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Discrimination, High-Effort Coping, and Cardiovascular Risk Profiles in the Jackson Heart Study: a Latent Profile Analysis

Amber J. Johnson[1] · Kenya McCloyn[2] · Mario Sims[3]

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Abstract

African Americans exhibit heightened risk of cardiovascular disease (CVD) necessitating further examination of biological pathways contributing to CVD incidence. An integrated specificity model suggests discrimination elicits psychophysiological responses that contribute to allostatic load (AL) but may differ by level of perceived discrimination and coping style. Differing psychophysiological risk responses may contribute to varying CVD risk patterns overtime. This study sought to (1) determine CVD risk profiles using AL biomarkers and (2) determine the extent discrimination and high-effort coping (HEC) contribute to inclusion in these profiles. Using Jackson Heart Study data ($N = 4476$), a latent profile analysis (LPA) using AL indicators was conducted to identify CVD risk profiles. Multinomial logistic regression analysis estimated the odds of risk profile inclusion according to everyday discrimination, lifetime discrimination, discrimination burden, and high-effort coping, adjusting for sex, age, body mass index, and smoking status. LPA identified five profiles exhibiting *inflammatory*, *diabetes*, *hyperlipidemia*, *hypertension*, and *low risk.* Greater lifetime discrimination lowered odds of inclusion in the inflammatory risk profile relative to the *low risk* profile (OR = .82, 95% CI [.73–.93]). Greater HEC increased odds of inclusion in the hyperlipidemia (OR = 1.03, 95% CI [1.00–1.05]) and hypertension (OR = 1.02, 95% CI [1.00–1.04]) risk profiles. Greater discrimination burden with greater HEC increased the likelihood of inclusion in the inflammatory risk profile (OR = 1.07, 95% = [1.06–1.13]). The study supports the use of an integrated specificity model to examine perceived discrimination and underlying CVD risk profiles among African American populations.

Keywords African Americans · Jackson Heart Study · Cardiovascular disease · High-effort coping · Latent profile analysis

Introduction

African Americans face a greater risk of developing cardiovascular disease (CVD) than other racial/ethnic groups [1]. Recent data suggests 48% of African American women and 44% of African American men have one or more health conditions directly related to CVD including high blood pressure, diabetes, and obesity [2]. Prior studies have shown perceived discrimination increases the risk for CVD among African Americans [3, 4]. Geronimus and colleagues have offered the weathering hypothesis as a framework to examine and explain the mechanisms by which discriminatory experiences contribute to CVD risk. The weathering hypothesis posits that health disparities among African Americans are a result of social, economic, and political exclusion and adversity [5–7]. This hypothesis suggests excess morbidity and mortality among African Americans are a consequence of physiological "wear and tear" resulting from chronic stress exposure to structural inequities, discrimination, and other psychosocial threats to African American identity. Studies consistent with this hypothesis have been reported among African American samples [8–10].

One central tenet of the weathering hypothesis highlights the physiological consequences of persistent, high-effort coping on health outcomes. James [11] referred to high-effort

✉ Amber J. Johnson
amber.johnson@csulb.edu

Kenya McCloyn
kenyamccloyn023@gmail.com

Mario Sims
msims2@umc.edu

[1] Department of Health Science, California State University, Long Beach, 1250 Bellflower Blvd. FOA-13, Long Beach, CA 90840, USA

[2] Long Beach 90806, CA, USA

[3] Department of Medicine, University of Mississippi Medical Center, Jackson 39216, MS, USA

coping in African Americans as "John Henryism," a folklore reference to an African American "steel driving" man who beat a mechanical steam drill in a contest only to die of exhaustion. This story illustrates the physiological price of high-effort coping with long-term psychosocial stressors. Like John Henry, the application of "effortful, active" coping is motivated by beliefs that hard work and determination can modulate, if not eliminate, threats to well-being posed by psychosocial stress. Recurrent and chronic high-effort coping elicits prolonged allostasis that can contribute to poor health outcomes. Allostasis activates the sympathetic nervous system, hypothalamic-pituitary-adrenal axis (HPA), and the immune systems to adapt to external stressors. Whereas allostasis is the process that promotes physiological stability to external stress, allostatic load (AL) is the price of coping with recurrent and chronic allostasis [12–14]. Prolonged allostasis leads to AL, a state of impaired ability to physiologically adapt to future stressors [15, 16]. It is speculated that chronic allostatic activation is an underlying mechanism linking discrimination and high-effort coping to cardiovascular risk among African Americans [17, 18]. Consequently, prior studies have shown that African Americans exhibit greater AL than their White counterparts [5, 19]. Greater AL has also been linked to CVD incidence and mortality [16, 20].

