.04 ± 2.98)	−0.036 (-0.079, 0.008)	0.0683	−0.038 (-0.081, 0.005)	0.0673	−0.022 (-0.070, 0.025)	**0.0207**
Weekly Stress						
Low (n = 741, 18.8 ± 10.1)	−0.088 (-0.139, −0.037)	–	−0.088 (-0.139, −0.038)	–	−0.093 (-0.148, −0.038)	–
Medium (n = 744, 58.1 ± 14.6)	−0.094 (-0.144, −0.043)	0.8899	−0.085 (-0.135, −0.035)	0.9323	−0.096 (-0.149, −0.042)	0.9519
High (n = 731, 175.7 ± 79.1)	−0.082 (-0.133, −0.030)	0.8557	−0.090 (-0.141, −0.038)	0.9691	−0.095 (-0.152, −0.038)	0.9691
Major Life Events[a]						
Low (n = 2184, 0.50 ± 0.50)	−0.100 (-0.130, −0.071)	–	−0.099 (-0.129, −0.070)	–	−0.090 (-0.123, −0.058)	–
Medium (n = 861, 2.0 ± 0)	−0.060 (-0.107, −0.013)	0.1567	−0.057 (-0.104, −0.010)	0.1387	−0.050 (-0.101, 0.001)	0.1797
High (n = 556, 3.5 ± 0.75)	0.006 (−0.053, 0.064)	**0.0016**	−0.005 (-0.064, 0.054)	**0.0049**	0.003 (−0.061, 0.067)	**0.0105**

Low = first tertile; medium = s tertile; high = third tertile.
Model 1 covariates: Individual stress score (by tertile), age, sex; Model 2 covariates: Model 1 covariates + education (< hs/hs-some college/college graduate); Model 3 covariates: Model 2 covariates + nutrition (poor heath/other), physical activity (poor/intermediate/ideal health), smoking status (current smoker/other), alcohol consumption (number of drinks per week).
Boldface indicates statistical significance ($p < 0.05$).
[a] Collected at the first annual follow-up interview.
[b] Calculated at median values of continuous covariates and observed marginal distribution of categorical covariates.
[c] Comparing to the "low" stress group.

3.2. Relationship between baseline psychosocial stressors, ATP-III MetS status, and MetS severity

In unadjusted models (Table 4), significant associations of increased MLEs and perceived global stress with MetS severity were observed, with individuals with high MLEs exhibiting higher mean MetS severity (0.006 [95% confidence interval (CI) −0.053, 0.064]) compared to those with low levels of MLEs (−0.100 [CI −0.130, −0.071]; p = .0016). Adjustment for potential confounders (age, sex, education, nutrition, physical activity, smoking status, and alcohol consumption) did not attenuate relationships between MetS severity, global, and MLE stress. WSI was not associated with MetS severity.

Tables 2 and 3 present associations of each stress variable with MetS severity and prevalence of ATP-III MetS stratified by sex. Table 2 shows a significant interaction between sex and stress levels for both weekly stress (p = .0020) and MLEs (p = .0456). Mean Z-scores for MLEs among women approached significance at p = .0676 in the highest tertile (compared to the lowest tertile), suggesting potentially higher MetS severity for women with high MLEs. Mean Z-scores for MLEs among men were significantly greater in the medium (versus low) tertile (p = .0057). This suggests a potential higher threshold for MetS severity among women as compared to men in response to MLE stress. However, this is an associative relationship and causality cannot be inferred. Table 3 indicates that the prevalence of ATP-III MetS by sex was not significant for all 3 measures including perceived global stress, weekly stress, and MLEs.

Table 5 describes the prevalence of ATP-III MetS with all 3 stress measures. Neither perceived global stress, weekly stress, nor MLEs were related to ATP-III. Table 6 shows the impact of stressors on MetS severity, by age. In this table, none of the stress measures were significantly associated with MetS severity.

