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Perceived Discrimination and Trajectories of C-Reactive Protein: The Jackson Heart Study

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Abstract

Introduction: Perceiving discriminatory treatment may contribute to systemic inflammation, a risk factor of cardiovascular pathophysiology. This study evaluated the association of self-reported discrimination with changes in high–sensitivity C-reactive protein and the mediating role of adiposity.

Methods: The sample included 5,145 African-Americans, aged 21–92 years, in the Jackson Heart Study. Everyday, lifetime, and burden from perceived discrimination comprised primary predictors in 3 sets of multivariable linear regression models of baseline (2000–2004) discrimination and natural logarithm of high–sensitivity C-reactive protein. Multivariable linear mixed models assessed mean changes in natural logarithm of high–sensitivity C-reactive protein over the study period (2000–2013). Mediation was quantified by percentage changes in estimates adjusted for BMI, waist circumference, and waist-to-height ratio. Multiple imputation addressed missingness in baseline covariates and in high–sensitivity C-reactive protein taken at all 3 study examinations. Analyses were conducted in 2018.

Results: In cross-sectional analyses, male participants in the middle and highest tertiles of lifetime discrimination had natural logarithm of high–sensitivity C-reactive protein levels that were 0.13 (95% CI= −0.24, −0.01) and 0.15 (95% CI= −0.27, −0.02) natural logarithm(mg/dL) lower than those in the lowest tertile. In longitudinal analyses, all participants reporting more frequent everyday discrimination had a 0.07 natural logarithm(mg/dL) greater increase in natural

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SUPPLEMENTAL MATERIAL

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logarithm of high–sensitivity C-reactive protein per examination than those reporting none (95% CI=0.01, 0.12). A similar trend emerged for lifetime discrimination and changes in natural logarithm of high-sensitivity C-reactive protein (adjusted mean increase per visit: 0.04 natural logarithm[mg/dL], 95% CI=0.01, 0.08). Adiposity did not mediate the longitudinal associations.

Conclusions: Everyday and lifetime discrimination were associated with significant high–sensitivity C-reactive protein increases over 13 years. The physiologic response to discrimination may lead to systemic inflammation.

INTRODUCTION

Compared with the general U.S. population, African-Americans develop cardiovascular disease (CVD) at younger ages, are subject to worse posthospitalization outcomes, and live more years with a CVD-related disability.[1,2] These racial disparities in heart health may be partially attributable to perceived discrimination, which has been linked to biomarkers indicative of physiologic stress, including inflammation.[3–5] One small study of older African-Americans in the Minority Aging Research Study reported a positive cross-sectional association of self-reported everyday discrimination and high–sensitivity C-reactive protein (hs-CRP),[6] a risk factor for stroke and myocardial infarction.[7] A Study of Women Across the Nation (SWAN) cohort study reported an association between discrimination and increases in hs-CRP in women without obesity.[8] Thus, hs-CRP could illuminate an important biological mechanism between discrimination and CVD disparities.

Discrimination may take any number of pathways (e.g., decreased likelihood to engage in health-preserving behaviors and fewer SES resources to prevent and manage comorbidities) toward increased CVD risk.[9–11] These risk factors can contribute to excess adipose tissue accumulation, which is associated with a low-grade inflammatory state.[12] Discrimination has been associated with prevalent obesity[13] and greater weight gain over time[14] among African-Americans. Furthermore, there is a strong association between obesity and inflammation.[15]

The present study builds upon the literature on discrimination and elevated hs-CRP levels[16–18] and is one of the first longitudinal studies to examine this association in women and men. The role of discrimination is evaluated in relation to baseline hs-CRP and changes in inflammation levels. Because waist-to-height ratio and waist circumference appear to be more strongly associated with CVD risk than BMI among African-Americans,[19] the mediating effects of these 3 anthropometric measures are compared. This research in the Jackson Heart Study (JHS), a large cohort of African-Americans, may advance the medical understanding of how discrimination contributes to CVD health.