AL has been conceptualized as the dysregulation of the multi-system interactions between primary stress mediators (e.g., cortisol, norepinephrine, epinephrine, inflammatory cytokines) and secondary stress outcomes related to cardiovascular (e.g., systolic and diastolic blood pressure), metabolic (e.g., glucose, cholesterol, and fat distribution), and immune (e.g., C-reactive protein) functioning [21]. This conceptualization suggests psychosocial stress elicits a generalized physiological response that is non-specific to physical or psychological stress [22, 23]. However, researchers have advocated for an integrated specificity model by which psychophysiological responses vary according to specific psychosocial stressors [24]. For example, individuals who are escaping a fire will activate a different set of psychological, behavioral, and physiological responses compared to discrimination-related stress responses [25]. Thus, distinct patterns of psychobiological responses to specific stressors may produce different patterns of physiological risks for CVD.

A recent study proposed the expansion of the weathering hypothesis by utilizing latent profile analysis (LPA) to examine the presence of risk profiles underlying AL [26]. LPA is a statistical person-centered approach that identifies subgroups or "profiles" that demonstrate similar response patterns among a set of indicator variables [27, 28]. This study showed individuals with AL (AL score ≥ 4) exhibited risk profiles associated with metabolic or inflammatory risks among a nationally representative sample. The current study built on this premise by proposing latent risk profiles using AL indicators may indicate various patterns of CVD risk that may be masked by examining allostatic load alone. Thus, this study first hypothesized that LPA would reveal latent CVD risk profiles using biomarkers commonly used to measure AL in a sample of African Americans. We also examined whether total AL scores would differ across CVD risk profiles. This study also sought to examine the integrated specificity of discrimination-related stress on CVD risk. It was hypothesized that discrimination and subsequent high-effort coping will contribute to inclusion in specific CVD risk profiles suggesting CVD risk patterns differ according to specific stressors and coping style.

Methods

The current study utilized data from the Jackson Heart Study (JHS), a large cohort study examining CVD among non-institutionalized African American men and women aged 35–84 living in the tri-county area (Hinds, Madison, and Rankin counties) of the Jackson, Mississippi metropolitan area. Data from this current study represents participants recruited between 2000 and 2004. Study design details have been previously reported elsewhere [29, 30]. Institutional Review Board approval was obtained from Jackson State University, University of Mississippi Medical Center, and Tougaloo College. All participants provided written informed consent before participation in the JHS.

Measures

Demographic and CVD Risk Factors

The study examined age (continuous), sex (men/women), lifestyle, and socioeconomic factors on latent risk profile group inclusion. Lifestyle factors included cigarette smoking, alcohol use, physical activity, and body mass index (BMI). Cigarette smoking was measure dichotomously as "smoking" or "not a current smoker." The JHS physical activity instrument was used to measure physical activity during sports, work/occupation, home life, and active living [31]. Physical activity consisted of a summed score across each physical activity index (range: 5–58). BMI was calculated by dividing weight (kg) by height in meters squared (m^2). Socioeconomic status comprised educational attainment and household income. Education categories included less than high school, high school graduate or some college (1–3 years), and a college degree or higher (including vocational, associate degree). Income was categorized by US Census poverty levels and family size in 2000–2004 (baseline clinic visit) as poor (less than poverty level), lower middle (1–1.5 times the poverty level), upper middle (more than 1.5 but less than 3.5 times the poverty level), and affluent (3.5 times or higher than poverty level [4].

Allostatic Load

Allostatic load (AL) was calculated using 10 biomarkers to reflect the status of the neuroendocrine system (serum cortisol); metabolic system (high-density lipoprotein [HDL] and low-density lipoprotein [LDL], triglycerides, hemoglobin A1C [HbA1c], serum creatinine, albumin); cardiovascular system (systolic and diastolic blood pressure); and immune system (high sensitivity C-reactive protein) [21]. All biomarker values were divided into quartiles with values above the 75th percentile (except HDL and albumin) considered high risk. Values below the 25th percentile for HDL and albumin were considered high risk. A continuous AL score was calculated based on the number of biomarker values in the high-risk category (0–10).

Discrimination

The JHS discrimination instrument was used to measure everyday and lifetime discrimination [32]. *Everyday discrimination* measured participants' responses to the question, "How often on a day-to-day basis do you have the following experiences?" Participants responded based on nine separate experiences such as "treated with less courtesy," "you are threatened," and "treated with less respect." The mean for participant responses ranging from 1 (never) to 7 (times) was calculated for an everyday discrimination score. *Lifetime discrimination* was measured according to participants' responses to unfair treatment over the lifetime (yes/no) across nine domains such as at school, at work, and obtaining medical care. Reports of unfair treatment were counted (0–9) across each domain to create a lifetime discrimination score [33]. Participants reported the reason for discrimination (race, sex, race, height, or weight) after answering questions for everyday and lifetime discrimination. Responses were divided into two categories that attributed discrimination to (1) race and (2) non-race-related. *Discrimination burden* was examined for participants who indicated at least one lifetime discrimination occurrence to assess the overall burden of discrimination. Discrimination burden was assessed across three questions; "When you had experiences like these, have they been …1 (not stressful), 2 (moderately stressful), or 3 (very stressful)?"; "Has discrimination interfered with your life?"; and "How much harder has life been?" Responses for these questions included 1 (not at all), 2 (a little), 3 (some), or 4 (a lot) [4]. The mean of these responses was calculated to create a discrimination burden score ranging from 1 (low burden) to 4 (greater burden). The internal reliability of everyday discrimination, lifetime discrimination, and discrimination burden in the current study was .88, .70, and .64, respectively.