4. Discussion

This study investigated the relationships between psychosocial stressors (global stress, weekly stress, and MLEs) and the severity of MetS among AA adults. To our knowledge, this is the first study to examine the influence of psychosocial stressors on MetS severity in a large sample of AA. We found partial support for associations between stress and MetS: individuals with high (versus low) perceived global stress and MLEs had significantly greater MetS severity. Additionally, women reported higher global and MLE stress than men, but MetS severity was significantly higher at medium levels of stress in men, whereas women's MetS severity was only significantly higher at high levels of MLEs. These data indicate differences both in reported stress of AA men and women and suggests a potential differential physiologic response to this stress. However, these relationships are derived from cross-sectional data and causality cannot be inferred. Thus, future research should be conducted to characterize sex-specific physiologic responses to perceived psychosocial stressors on MetS severity.

High global stress and MLEs were positively associated with MetS severity, independent of demographic and lifestyle factors known to contribute to MetS (Cameron et al., 2004; Irwin et al., 2002). Given that global stress and MLEs were significantly related to MetS severity, yet weekly stress was not, this suggests that both the level and type of stress are important for predicting MetS severity in AA adults. Specifically, high levels of chronic stress, as reflected by perceived global stress and MLEs, may be significantly associated with MetS severity, whereas daily stressors captured by the WSI appear to not be associated with MetS severity. This builds on a prospective

Table 5
Prevalence of ATP-III MetS at Visit 1 (Overall; unadjusted).

	n	% (95% CI)[b]	p-value[c]
Global Stress			0.3668
Low (n = 1633, 1.31 ± 1.18)	363	22.2 (20.2, 24.3)	
Med (n = 1134, 5.36 ± 1.09)	246	21.7 (19.3, 24.2)	
High (n = 1076, 11.04 ± 2.98)	215	20.0 (17.6, 22.5)	
Weekly Stress			0.3720
Low (n = 741, 18.8 ± 10.1)	157	21.2 (18.3, 24.3)	
Med (n = 744, 58.1 ± 14.6)	141	19.0 (16.2, 22.0)	
High (n = 731, 175.7 ± 79.1)	135	18.5 (15.7, 21.5)	
Major Life Events[a]			0.4353
Low (n = 2184, 0.50 ± 0.50)	458	21.0 (19.3, 22.7)	
Med (n = 861, 2.0 ± 0)	190	22.1 (19.3, 25.0)	
High (n = 556, 3.5 ± 0.75)	130	23.4 (20.0, 27.1)	

[a] Collected at the first annual follow-up interview.
[b] Calculated at median values of continuous covariates and observed marginal distribution of categorical covariates.
[c] Comparing to the "low" stress group.

Table 6
Linear Models of MetS Severity at Visit 1, by Age (Model 3).

	21–44 Year			45–64 Years			65 + Years			
	N	Mean Z-score (95% CI)[b]	p-value[c]	N	Mean Z-score (95% CI)	p-value	N	Mean Z-score (95% CI)	p-value	p-value for Age*Stress Interaction
Global Stress										0.1738
Low	312	−0.28 (-0.37, −0.19)	–	795	−0.020 (-0.073, 0.033)	–	526	0.060 (−0.002, 0.122)	–	
Medium	343	−0.28 (-0.37, 0.20)	0.9508	611	0.029 (−0.030, 0.088)	0.2204	180	−0.019 (-0.125, 0.086)	0.2002	
High	426	−0.19 (-0.27, −0.11)	0.1435	549	−0.000 (-0.063, 0.063)	0.6339	101	0.144 (0.002, 0.286)	0.2885	
Weekly Stress										0.8686
Low	155	−0.27 (-0.39, −0.14)	–	385	−0.018 (-0.093, 0.057)	–	201	0.045 (−0.056, 0.146)		
Medium	250	−0.33 (-0.42, −0.23)	0.4468	388	−0.012 (-0.084, 0.061)	0.8992	106	−0.014 (-0.147, 0.119)	0.4904	
High	306	−0.31 (-0.39, −0.21)	0.6530	359	−0.017 (-0.095, 0.061)	0.9798	66	−0.060 (-0.231, 0.111)	0.2940	
Major Life Events[a]										0.0573
Low	608	−0.25 (-0.32, −0.19)	–	1096	−0.029 (-0.074, 0.016)	–	480	0.022 (−0.043, 0.086)	–	
Medium	238	−0.31 (-0.42, −0.21)	0.3463	435	0.049 (−0.020, 0.119)	0.0573	188	0.102 (−0.000, 0.205)	0.1917	
High	160	−0.13 (-0.26, 0.0038)	0.0894	307	0.017 (−0.066, 0.099)	0.3358	89	0.116 (−0.042, 0.274)	0.2789	