METHODS

Study Sample

The design of the JHS, the largest prospective cohort of CVD among African-Americans (n=5,306), has been detailed extensively elsewhere.[20] Non–institutionalized African-American residents of Jackson, Mississippi, aged 21–92 years, were recruited via volunteering (30%), participating in the Atherosclerosis Risk in Communities Study (31%),

being secondary family members of existing participants (22%), or random selection from the Mississippi driver's license and identification list (17%). The main study outcomes include clinical and subclinical manifestations of coronary disease and overall mortality. Three examinations have been conducted from 2000 to 2013, consisting of biospecimen collection, interviews about health behaviors, and questionnaires regarding lived experiences. The parent study protocol was approved by the IRBs of Jackson State University, University of Mississippi Medical Center, and Tougaloo College.

Measures

An African-American interviewer administered the JHS Discrimination Instrument at Examination 1. This multidimensional measure included the occurrence, frequency, attribution, and coping responses to discrimination. Adapted from existing measures of discrimination and stress, the JHS Discrimination Instrument subscales are psychometrically sound (Cronbach α=0.78, 0.84, and 0.77, respectively, for the everyday, lifetime, and burden of lifetime discrimination subscales). The 11 dimensions assumed a priori were supported by confirmatory factor analysis.[21] The following categorizations obtained more clinically relevant effect sizes than those provided by continuous scores.

The frequency of everyday discrimination was derived from 9 questions following the prompt: *How often on a day-to-day basis do you have the following experiences?* Based on the mean of these responses, a participant's subscale score could range from never (1) to several times a day (7). Owing to the right skew of this variable's distribution, the current analysis divided scores on this subscale as follows: never (1), less frequent (>1–3), and more frequent (>3–7).

Lifetime discrimination was operationalized as the sum of participants' affirmatives to 8 questions that followed the prompt: *Now let's talk about things that may have happened over your lifetime because of such issues as your race, ethnicity, gender, age, religion, physical appearance, sexual orientation, or other characteristics*. The lifetime discrimination domains included school, medical care, housing, and employment. If participants scored 0, indicating that they had not experienced discrimination in any of this subscale's domains, they subsequently were not asked the questions regarding discrimination burden. Because lifetime discrimination followed an approximately normal distribution, scores were divided into tertiles for the current analysis: lowest (0 ndash;2), middle (3–4), and highest (5–8).

The burden of lifetime discrimination came from the mean of 3 answers regarding gradients of stress, interference with a productive life, and life difficulty. Among those who were asked questions on the subscale, possible scores ranged from 1 to 4. Those who reported no lifetime discrimination were not asked the questions about burden of discrimination (0) and were the reference group of none compared with some (1–2.5) and high (2.5–4) burden. Participants were weighed wearing light clothing and with shoes removed. BMI was calculated as weight (kg)/height (m^2). Waist circumference to the nearest centimeter was taken at umbilicus level with anthropometric tape. Waist-to-height ratio was calculated by dividing waist circumference by height in centimeters.[19] Models were fitted considering each of these measures as confounders at Examination 1 and mediators at Examination 2.

Serum hs-CRP levels (mg/L) were collected at all 3 examinations, using a latex immunoturbidimetric assay. The measurements were conducted in duplicate; any duplicates whose results fell outside 3 SDs from each other were rerun. The reliability coefficient for these replicates was 0.95 for the hs-CRP assay.[22] Because hs-CRP levels among the JHS cohort were right skewed, the values were natural logarithm (ln) transformed in all analyses to normalize their distribution.

Potential confounders (assessed at Examination 1) were selected based on the existing literature regarding factors related to discriminatory experiences, adiposity, and systemic inflammation. Age in years,[23] biological sex,[18] and educational attainment[17](less than high school; high school diploma or GED; or attended trade school, vocational school, or college) were self-reported. Self-reported smoking[23] was categorized based on American Heart Association criteria: poor (current smokers), intermediate (quit <12 months ago), and ideal (quit ≥12 months ago or never smokers).[24] Income status[25] was derived from family size and calendar year–specific poverty level (lower, lower-middle, higher-middle, and affluent). Physical activity[26] was categorized according to the American Heart Association recommendations, with participants who engaged weekly in ≥150 minutes of combined moderate and vigorous physical activity as ideal, those who engaged in <150 minutes of combined moderate and vigorous physical activity as intermediate, and those engaging in 0 minutes of physical activity as poor.[24]