High-Effort Coping

High-effort coping was measured using the 12-item John Henryism (JHN) Active Coping Scale. The JHN scale [34] captures James's conceptual definition of "effortful active coping" by African Americans as hard work and perseverance to overcome stressors such as discrimination. This included statements such as "things go wrong I work harder," "do things right myself," and "I make my life how I want." The summation of responses to a 4-point Likert scale ranging from completely true (4) to completely false (1) created a total active coping score (range: 16–48) with higher scores representing higher effortful coping. The internal reliability of this measure within this study was .78.

Statistical Analysis

This study employed LPA to develop CVD risk profiles [27, 28]. LPA utilizes maximum likelihood estimates to determine population subgroups using the probability of latent profile membership. Indicator variables comprised biomarkers commonly used to measure AL. Individuals with missing data for any indicator were excluded from the study. Values for each biomarker were standardized using Z-scores to account for the different measurement units across the ten biomarkers. Using Mplus version 7 [35], a series of models were examined using Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), and Sample Size Adjusted Bayesian Information Criterion (ssBIC) [36]. Furthermore, entropy statistics determined classification accuracy. Entropy values (0–1) closer to 1 represented a higher classification accuracy. Lastly, the Lo-Mendell-Rubin (LMR) likelihood ratio test assessed the appropriateness of K versus K-1 number of classes. Multinomial logistic regression analysis determined participants' odds of inclusion in CVD risk profiles. A preliminary analysis examined demographics, SES, and CVD risk factors to identify the impact of potential covariates on profile inclusions. This analysis identified age, sex, BMI, and smoking status as significant predictors of profile inclusion which were included in the subsequent analysis. The main analysis consisted of three models that examined the odds of profile inclusion given participants' reports of discrimination and high-effort coping. The first model examined the unadjusted odds of profile inclusion according to participants' scores for everyday discrimination, lifetime discrimination, discrimination burden, and high-effort coping. The second model examined discrimination and high-effort coping on odds of profile inclusion after adjusted for age, sex, BMI, and smoking status. A third model, fully adjusted, included interaction terms for discrimination (everyday, lifetime, burden) with high-effort coping.

Results

Fit indices (e.g., AIC, BIC, ssBIC), entropy, and LMR likelihood test using six latent profile models determine the best model fit within a final sample size of 4476 (Table 1). Though values for AIC, BIC, and ssBIC slightly decreased with increasing class, the LMR indicated a 5-class model was a better fit than a 6-class model ($p = .278$). The standardized means for each biomarker comprising the five profiles are depicted in Fig. 1. One-way analysis of variance using the F statistic examined significant variations for each continuous indicator (e.g., AL, age) across groups. Pearson's chi-squared analysis examined significant differences between categorical indicators (e.g., sex, education status) across groups.

The process of assigning labels to each profile was guided by the dominant biomarkers in each profile. Participants in the first profile (n = 147, 3.3%) exhibited higher levels of C-reactive protein and urinary albumin (see Table 2). This profile was characterized as an *inflammatory risk* resulting from existing literature suggesting elevated C-reactive protein and urinary albumin are indicative of chronic inflammation [37]. Participants in this profile yielded the highest mean age (M = 65.61, SD = 10.31), and the second highest mean AL score (M = 3.22, SD = 1.26). Participants in the second profile (n = 301, 6.7%) demonstrated higher levels of HbA1c, thus was characterized as the *diabetes risk* profile. The mean age for participants in the profile was 61.51 (SD = 9.26) with a mean allostatic load score of 2.78 (SD = 1.31). The third profile (n =2106, 47.1%) was characterized as the *low risk* profile. Participants in this profile exhibited relatively lower values for each biomarker compared to participants in other profiles, lowest mean age (M =51.91, SD =12.38), and lowest mean AL score (M = 1.06, SD =.99). Participants in the fourth profile (n = 506, 11.3%) were characterized by high levels of triglycerides and LDL with lower levels of HD and were classified as the *hyperlipidemia risk*. The mean age in this profile was 57.25 (SD = 11.76) and comprised the highest mean AL score of all profiles (M = 3.52, SD = 1.35). The fifth profile (n =1416, 31.6%) was characterized by higher systolic and diastolic blood pressure values and was labeled *hypertension risk*. The mean age in this profile was 57.06 (SD = 11.61) while the mean AL score was 2.57 (SD = 1.30).