Low = first tertile; medium = s tertile; high = third tertile.
Model 3 covariates: Individual stress score (by tertile), sex, education (< hs/hs-some college/college graduate), nutrition (poor heath/other), physical activity (poor/intermediate/ideal health), smoking status (current smoker/other), alcohol consumption (number of drinks per week).
[a] Collected at the first annual follow-up interview.
[b] Calculated at median values of continuous covariates and observed marginal distribution of categorical covariates.
[c] Comparing to the "low" stress group.

study conducted primarily in non-Hispanic white women in the U.S. reporting that stressful life events at baseline resulted in increased risk for developing MetS over 15 years of follow-up (Räikkönen et al., 2007). Further, a large study (n = 1099) completed in a Dutch population ages 50–75 also found that men and women who reported a greater number of stressful life events had a higher risk of developing MetS during 6.5 years of follow-up (Rutters et al., 2015). However, when looking at more objective measures of stress, such as salivary cortisol, no relationship was found between level or diurnal pattern of salivary cortisol output in the Multi-Ethnic Study of Atherosclerosis (MESA) study, which included African Americans (DeSantis et al., 2011).

We also observed a significant interaction between sex and MLEs on MetS severity. Others have proposed that the ability to adapt to chronic stress may differ according to sex, and may be influenced by the regulatory effects of sex hormones (Pasquali, 2012). Specifically, alterations in sex hormones, particularly androgens, are associated with different patterns of fat deposition and distribution between men and women. Given that men tend to distribute their adiposity to abdominal and visceral regions, whereas women usually deposit fat in subcutaneous and gluteofemoral regions (Pasquali, 2006, 2012; Pasquali et al., 2008), sex differences in adiposity may influence MetS severity. Additionally, a stressful environment may lead to visceral fat accumulation as a result of chronic hypercortisolism (Kaur, 2014) and activate central pathways that stimulate the adrenals to release glucocorticoids. This process can mediate a pathogenetic role in the development and severity of MetS by leading to increased visceral adiposity, dyslipidemia, and hypertension (Rosmond, 2005). Beyond physiological sex differences, sex differences have also been cited in stress and coping styles. For example, women report more stress than men, but their coping style is more emotion-focused (Matud, 2004). How this plays out in terms of health outcomes is paradoxical in that women have lower rates of mortality, yet report higher levels of depression, psychiatric disorders, distress, and chronic illnesses than men (Denton et al., 2004). Thus, it is likely that sex differences in stress response and related health outcomes are complicated and reflect both biological and social factors, and the interplay between them. Further research is needed to confirm our observed associations and examine potential sex and chronic stress interactions in other populations.

Interestingly, when examining MetS prevalence using ATP-III definitions, psychosocial stressors were not associated with MetS in the overall sample or by sex or age stratifications. This is likely a reflection that MetS classification using the adult ATP-III criteria only takes into account the 5 physiological endpoints that define MetS and does not take into account factors that influence development of MetS including sex, race/ethnicity, and age (Cameron et al., 2004; Irwin et al., 2002). This suggests that our MetS severity Z-score may be a more relevant measure than using ATP-III criteria alone, as it allows for a weighted contribution of each of these 5 clustered components to a latent MetS "factor" on a sex- and race/ethnicity-specific basis.