Systolic and diastolic blood pressure[27] were derived from the mean of 2 sitting Hawksley random 0 sphygmomanometer readings, separated by 1 minute. Lipid panel readings provided fasting high- and low-density lipoprotein cholesterol and triglycerides.[28] Serum adiponectin concentrations[29] and percentage of HbA1c[30] (in National Glycohemoglobin Standardization Program units, using a high-performance liquid chromatography system) were collected after a minimum 8 hours of fasting. Left ventricle fraction to the nearest 5%, derived by a study cardiologist from a modified Quinones technique, served as a semiquantitative measurement of left ventricular systolic function.[31] History of CVD was based on participant self-report, diagnostic via electrocardiogram by study investigators, or verified by participants bringing in prescriptions. Renal function[32] was derived from the Chronic Kidney Disease Epidemiology Collaboration creatinine equation for estimated glomerular filtration rate, which takes into account sex, body size, and age.[33] Forced expiratory volume in 1 second and forced vital capacity in liters[34] were taken by a technician trained to use a dry rolling sealed spirometer. Current medication status was confirmed by participants bringing in prescriptions or self-reported taking each of the following classes of medication within the 2 weeks preceding Examination 1: anti-arrhythmic, anti-hypertensive, statin, and diabetes management.

Statistical Analysis

All analyses were performed in 2018 using Stata, version 15.1, and restricted to participants who had completed both the everyday and lifetime discrimination subscales (n=5,145). Baseline characteristics of the study population by frequency of everyday discrimination (never, less frequent, or more frequent) were described, testing for differences using

ANOVA (or nonparametric equivalent) for continuous variables and Pearson chi-squared test for categorical variables.

For the cross-sectional and longitudinal analyses, multiple imputation by chained equations (MICE) was performed to account for the missingness in covariates and hs-CRP. Using the predictive distribution of complete data values to fill in missing values, MICE can account for the distributions of continuous, binary, and ordinal missing variables, conditional on all other variables in the MICE chain.[35] Fifty imputed data sets were created, approximately 1 for each percent of maximal missingness in the variables. The results across imputed data sets were then pooled; the accompanying SEs were derived according to Rubin's rule, which accounts for variability within and between the imputed data sets.[36] Predictive mean matching based on a kernel set at 10 nearest neighbors addressed the non-normality of continuous variables.[37] Included in the MICE model were the following:

1. variables with no missingness—age, sex, and the daily, lifetime, and burden of lifetime discrimination subscales;
2. variables that required imputing (with their respective proportions of missingness)—weekly units of alcohol consumption (2%), estimated glomerular filtration rate (2%), HbA1c (4%) adiponectin (4%), triglycerides (9%), forced vital capacity (6%), forced expiratory volume in 1 second (6%) diastolic blood pressure (<1%) highest educational attainment (<1%), smoking categorization (2%), physical activity categorization (<1%), ln(hs-CRP) at Examination 1 (2%), ln(hs-CRP) at Examination 2 (41%), ln(hs-CRP) at Examination 3 (29%), Examination 1 BMI (<1%), Examination 2 BMI (23%), and the variables needed to create waist-to-height ratio—Examination 1 waist circumference (<1%), Examination 2 waist circumference (21%), Examination 1 height (<1%), Examination 2 height (23%); and
3. an auxiliary variable not included in the final models that was nevertheless predictive of variable missingness[38]—recruitment from the Atherosclerosis Risk in Communities Study.

In cross-sectional and longitudinal analyses, the everyday, lifetime, and burden domains of perceived discrimination were evaluated separately as the primary predictor in 3 sets of models. Multivariable linear regression was used to estimate the mean differences in the cross-sectional association of perceived everyday, lifetime, and burden of lifetime discrimination with ln(hs-CRP) levels, before and after adjustment for the covariates. The adjusted models were fitted using a backward stepwise process, based on a p-value of 0.10 for retention.