Table 1 Fit indices for latent profile models

	AIC	BIC	SSBIC	Entropy	LMR p
2-class	89254.96	89458.77	89360.26	0.93	.03
3-class	87910.69	88186.83	88053.37	0.83	<.001
4-class	86801.71	87150.17	86981.75	0.73	<.001
5-class	**85914.56**	**86335.34**	**86131.97**	**0.73**	**<.001**
6-class	85464.47	85957.33	85719.33	0.73	.28

The p-value is provided under the column LMR p. It is less than .001

AIC, Akaike Information Criterion; *BIC*, Bayesian Information Criterion; *SSA BIC*, Sample Size Adjusted Bayesian Information Criterion; *LMR*, Lo-Mendell-Rubin likelihood ratio test

Multinomial Regression

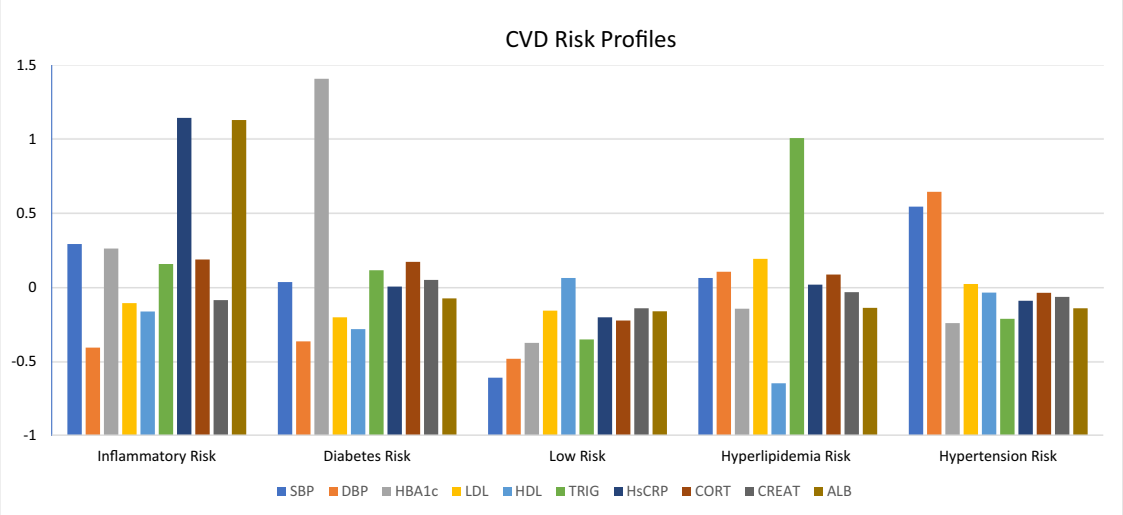
Demographic and Cardiovascular Risk Factors

Multinomial regression analysis first examined how demographic variables and cardiovascular risk factors contributed to the inclusion of each cardiovascular risk profile using the lower risk profile as the referent (Table 3). Male sex, increased age, and BMI contributed to a higher likelihood of inclusion in each cardiovascular risk profile. Those who were poor, had a high school education, and current smokers were significantly more likely to be included in the hypertension (vs. low risk) profile, while those with at least vocational, trade, or college education were significantly less likely to be in this profile. Current smokers were also significantly more likely to be included in the hyperlipidemia and hypertension profile*s* while physical activity slightly reduced the likelihood of inclusion in the inflammatory risk profile. Subsequent regression analysis controlled for sex, age, BMI, and smoking, given the extent these factors contributed to inclusion for at least two CVD risk profiles relative to the low risk profile.

Discrimination and High-Effort Coping

Multinomial logistic regression assessed the extent to which dimensions of discrimination and active coping contributed to inclusion of each CVD risk profile relative to the low risk profile (Table 4). Models adjusting for age, sex, BMI, and smoking suggested that those with higher reports of perceived lifetime discrimination had a lower likelihood of being in the inflammatory risk profile than in the low risk profile (OR = .82, 95% CI [.73–.93]). Higher effort coping scores increased the likelihood of inclusion in the hyperlipidemia (OR = 1.03, 95% CI [1.00–1.05]) and hypertension (OR = 1.02, 95% CI [1.00–1.04]) profiles. A third model included interaction terms for high-effort coping with everyday discrimination, lifetime discrimination, and discrimination burden. This model revealed a significant interaction of discrimination burden and high-effort coping suggesting higher discrimination burden with greater high-effort coping scores significantly increased the likelihood of inclusion in the inflammatory risk profile (OR = 1.07, 95% = [1.06–1.13]).