4.1. Limitations

This study has many strengths including the assessment of multiple psychosocial stressors, demographic and lifestyle factors, and MetS-related variables. It also includes a well-characterized, large sample of AA and assesses MetS severity in addition to incidence of MetS. However, there are also some limitations that warrant discussion. First, only baseline measures of psychosocial stress (GPSS, MLE, and WSI) were obtained and thus, this study was unable to track for changes over time. The assessment of MLEs over the past 12 months may be subject to recall bias compared to the stress measurements that were used to determine current global and weekly stress, which were more acute measures. Additionally, perceived impact (whether negative or positive) of each MLE was not assessed as it was for global and weekly stress. For example, a MLE such as a divorce may be perceived as a positive event (Hollis et al., 1990). Further, the study was conducted in a single metropolitan area in Jackson, Mississippi, potentially limiting its generalizability to AA populations in other geographic regions.

5. Conclusions

In summary, psychosocial stressors were associated with MetS severity, with the strongest evidence displayed for MLEs impacting MetS severity. Medium levels of MLEs were associated with MetS severity in men, whereas stress from MLEs was not significantly associated in women until high levels were considered. Psychosocial stressors may be a point of intervention among AA for decreasing MetS severity and reducing health consequences and MetS risk.

Funding

The Jackson Heart Study is supported by contracts HHSN268201300046C, HHSN268201300047C, HHSN268201300048C, HHSN268201300049C, HHSN268201300050C from the National Heart, Lung, and Blood Institute (NHLBI) and the National Institute on Minority Health and Health Disparities (NIMHD). This work was supported by the National Institutes of Health National Heart, Lung, and Blood Institute (R01HL120960) and the National Center For Advancing Translational Sciences of the National Institutes of Health (UL1TR001427). The views expressed in this manuscript are those of the authors and do not necessarily represent the views of the National Heart, Lung, and Blood Institute; the National Institutes of Health; or the U.S. Department of Health and Human Services. Dr. Sims is supported by the grants P60MD002249 and U54MD008176 from the NIMHD.

Financial disclosure

No financial disclosures were reported by the authors of this paper.

Declarations of interest

None.

Acknowledgments

The Jackson Heart Study (JHS) is supported and conducted in collaboration with Jackson State University (HHSN268201300049C and HHSN268201300050C), Tougaloo College (HHSN268201300048C), and the University of Mississippi Medical Center (HHSN268201300046C and HHSN268201300047C) contracts from the National Heart, Lung, and Blood Institute (NHLBI) and the National Institute for Minority Health and Health Disparities (NIMHD). The authors wish to thank the staff and participants of the JHS. Special thanks to Darci Miller for her careful review of the manuscript.