Longitudinal changes in hs-CRP were assessed using 3 sets of linear mixed models. An independent covariance structure was specified, allowing a distinct variance for each random effect and assuming all covariances equaled 0. Robust SEs were reported to account for heteroscedasticity. Model 1 evaluated the unadjusted association of each baseline discrimination measure by examination interaction terms and per-examination changes in ln(hs-CRP) between 2000 and 2013. Model 2 was adjusted for clinical, behavioral, and sociodemographic factors—retained again at a *p*-value of 0.10. Model 3 was fully adjusted

for the mediator waist-to-height ratio and the Model 2 covariates. Mediation by waist-to-height ratio at Examination 2 was evaluated using the Baron and Kenny method,[39] comparing the coefficient of interest in Models 2 and 3. Multiple sensitivity analyses were conducted to evaluate the mediating effects of waist circumference and BMI (Appendix Tables 1 and 2, available online).

RESULTS

Table 1 details the baseline characteristics of the JHS cohort. Compared with those who never experienced everyday discrimination, those who experienced most frequent discrimination were significantly younger (median age, 50 years vs 63 years, $p<0.001$); more likely to have attended vocational, trade school, or college (64% vs 42%, $p<0.001$); and more often male (41% vs 36%, $p=0.04$). Those reporting more frequent everyday discrimination also had more favorable clinical characteristics than those who reported never, including a lower percentage of those with a history of CVD (9% vs 15%, $p<0.001$) and an estimated glomerular filtration rate indicative of better kidney function (100 vs 89 mL/min/1.73 m^2, $p<0.001$). Participants who experienced the most frequent everyday discrimination had slighter higher median BMI (31 vs 30, p=0.002) and waist circumference (101 cm vs 99 cm, $p=0.02$).

Median hs-CRP levels by study visit were 0.27 (IQR=0.11, 0.57) for Visit 1, 0.28 (IQR=0.12, 0.65) for Visit 2, and 0.29 (IQR=0.12, 0.65) for Visit 3. Table 2 presents the unadjusted and adjusted cross-sectional associations of perceived discrimination with ln(hs-CRP) levels at baseline; Appendix Table 3 (available online) presents the sex-stratified unadjusted and adjusted cross-sectional associations. Male participants reporting less frequent everyday discrimination had 0.17 lower ln(hs-CRP) than those reporting never (95% CI= −0.31, −0.04, $p=0.01$; $p=0.03$ for sex interaction). A similar paradoxical negative association emerged for male participants reporting lifetime discrimination (highest vs lowest tertile: β= −0.13, 95% CI= −0.24, −0.01, $p=0.03$; $p=0.03$ for sex interaction). Conversely, everyday or lifetime discrimination was not associated with baseline levels of ln(hs-CRP) among female participants. Burden of lifetime discrimination was not cross-sectionally associated with ln(hs-CRP) among either sex.

The associations between baseline discrimination measures and 2000–2013 longitudinal changes in ln(hs-CRP) are presented in Table 3. There were not significant differences by sex (Appendix Table 4, available online). Participants who reported more frequent everyday discrimination (vs never) had a 0.07 greater increase in ln (hs-CRP) per examination in the model fully adjusted for covariates (β=0.07, 95% CI=0.01, 0.12, $p=0.02$; $p=0.51$ for sex interaction). Those in the highest (vs lowest) tertile of lifetime discrimination had increases in ln(hs-CRP) levels that were 0.04 ln(mg/dL) higher (adjusted 95% CI=0.01, 0.08, $p=0.02$; $p=0.06$ for sex interaction). Burden of lifetime discrimination was not associated with changes in ln (hs-CRP). BMI, waist circumference, and waist-to-height ratio did not mediate any of the inflammation trajectories; adjustment for these factors did not alter the magnitude of the coefficients. Figure 1 illustrates that JHS participants in the more frequent everyday discrimination category had both lowest baseline and relatively greater increases in ln(hs-

CRP) over the 3 examinations. Results in Tables 2 and 3, as well as Figure 1 used imputed data, which did not alter the overall findings from the unimputed data.

DISCUSSION

Among JHS participants, those who reported more frequent everyday discrimination and greater lifetime discrimination were observed to have a significantly greater increase in hs-CRP over 13 years than those who reported the least discrimination. For example, for a participant with an average level of hs-CRP (0.27 mg/dL), this would represent a 0.04 mg/dL increase over 13 years for those reporting more frequent everyday discrimination at baseline, compared with those reporting no discrimination. Self-reported burden of lifetime discrimination was not associated with ln(hs-CRP) over time, and adiposity measures did not mediate this association. Unexpected inverse cross-sectional associations of lifetime and everyday discrimination with hs-CRP were observed for male participants. Although modest, an independent effect of discrimination on inflammatory trajectories existed after adjustment for established risk factors among African-Americans.