Post hoc analysis examined the impact of discrimination and high-effort coping on profile inclusion–identified sex differences, after adjusting for age, BMI, and smoking (Table 5). The results found that for men, higher scores of lifetime discrimination decreased the likelihood of inflammatory risk (OR = .79, 95% = [.64–.96]). However, higher effort coping



SPB: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; HBA1c: Hemoglobin A1c; TRIG: Triglycerides; HsCRP: High Sensitivity C-Reactive Protein; CORT: Cortisol; CREAT: Creatinine; ALB: Albumin.

Fig. 1 CVD risk profiles using standardized biomarker scores

significantly increased the likelihood of inclusion in the hypertension risk profile but only moderately (OR = 1.04, 95% = [1.02–1.07]). When accounting for the combined effect of greater lifetime discrimination and health effort coping, men had an increased likelihood of inclusion in the diabetes risk profile (OR = 1.04, 95% = [1.01–1.08]). For women, higher scores for everyday discrimination decreased the likelihood of inclusion in the hypertension risk profiles (OR = .89, 95% = [.80–.99]). The results revealed a significant interaction between discrimination burden and high-effort coping on inclusion in the inflammatory risk profile was observed among women only (OR = 1.11, 95% = [1.04–1.17]).

Discussion

This study tested the hypothesis that CVD risk profiles will exist among biomarkers commonly used to assess AL. The presence of four unique CVD risk profiles confirmed this hypothesis. African Americans in this study exhibited inflammatory, diabetes, hyperlipidemia, and hypertension risk profiles with varying AL scores. This finding is consistent with a previous study identifying physiological risk profiles in a national population-based study [26]. AL represents multisystem dysregulation of the physiological stress response systems. Though natural biological aging contributes to AL [38], age was not a sole determinant of risk profile inclusion. Participants in the *hyperlipidemia* and *hypertension risk profiles* were of similar ages, yet the mean AL score was 1 point higher for participants fitting the *hyperlipidemia risk profile*. Thus, the identified CVD risk profiles may depict varying physiological pathways that may accelerate AL.

This study also sought to explore whether inclusion in CVD risk profiles differed according to experiences of generalized perceived discrimination and high-effort coping. Though the main analysis suggested everyday racism did not contribute to CVD risk profile inclusion, post hoc analysis revealed the likelihood of profile inclusion differed by sex. While we did observe in the main analysis that participants who reported greater lifetime discrimination were more likely to be in the *low risk profile* than in the *inflammatory risk profile*, this seemed to be largely driven by men. This is consistent with population-based studies that demonstrate an inverse relationship between experiences of discrimination and inflammatory risks among African American men [37, 39]. Consistent with other studies, greater high-effort coping scores increased the likelihood of inclusion in the hypertension risk profile [11, 18].

When stratifying by sex, only high-effort coping among men increased inclusion in the hypertension risk profile, supporting research suggesting high-effort coping contributes to hypertension risk among men [11]. Low socioeconomic status and limited resources have been shown to amplify the negative impact of high-effort coping on hypertension risk. However, the modest effect of high-effort coping on hypertension profile inclusion may be explained by the potential availability of resources and support for men in this profile. The majority of participants in the hypertension risk profile had at least a vocational degree and considered upper middle class/affluent which may offer additional access to resources to buffer the physiological impact of high-effort coping.

Alternatively, everyday discrimination significantly reduced inclusion in this profile for women in favor of inclusion in the low risk profile. The low risk profile

Table 2 Descriptive statistics for allostatic load, demographic, and study variables for the latent profile classes