References

Aguilar, M., Bhuket, T., Torres, S., Liu, B., Wong, R.J., 2015. Prevalence of the metabolic syndrome in the united states, 2003–2012. JAMA 313, 1973–1974.
Björntorp, P., 1996. The regulation of adipose tissue distribution in humans. Int. J. Obes. Relat. Metab. Disord. 20, 291–302.
Björntorp, P., 2001. Do stress reactions cause abdominal obesity and comorbidities? Obes. Rev. 2, 73–86.
Brantley, P.J., Waggoner, C.D., Jones, G.N., Rappaport, N.B., 1987. A daily stress inventory: development, reliability, and validity. J. Behav. Med. 10, 61–74.
Cameron, A.J., Shaw, J.E., Zimmet, P.Z., 2004. The metabolic syndrome: prevalence in worldwide populations. Endocrinol. Metab. Clin. North Am. 33, 351–375.
Cardel, M., Higgins, P.B., Willig, A.L., Keita, A.D., Casazza, K., Gower, B.A., Fernandez, J.R., 2011. African genetic admixture is associated with body composition and fat distribution in a cross-sectional study of children. Int. J. Obes. 35, 60–65.
Cohen, S., Kamarck, T., Mermelstein, R., 1983. A global measure of perceived stress. J. Health Soc. Behav. 24, 385–396.
Das, A., 2013. How does race get under the skin? : Inflammation, weathering, and metabolic problems in late life. Social Sci. Med. (1982) 77, 75–83.
DeSantis, A.S., DiezRoux, A.V., Hajat, A., Golden, S.H., Jenny, N.S., Sanchez, B.N., Shea, S., Seeman, T.E., 2011. Associations of salivary cortisol levels with metabolic syndrome and its components: the multi-ethnic study of atherosclerosis. J. Clin. Endocrinol. Metab. 96, 3483–3492.
Denton, M., Prus, S., Walters, V., 2004. Gender differences in health: a Canadian study of the psychosocial, structural and behavioural determinants of health. Social Sci. Med. 58, 2585–2600.
Folsom, A.R., Yatsuya, H., Nettleton, J.A., Lutsey, P.L., Cushman, M., Rosamond, W.D., 2011. Community prevalence of ideal cardiovascular health, by the American Heart Association definition, and relationship with cardiovascular disease incidence. J. Am. Coll. Cardiol. 57, 1690–1696.
Ford, E.S., Li, C., Sattar, N., 2008. Metabolic syndrome and incident diabetes. Curr. State Evidence 31, 1898–1904.
Gami, A.S., Witt, B.J., Howard, D.E., Erwin, P.J., Gami, L.A., Somers, V.K., Montori, V.M., 2007. Metabolic syndrome and risk of incident cardiovascular events and death: a systematic review and meta-analysis of longitudinal studies. J. Am. Coll. Cardiol. 49, 403–414.
Gebreab, S.Y., Diez-Roux, A.V., Hickson, D.A., Boykin, S., Sims, M., Sarpong, D.F., Taylor, H.A., Wyatt, S.B., 2012. The contribution of stress to the social patterning of clinical and subclinical CVD risk factors in African Americans: the Jackson Heart Study. Soc. Sci. Med. 75, 1697–1707.
Grundy, S.M., Cleeman, J.I., Daniels, S.R., Donato, K.A., Eckel, R.H., Franklin, B.A., Gordon, D.J., Krauss, R.M., Savage, P.J., Smith, S.C., 2005a. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation 112.
Grundy, S.M., Cleeman, J.I., Daniels, S.R., Donato, K.A., Eckel, R.H., Franklin, B.A., Gordon, D.J., Krauss, R.M., Savage, P.J., Smith Jr., S.C., Spertus, J.A., Costa, F., 2005b. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation 112, 2735–2752.
Gurka, M.J., Ice, C.L., Sun, S.S., DeBoer, M.D., 2012. A confirmatory factor analysis of the metabolic syndrome in adolescents: an examination of sex and racial/ethnic differences. Cardiovasc. Diabetol. 11, 1–10.
Gurka, M.J., Lilly, C.L., Oliver, M.N., DeBoer, M.D., 2014. An examination of sex and racial/ethnic differences in the metabolic syndrome among adults: a confirmatory factor analysis and a resulting continuous severity score. Metabolism 63, 218–225.