Aspects of the current study's findings are consistent with results from the Minority Aging Research Study, which reported a positive association of self-reported everyday discrimination and hs-CRP in 296 older African-Americans after adjustment for clinical, behavioral, and socioeconomic factors (β=0.11, p=0.02). In contrast to this JHS study, adjustment for BMI modestly attenuated the associations observed in the Minority Aging Research Study (β =0.09, p=0.07).[6] This may be because the JHS cohort had on average lower baseline levels of hs-CRP. The longitudinal findings also somewhat conflict with those observed in SWAN (n=2,490), where discrimination did not appear to have a main effect on changes in hs-CRP among a racially diverse sample of middle-aged female participants. Moreover, in the SWAN study, there was a significant interaction between discrimination and BMI (p=0.03), where everyday discrimination was only associated with annual hs-CRP increases over 7 years among women without obesity.[8] The differences from the SWAN study results may be because the JHS cohort included men who on average reported more everyday and lifetime discrimination than women. JHS also has less frequent hs-CRP assessments and a longer follow-up period than SWAN.

The 3 measures of adiposity did not mediate longitudinal associations. Obesity was prevalent in the JHS population at Examination 1 (prevalence, 53%; median BMI, 30; IQR=26, 35), but this population had a favorable distribution of adiposity (median waist-to-height ratio, 0.6; IQR=0.5, 0.7). The role of adiposity in relation to be discrimination and inflammatory pathways may differ across racial-sex subpopulations.

Inter-related factors may explain the paradoxical negative associations between everyday and lifetime discrimination and hs-CRP at Examination 1 among male participants. The wide CIs reflect high variance in the reporting of discrimination and reduced power because of the disproportionally small sample of men in this cohort. JHS men may represent a nongeneralizable sample: male African-Americans, particularly those of older ages and lower SES, are under-represented in population health research.[40]

Limitations

Because the full JHS Discrimination Instrument was only administered at study baseline, inflammatory outcomes of changes in discrimination could not be assessed. The psychometric tool only asks the main reason for discrimination, which fails to capture the intersectional identity (e.g., being an obese older adult woman) of many JHS participants. As weight status attenuated the positive association between discrimination attributable to weight and serum hs-CRP levels in the Health and Retirement Study,[16] and adiposity influences the levels of hs-CRP produced by the liver, it is quite possible that this inflammatory effect may be compounded among African-Americans with obesity who reported multiple reasons for their discriminatory experiences.

CONCLUSIONS

This study constitutes one of the largest longitudinal analyses of baseline discrimination and hs-CRP trajectories in an all–African-American population. Use of multidimensional measures of discrimination allowed for the exploration of the inflammatory response to unfair treatment experienced on a day-to-day basis and over one's lifetime. Everyday domain of self-reported discrimination may impact hs-CRP more acutely than lifetime discrimination. Few previous studies have factored in the mediating effect of adiposity on the inflammatory response to a stressor. Use of the JHS data also allowed for informative comparisons of several adiposity phenotypes.