	Inflammatory $n = 147$	Diabetes $n = 301$	Low risk $n = 2106$	Hyperlipidemia $n = 506$	Hypertension $n = 1416$	F (X^2)
AL (*M*, *SD*) [range: 0–8]	3.22 (1.26)	2.78 (1.31)	1.06 (.99)	3.52 (1.35)	2.57 (1.30)	704.23***
Demographic factors (*n*, %)						
Age (M/SD)	65.61 (10.31)	61.51 (9.26)	51.91 (12.38)	57.25 (11.76)	57.06 (11.61)	94.99***
Female	95 (64.63%)	207 (68.77%)	1440 (68.37%)	264 (52.28%)	833 (58.83%)	66.57**
Male	52 (35.37%)	94 (31.23%)	666 (31.62%)	242 (47.82%)	583 (41.17%)	66.57***
Poor	33 (22.45%)	56 (18.60%)	296 (14.05%)	74 (14.62%)	239 (16.88%)	66.57***
Lower middle	47 (31.97%)	91 (30.23%)	481 (22.84%)	129 (25.49%)	350 (24.71%)	13.22**
Upper middle	38 (25.85%)	83 (27.57%)	655 (31.10%)	141 (27.87%)	407 (28.74%)	5.09
Affluent	29 (19.73%)	71 (23.58%)	674 (32.00%)	162 (32.02%)	420 (29.66%)	18.06***
Less than HS	58 (39.46%)	83 (27.57%)	327 (15.53%)	108 (21.34%)	286 (20.20%)	72.47***
HS only	22 (14.97%)	60 (19.93%)	333 (15.81%)	86 (17.00%)	289 (20.40%)	14.06**
Vocational/trade/university	67 (45.58%)	158 (52.49%)	1439 (68.33%)	310 (61.26%)	839 (59.25%)	69.83***
CVD risk factors (*n*, %)						
Physical activity (*M/SD*)	21.96 (8.40)	25.72 (9.45)	28.70 (9.17)	27.27 (9.45)	27.27 (9.44)	24.65**
Current smoker	18 (12.24%)	33 (11.00%)	239 (11.4%)	91 (17.98%)	216 (15.25%)	23.61***
BMI (*M/SD*)	33.82 (7.86)	34.27 (6.59)	30.99 (7.25)	32.18 (5.98)	32.07 (7.46)	18.79**
Study variables (*n*, %)						
Everyday discrimination (*M/SD*)	6.23 (.87)	6.02 (.95)	5.89 (.99)	5.94 (1.05)	5.88 (1.04)	4.86**
Attributed to race	29 (19.4%)	100 (33.22%)	856 (40.65%)	194 (39.34%)	551 (38.91%)	17.90***
Lifetime discrimination (*M/SD*)	2.23 (1.94)	2.82 (2.11)	2.99 (2.09)	3.08 (2.11)	2.97 (2.11)	5.27***
Attributed to race	67 (45.58%)	146 (48.50%)	1119 (53.13%)	275 (54.35%)	751 (53.03%)	2.47
Mean discrimination burden *(M/SD)*	2.12 (.97)	2.27 (.92)	2.14 (.89)	2.18 (.93)	2.19 (.90)	1.92
High-effort coping	41.88 (5.55)	41.75 (4.82)	41.41 (4.44)	41.98 (4.17)	41.87 (4.63)	2.71*

*$p < .05$, **$p < .01$, ***$p < .001$

yielded the largest number of participants who were younger and better educated than participants in the CVD risk profiles. Prior literature suggests younger participants who fit this profile may perceive discrimination and unfair treatment more frequently than their older counterparts [40]. Yet, African American women may not appraise everyday discrimination as inherently stressful, reducing cardiovascular responses that may increase hypertension

Table 3 Multinomial logistic regression for demographic and traditional CVD risk factors (vs. low risk) on latent profiles

Predictors	Inflammatory risk (OR, 95%CI)	Diabetes risk (OR, 95%CI)	Hyperlipidemia risk (OR, 95%CI)	Hypertension risk (OR, 95%CI)
Age	1.10 (1.08–1.12)**	1.08 (1.06–1.09)**	1.04 (1.03–1.05)**	1.04 (1.03–1.05)**
Female	.50 (.34–.74)**	.67 (.50–.89)**	.41 (.33–.51)**	.55 (.47–.65)**
Poor	1.67 (.88–2.99)	1.45 (.94–2.25)	1.07 (.75–1.53)	1.33 (1.04–1.70)*
Lower middle	1.23 (.71–2.14)	1.24 (.85–1.71)	1.04 (.773–1.39)	1.04 (.85–1.05)
Upper middle	1.31 (.78–2.21)	1.21 (.85–1.81)	.97 (.75–1.52)	1.04 (.87–1.25)
Affluent	.76 (.45–1.28)	.83 (.59–1.17)	1.03 (.79–1.33)	.96 (.80–1.56)
Less than HS	.960 (60–1.53)	.90 (.62–1.29)	.87 (.64–1.89)	.88 (.70–1.10)
HS Only	.81 (.48–1.38)	1.14 (.81–1.62)	1 (.75–1.270)	1.23 (1.01–1.50)*
Vocational/trade/college	1.23 (.73–2.10)	.875 (.62–1.329)	1 (.75–1.33)	.81 (.67–.99)*
Current smoker	1.70 (.98–2.91)	1.37 (.91–2.04)	1.90 (1.44–2.51)**	1.57 (1.27–1.93)**
BMI	1.09 (1.06–1.11)**	1.08 (1.07–1.10)**	1.05 (1.03–1.06)**	1.04 (1.03–1.05)**
Physical activity	.96 (.94–.98)**	1.00 (.99–1.02)	.99 (.98–1.00)	1.00 (.99–1.01)

OR, odds ratio; *CI*, confidence interval; *HS*, high school; *BMI*, body mass index. *$p < .05$, **$p < .01$, ***$p < .001$

Table 4 Multinomial logistic regression for racism, high-effort coping, and interaction effects on latent profiles inclusion (vs low risk)

		Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)
Inflammatory risk	ED	1.24 (.96–1.59)	1.02 (.79–1.31)	1.40 (.15–12.83)
	LD	.79 (.69–.89)***	.82 (.73–.93)**	.72 (.21–2.48)
	DB	1.33 (1.04–1.68)**	1.85 (.85–1.39)	.05 (.001–.55)*
	HEC	1.02 (.98–1.06)	1.013 (.97–1.06)	.92 (.64–1.33)
	ED X HEC			.99 (.94–1.05)
	LD X HEC			1.00 (.98–1.03)
	DB X HEC			1.07 (1.06–1.13)*
Diabetes risk	ED	1.18 (1.00–1.39)*	1.01 (.85–1.12)	1.21 (.27–5.41)
	LD	.95 (.88–1.03)	.98 (.90–1.07)	.76 (.35–1.65)
	DB	1.31 (1.10–1.56)**	1.12 (.94–1.34)	5.15 (1.02–25.52)
	HEC	1.01 (.98–1.04)	1.00 (.97–1.03)	1.10 (.85–1.43)
	ED X HEC			.99 (.96–1.03)
	LD X HEC			1.01 (.99–1.02)
	DB X HEC			.97 (.93–1.00)
Hyperlipidemia risk	ED	1.13 (.99–1.27)	1.06 (.93–1.208)	2.85 (.81–9.91)
	LD	1.03 (.97–1.09)	1.047 (.98–1.15)	1.34 (.72–2.50)
	DB	1.10 (.96–1.25)	.96 (.84–1.11)	.96 (.25–3.70)
	HEC	1.03 (1.0–1.06)*	1.03 (1.00–1.05)**	1.20 (.97–1.49)
	ED X HEC			.98 (.95–1.01)
	LD X HEC			.99 (.98–1.01)
	DB X HEC			1.00 (.97–1.03)
Hypertension risk	ED	1.00 (.92–1.08)	.93 (.85–1.02)	1.69 (.74–3.87)
	LD	.97 (.923–1.09)	.93 (.85–1.01)	1.48 (.96–2.89)
	DB	1.10 (1.00–1.21)*	.98 (.94–1.02)	.90 (.35–2.29)
	HEC	1.02 (1.01–1.04)**	1.02 (1.00–1.04)*	1.14 (.99–1.31)
	ED X HEC			.98 (.97–1.01)
	LD X HEC			.99 (.98–1.00)
	DB X HEC			.99 (.98–1.00)

*$p < .05$, **$p < .01$, ***$p < .001$

Model 1: Unadjusted model

Model 2: Adjusted model for sex, age, body mass index, and smoking status

Model 3: Fully adjusted, including interaction terms for discrimination (everyday, lifetime, burden) with high-effort coping

OR, odds ratio; *ED*, everyday discrimination; *LD*, lifetime discrimination; *DB*, discrimination burden; *HEC*, high-effort coping

risk. However, these findings necessitate further exploration of mechanisms that contribute to the perception of discrimination burden for men and women.

When accounting for the collective impact of discrimination burden and high-effort coping, participants were more likely to fit in an *inflammatory risk profile*. This is consistent with prior work showing greater immune activity among resilient individuals who engage in high-effort coping to noxious conditions such as discrimination [10]. However, stratification by sex revealed this relationship was observed only among African American women. Approximately 55% of participants in the profiles were poor to lower middle class, while approximately 54% had a high school degree or less. It is possible that women in this profile may feel more discrimination burden due to lack of the appropriate financial and educational support to resist or overcome discriminatory threats, promoting chronic high-effort coping. This is most consistent with the weathering hypothesis suggesting African American women often bear the physiological brunt of discriminatory experiences through chronic high-effort coping with social and economic exclusion [5, 7]. Elevated inflammation may be one underlying mechanism driving allostatic load and subsequent CVD for African American women.

The present study held several limitations. This study examined biomarkers commonly used to assess AL as indicators in

Table 5 Multinomial logistic regression for racism, high-effort coping (HEC), and interaction effects on latent profiles inclusion (vs low risk) for females vs males