Gurka, M.J., Vishnu, A., Okereke, O.I., Musani, S., Sims, M., DeBoer, M.D., 2016. Depressive symptoms are associated with worsened severity of the metabolic syndrome in African American women independent of lifestyle factors: a consideration of mechanistic links from the Jackson heart study. Psychoneuroendocrinology 68, 82–90.
Hjemdahl, P., 2002. Stress and the Metabolic Syndrome. An Interesting but Enigmatic Association 106, pp. 2634–2636.
Hollis, J.F., Connett, J.E., Stevens, V.J., Greenlick, M.R., 1990. Stressful life events, Type A behavior, and the prediction of cardiovascular and total mortality over six years. J. Behav. Med. 13, 263–280.
Holmes, T.H., Rahe, R.H., 1967. The social readjustment rating scale. J. Psychosom. Res. 11, 213–218.
Irwin, M.L., Ainsworth, B.E., Mayer-Davis, E.J., Addy, C.L., Pate, R.R., Durstine, J.L., 2002. Physical activity and the metabolic syndrome in a tri-ethnic sample of women. Obes. Res. 10, 1030–1037.
Kaur, J., 2014. A comprehensive review on metabolic syndrome. Cardiol. Res. Pract. 2014, 21.
Kohn, P.M., Macdonald, J.E., 1992. The survey of recent life experiences: a decontaminated hassles scale for adults. J. Behav. Med. 15, 221–236.
Matud, M.P., 2004. Gender differences in stress and coping styles. Personality Individual Differences 37, 1401–1415.
Mottillo, S., Filion, K.B., Genest, J., Joseph, L., Pilote, L., Poirier, P., Rinfret, S., Schiffrin, E.L., Eisenberg, M.J., 2010. The metabolic syndrome and cardiovascular risk: a systematic review and meta-analysis. J. Am. Coll. Cardiol. 56, 1113–1132.
Pasquali, R., Vicennati, V., Gambineri, A., Pagotto, U., 2008. Sex-dependent role of glucocorticoids and androgens in the pathophysiology of human obesity. Int. J. Obes. 32, 1764–1779.
Pasquali, R., 2006. Obesity and androgens: facts and perspectives. Fertil. Steril. 85, 1319–1340.
Pasquali, R., 2012. The hypothalamic–pituitary–adrenal axis and sex hormones in chronic stress and obesity: pathophysiological and clinical aspects. Ann. N. Y. Acad. Sci. 1264, 20–35.
Payne, T.J., Wyatt, S.B., Mosley, T.H., Dubbert, P.M., Guiterrez-Mohammed, M.L., Calvin, R.L., Taylor Jr., H.A., Williams, D.R., 2005. Sociocultural methods in the Jackson Heart Study: conceptual and descriptive overview. Ethn. Dis. 15 (S6-38-48).
Räikkönen, K., Matthews, K.A., Kuller, L.H., 2007. Depressive Symptoms and Stressful Life Events Predict Metabolic Syndrome Among Middle-Aged Women. A Comparison of World Health Organization, Adult Treatment Panel III, and International Diabetes Foundation Definitions 30. pp. 872–877.
Rosmond, R., 2005. Role of stress in the pathogenesis of the metabolic syndrome. Psychoneuroendocrinology 30, 1–10.
Rutters, F., Pilz, S., Koopman, A.D.M., Rauh, S.P., Pouwer, F., Stehouwer, C.D.A., Elders, P.J., Nijpels, G., Dekker, J.M., 2015. Stressful life events and incident metabolic syndrome: the Hoorn study. Stress 18, 507–513.
Sarason, I.G., Johnson, J.H., Siegel, J.M., 1978. Assessing the impact of life changes: development of the Life Experiences Survey. J. Consult. Clin. Psychol. 46, 932–946.
Taylor Jr., H.A., Wilson, J.G., Jones, D.W., Sarpong, D.F., Srinivasan, A., Garrison, R.J., Nelson, C., Wyatt, S.B., 2005. Toward resolution of cardiovascular health disparities in African Americans: design and methods of the Jackson Heart Study. Ethn. Dis. 15 (S6-4-17).
Thorpe, R.J., Fesahazion, R.G., Parker, L., Wilder, T., Rooks, R.N., Bowie, J.V., Bell, C.N., Szanton, S.L., LaVeist, T.A., 2016. Accelerated health declines among African Americans in the USA. J. Urban Health 93, 808–819.
Wagner, D.R., Heyward, V.H., 2000. Measures of body composition in blacks and whites: a comparative review. Am. J. Clin. Nutr. 71, 1392–1402.