Future studies should continue to specify the varied reasons to which a person may attribute their unequal experiences and tease apart the factors that buffer against discrimination. To reduce the healthcare costs associated with the leading cause of disability among African-Americans, it is important for the medical community to understand that poor heart outcomes may be partially attributable to unfair treatment.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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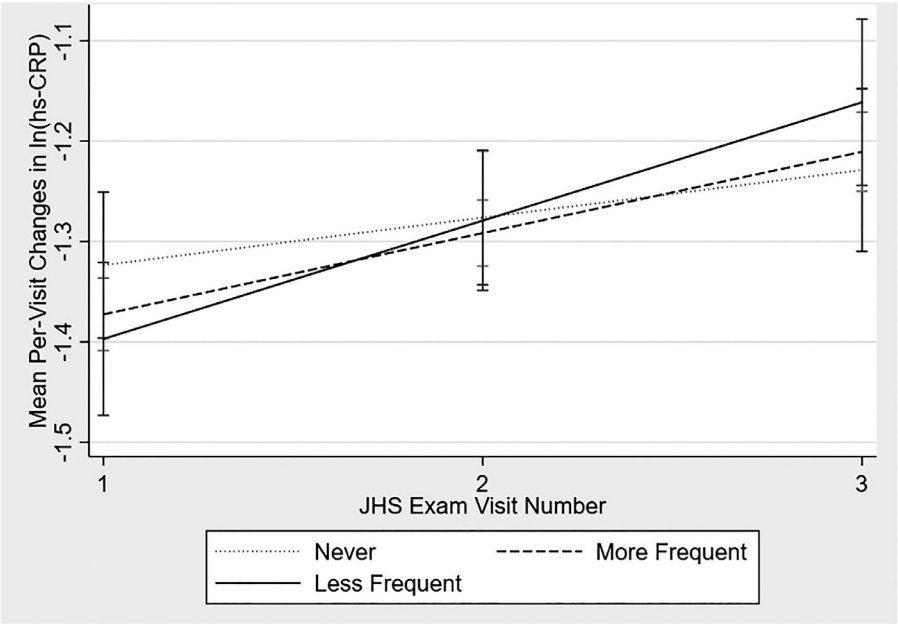

Figure 1.

Predicted ln(hs-CRP), by frequency of everyday discrimination (fully adjusted model). JHS, Jackson Heart Study; ln(hs-CRP), natural logarithm of high–sensitivity C-reactive protein.

Table 1.

Baseline Characteristics of Jackson Heart Study Participants, Aged 21–92 Years, by Frequency of Everyday Discrimination, 2000–2004 (*n*=5,145)

Characteristic	Never (1) (*n*=893)	Less frequent (>1–3) (*n*=3,441)	More frequent (>3–7) (*n*=811)	*p*-value
Age, years (IQR)	63 (54–70)	55 (45–64)	50 (43–60)	<0.001
Sex, male, *n* (%)	323 (36)	1,244 (36)	331 (41)	0.04
Highest educational attainment				<0.001
Less than high school, *n* (%)	317 (35)	613 (18)	135 (17)	
High school graduate/GED, *n* (%)	211 (23)	574 (17)	162 (20)	
Attended vocational school, trade school, or college, *n* (%)	378 (42)	2,268 (65.6)	517 (63.5)	
Income[a]				<0.001
Poor, *n* (%)	148 (20)	408 (13.8)	124 (18.2)	
Lower-middle, *n* (%)	233 (31)	667 (22.6)	169 (24.7)	
Upper-middle, *n* (%)	198 (26)	896 (30.3)	210 (30.7)	
Affluent, *n* (%)	174 (23)	983 (33.3)	180 (26.4)	
Insured, *n* (%)	792 (88)	2,992 (87)	692 (85)	0.31
AHA smoking categorization[b]				<0.001
Current, *n* (%)	97 (11)	425 (12)	149 (19)	
Quit <12 months ago, *n* (%)	7 (0.8)	42 (1.2)	12 (1.5)	
Quit 12 or months ago or never, *n* (%)	785 (88)	2,944 (86)	642 (80)	
AHA physical activity categorization[c]				<0.001
Poor, *n* (%)	517 (57)	1,614 (47)	404 (50)	
Intermediate, *n* (%)	245 (27)	1,147 (33)	258 (32)	
Ideal, *n* (%)	144 (16)	703 (20)	153 (19)	
hs-CRP, mg/L (IQR)	0.3 (0.1–0.6)	0.3 (0.1–0.6)	0.3 (0.1–0.6)	0.20
BMI, kg/m^2 (IQR)	30 (27–35)	31 (27–35)	31 (27–37)	0.002
WC, cm (IQR)	99 (90–110)	99 (89–109)	101 (90–112)	0.02
WHtR, (IQR)[d]	0.6 (0.5–0.7)	0.6 (0.5–0.7)	0.6 (0.5–0.7)	0.11
SBP, mmHg (IQR)	129 (118–140)	125 (116–136)	124 (116–136)	<0.001
DBP, mmHg (IQR)	75 (68–81)	76 (70–81)	77 (71–83)	<0.001
HDL, mg/dL (IQR)	51 (42–61)	50 (41–60)	49 (40–57)	<0.001