		Females			Males		
		Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)
Inflammatory risk	ED	1.15 (.88–1.52)	.93 (.71–1.21)	1.72 (.17–16.94)	1.53 (.994–2.34)	1.23 (.80–1.90)	.09 (.003–2.9)
	LD	.82 (.72–.95)**	.90 (.78–1.03)	1.45 (.41–5.17)	75 (.62–.92) **	.79 (.64–.96)*	.26 (.04–1.86)
	DB	1.17 (.90–1.54)	.95 (.73–1.25)	.01 (.001–.17)	1.71 (1.15–2.51) **	1.46 (.97–2.18)	.42 (.01–12.94)
	HEC	1.01 (.96–1.05)	.99 (.96–1.04)	94 (.65–1.37)	.99 (.923–1.06)	.99 (.93–1.07)	.60 (.34–1.03)
	ED X HEC			.98 (.93–1.04)			1.07 (.98–1.12)
	LD X HEC			.99 (.96–1.02)			1.03 (.98–1.08)
	DB X HEC			1.11 (1.04–1.17)**			1.03 (.95–1.12)
Diabetes risk	ED	1.26 (1.04–1.53)**	1.09 (.89–1.32)	3.11 (.51–19.10)	1.08 (.85–1.37)	.88 (.69–1.12)	.17 (.018–1.60)
	LD	.98 (.89–1.08)	1.02 (.93–1.12)	1.88 (.80–4.43)	.939 (.83–1.07)	.96 (.84–1.09)	.17 (.04–.65)**
	DB	1.31 (1.09–1.60)**	1.14 (.93–1.39)	1.75 (.28–10.75)	1.31 (.996–1.72)	1.13 (.85–1.50)	7.66 (.49–118.7)
	HEC	1.02 (.97–1.03)	.99 (.961–1.03)	1.23 (.90–1.69)	1.04 (.986–1.095)	1.03 (.98–1.08)	.80 (.55–1.17)
	ED X HEC			.98 (.93–1.02)			1.04 (.99–1.10)
	LD X HEC			.99 (.97-1.01)			1.04 (1.01-.08)**
Hyperlipidemia risk	ED	1.19 (1.00–.40)*	1.05 (.89–1.24)	4.16 (.75–23.04)	1.09 (.93–1.27)	1.10 (.92–1.30)	1.46 (.31–6.90)
	LD	1.05 (.98–1.14)	1.10 (1.01–1.19)	1.40 (.61–3.19)	1.02 (.94–1.11)	1.02 (.94–1.12)	.80 (.35–1.83)
	DB	1.06 (.89–1.25)	.92 (.77–1.10)	.40 (.07–2.32)	1.00 (.83–1.21)	.97 (.80–1.18)	2.62 (.45–5.41)
	HEC	1.04 (1.01–1.08)**	1.04 (1.00–1.07)	1.23 (.93–1.64)	1.01 (.98–1.05)	1.02 (.98–1.05)	1.10 (.84–1.42)
	ED X HEC			.97 (.93–1.01)			.99 (.96–1.03)
	LD X HEC			.99 (.98–1.01).			1.01 (.99–1.03)
	DB X HEC			1.02 (.98–1.06)			.98 (.94–1.02)
Hypertension risk	ED	.97 (.88–1.07)	.89 (.80–.99)*	1.93 (.73–5.14)	1.08 (.96–1.22)	1.04 (.91–1.18)	.71 (.22–2.34)
	LD	.98 (.93–1.03)	1.00 (.95–1.05)	1.45 (.88–2.40)	.96 (.90–1.03)	.97 (.91–1.04)	1.09 (.58–2.09)
	DB	1.04 (.92–1.16)	.95 (.85–1.07)	1.43 (.49–4.15)	1.12 (.97–1.29)	1.03 (.89–1.20)	1.00 (.25–3.99)
	HEC	1.00 (.98–1.02)	1.00 (.98–1.02)	1.17 (.99–1.38)	1.05 (1.02–1.07)***	1.04 (1.02–1.07)**	1.00 (.82–1.22)
	ED X HEC			.98 (.96–1.01)			1.01 (.98–1.04)
	LD X HEC			.99 (.98–1.00)			1.00 (.98–1.01)
	DB X HEC			.99 (.97-1.02)			1.01 (.97–1.03)

*$p < .05$, **$p < .01$, ***$p < .001$

Model 1: Unadjusted model

Model 2: Adjusted model for sex, age, body mass index, and smoking status

Model 3: Fully adjusted, including interaction terms for discrimination (everyday, lifetime, burden) with high-effort coping

OR, odds ratio; *ED*, everyday discrimination; *LD*, lifetime discrimination; *DB*, discrimination burden; *HEC*, high-effort coping

the LPA. It is known that different biomarkers may be useful for assessing AL and subsequent CVD [21]. Therefore, cardiovascular risk profiles may manifest differently given the various indicator variables used in the LPA. Moreover, the number of indicator variables included in the LPA is limited due to the diminished LPA quality when inputting too many indicator variables. Thus, future studies should determine which AL variables provide the most meaningful cardiovascular risk profiles. Furthermore, this study was unable to determine if CVD risk profiles predicted specific incidents of CVD or events given this study design. Future studies can address this limitation by applying a prospective study design to determine the relationship between research participants who exhibit CVD risk profile characteristics and subsequent CVD events.

Despite the study limitations, this study is the first to employ latent profile analysis using AL biomarkers to identify underlying CVD risk profiles in a large sample of African Americans. The study supports the utility of an integrated specificity model in examining psychosocial predictors as the findings suggest the impact of discrimination and high-effort coping on CVD risk may vary within African American populations. This study provides much to be discovered regarding the identification of specific psychophysiological patterns of risk, AL, and CVD risk. Further exploration of both the replicability of the study findings and specific psychophysiological responses contributing to AL is necessary.

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Declarations

Ethics Approval Institutional Review Board approval was obtained from Jackson State University, University of Mississippi Medical Center, and Tougaloo College. All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all patients for being included in the study.

Conflict of Interest The authors declare no competing interests.

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