Characteristic	Never (1) (*n*=893)	Less frequent (>1–3) (*n*=3,441)	More frequent (>3–7) (*n*=811)	*p*-value
LDL, mg/dL (IQR)	123 (99–150)	125 (102–149)	123 (100–148)	0.12
Triglycerides, mg/dL (IQR)	92 (66–132)	90 (65–127)	89 (64–122)	0.45
Forced expiratory volume, L (IQR)[e]	2.2 (1.7–2.6)	2.3 (1.9–2.8)	2.5 (2.0–3.0)	<0.001
Ejection fraction, % (IQR)	65 (55–65)	65 (55–65)	65 (55–65)	0.08
Glomerular filtration rate, ml/min/1.73 m^2 (IQR)[f]	89 (74–104)	97 (82–110)	100 (86–114)	<0.001
Forced vital capacity, L (IQR)[g]	2.7 (2.2–3.3)	2.9 (2.4–3.5)	3.0 (2.5–3.7)	<0.001
HbA1c, % (IQR)	5.8 (5.4–6.3)	5.7 (5.3–6.1)	5.6 (5.3–6.1)	<0.001
Adiponectin, μg/mL (IQR)	4.7 (2.9–7.7)	4.3 (2.7–6.7)	3.7 (2.4–6.1)	<0.001
CVD history, *n* (%)[h]	136 (15)	339 (10)	75 (9)	<0.001
Currently taking classes of medication[i]				
Anti-arrhythmics, *n* (%)	47 (5)	145 (4)	23 (3)	0.05
Anti-hypertensives, *n* (%)	563 (63)	1,744 (51)	367 (46)	<0.001
Diabetes management, *n* (%)	168 (19)	539 (16)	108 (13)	0.01
Statins, *n* (%)	154 (17)	460 (14)	88 (11)	<0.001

[a] Income classification was derived from family income and family size and adjusted by interview year of data collection to account for inflation.

[b] Self-reported smoking status was categorized according to AHA's Life Simple 7 criteria.

[c] AHA physical activity categorization came via self-report of more than 150 weekly minutes of combined moderate and vigorous physical activity (ideal), <150 minutes of combined moderate and vigorous physical activity (intermediate), and 0 minutes of physical activity (poor).

[d] WHtR was derived from umbilical-level WC in centimeters divided by height in centimeters.

[e] Forced expiratory volume in 1 second was measured in liters.

[f] GFR was assessed via CKD-EPI Creatinine Equation.

[g] Forced vital capacity in liters came from the top 3 maneuvers.

[h] History of CVD was determined via self-report of coronary heart disease, carotid angioplasty, or stroke.

[i] Current medication status was derived from participant's self-report or bringing anti-arrhythmic, diabetes management, anti-hypertensive, statins, or blood pressure medication.

AHA, American Heart Association; CKD-EPI, Chronic Kidney Disease Epidemiology Collaboration; CVD, cardiovascular disease; DBP, diastolic blood pressure; GFR, glomerular filtration rate; HDL, high-density lipoprotein; hs-CRP, high–sensitivity C-reactive protein; LDL, low-density lipoprotein; SBP, systolic blood pressure; WHtR, waist-to-height ratio; WC, waist circumference.

Table 2.

Cross-Sectional Associations[a] of Baseline Perceived Discrimination and Serum ln(hs-CRP) Levels,[b] Jackson Heart Study, 2000–2004

	Model 1[c]			Model 2[d]		
JHSDIS subscale	**β**	**95% CI**	***p*-value**	**β**	**95% CI**	***p*-value**
Frequency of everyday discrimination						
Never (1)	ref			ref		
Less frequent (>1–3)	−0.07	−0.16, 0.02	0.12	−0.03	−0.11, 0.05	0.49
More frequent (>3–7)	−0.03	0.14,0.09	0.67	−0.05	−0.15, 0.06	0.40
Tertiled sum of lifetime discrimination						
Lowest (0–2)	ref			ref		
Middle (3–4)	−0.04	−0.16, 0.04	0.36	−0.03	−0.10, 0.04	0.42
Highest (5–8)	**−0.12**	**−0.20, −0.03**	**0.008**	**−0.08**	**−0.16, −0.01**	**0.05**
Burden of lifetime discrimination						
None (0)	ref			ref		
Some (1–2.5)	0.09	−0.01, 0.19	0.82	0.08	−0.01, 0.17	0.07
High (>2.5–4)	0.06	−0.05, 0.17	0.29	0.06	−0.05, 0.14	0.38

Note: Boldface indicates statistical significance ($p<0.05$).

[a] Using 50 imputed datasets.

[b] Natural logarithm of hs-CRP (mg/L).

[c] Univariate linear regression model of JHSDIS subscale (everyday discrimination lifetime discrimination and discrimination burden, respectively).

[d] Multivariate regression model adjusted for the following confounders as assessed at Examination 1: age in years; sex; American Heart Association smoking categorization (current, quit 12 months or less ago, quit more than 12 months ago); HbA1c; diastolic blood pressure; forced vital capacity; forced expiratory volume in 1 second; triglycerides; adiponectin; weekly units of alcohol consumed; income classification (low, lower-middle, middle-upper, affluent); taking statins, anti-hypertensives, or diabetes management medications; and American Heart Association physical activity categorization (poor, intermediate, ideal).

β, beta coefficient; hs-CRP, high–sensitivity C-reactive protein; JHSDIS, Jackson Heart Study Discrimination Instrument; ln, natural logarithm.

Table 3.

Longitudinal Associations[a] of Perceived Discrimination and Mean Change in ln(hs-CRP)[b], Jackson Heart Study, 2000–2013

	Model 1[c]			Model 2[d]			Model 3[e]		
JHSDIS subscale	***β***	**95% CI**	***p*-value**	***β***	**95% CI**	***p*-value**	***β***	**95% CI**	***p*-value**
Frequency of everyday discrimination									
Never (1)	ref			ref			ref		
Less frequent (>1–3)	0.03	−0.01, 0.07	0.13	0.03	−0.01, 0.07	0.14	0.03	−0.01, 0.07	0.14
More frequent (>3–7)	**0.07**	**0.01, 0.12**	**0.02**	**0.07**	**0.01, 0.12**	**0.01**	**0.07**	**0.01, 0.12**	**0.01**
Sum of lifetime discrimination tertiles									
Lowest (0–2)	ref			ref			ref		
Middle (3–4)	0.03	−0.01, 0.06	0.17	0.02	−0.01, 0.06	0.20	0.02	−0.01, 0.06	0.17
Highest (5–8)	**0.05**	**0.01, 0.08**	**0.03**	**0.04**	**0.01, 0.08**	**0.03**	**0.04**	**0.01, 0.08**	**0.02**
Burden of lifetime discrimination									
None (0)	ref			ref			ref		
Some (1–2.5)	0.01	−0.04, 0.06	0.64	0.01	−0.04, 0.06	0.75	0.01	−0.04, 0.06	0.70
High (>2.5–4)	0.02	−0.03, 0.07	0.41	0.02	−0.03, 0.07	0.49	0.02	−0.03, 0.07	0.45

Note: Boldface indicates statistical significance ($p<0.05$).

[a] Using 50 imputed datasets.

[b] Changes in ln(hs-CRP) (mg/L) from Examination 1 (2000–2004), to levels assessed at Examination 2 (2004–2008) and Examination 3 (2009–2013).

[c] Base LMM: JHSDIS subscale (everyday discrimination lifetime discrimination and discrimination burden, respectively), examination visit, and JHSDIS subscale by visit interaction.

[d] Adjusted for the following confounders as assessed at Examination 1: age in years; biological sex; BMI; American Heart Association smoking categorization (poor, intermediate, ideal); adiponectin; triglycerides; NGSP HbA1c; forced vital capacity; glomerular filtration rate; average weekly units of alcohol; highest educational attainment (less than high school, high school graduate/GED, attended vocational school, trade school, or college); American Heart Association physical activity categorization (poor, intermediate, and ideal); and taking statins, anti-hypertensives, or diabetes management medications.

[e] Adjusted WHtR at Examination 2 and confounders detailed in Model 2.

β, beta coefficient; hs-CRP, high–sensitivity C-reactive protein; JHSDIS, Jackson Heart Study Discrimination Instrument; ln, natural logarithm; LMM, linear mixed model; NGSP, National Glycohemoglobin Standardization Program; WHtR, waist-to-height ratio.