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Publication Date

2019-07-01

DOI

10.1016/j.annepidem.2019.05.002

Peer reviewed

HHS Public Access

Author manuscript

Ann Epidemiol. Author manuscript; available in PMC 2020 July 01.

Published in final edited form as:

Ann Epidemiol. 2019 July ; 35: 20–28.e3. doi:10.1016/j.annepidem.2019.05.002.

Differential associations between everyday versus institution-specific racial discrimination, self-reported health, and allostatic load among black women: implications for clinical assessment and epidemiologic studies

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Abstract

Purpose—Black women have the highest estimated allostatic load (AL). AL and self-perceived health are strong health predictors and have been linked to racial discrimination. Research suggests that everyday and institution-specific racial discrimination may predict different AL and self-reported health (SRH) outcomes. Furthermore, discrepancies between AL and self-perceived health could widen disparities. We estimated associations between everyday versus institution-specific racial discrimination with AL and SRH.

Methods—Data are from a San Francisco Bay Area community sample of 208 black women aged 30–50 years. Participation involved a questionnaire, self-interview, blood draw, and anthropometric measurements. Adjusted generalized linear regression models estimated associations of racial discrimination with AL and SRH.

Results—After adjusting for age, socioeconomic position, and medication use, institution-specific discrimination was negatively associated with AL (i.e., better health), whereas everyday experiences showed no association. Those reporting very-high (vs. moderate) institution-specific discrimination had lower AL ($\beta = -1.31$ [95% CI: −2.41, −0.20]; AL range: 0–15). No racial discrimination—SRH association was found.

Conclusions—For black women, (1) institution-specific racial discrimination may be differentially embodied compared with everyday experiences and (2) institutional racism may contribute to physiologic stress-regulation regardless of self-perceived health status. Potential

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Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.annepidem.2019.05.002.

factors that may contribute to an inverse racial discrimination—AL association, and future research, are discussed.

Keywords

African American; Allostatic load; Black; Minority health; Race/ethnicity; Racial discrimination; Self-reported health; Social determinants of health; Stress; Women's health

Introduction

Racial discrimination—the process by which members of a racial/ethnic group are treated unfairly based on their race/ethnicity—is significantly associated with a wide variety of adverse health outcomes and unhealthy behaviors [1–8]. The negative health consequences of racial discrimination are theorized to be cumulative over the life course with biological dysfunction emerging by mid- to late-life [9–11], eventually resulting in allostatic load—multisystem physiologic dysregulation due to chronic adaptation to stress [12–14]. Allostatic load can lead to increased risk of numerous chronic diseases such as heart disease and diabetes, and even mortality [15,16].

Blacks consistently show disproportionately higher allostatic load (AL) than other racial/ethnic groups with black women having the highest predicted values [12,17–19]. Researchers suggest that this "weathering" (i.e., physiologic wear and tear) observed among black women may be influenced by lifetime exposure to stressors related to social identity such as gender and racial discrimination [12,20–22]. That is, experiences of social marginalization are likely incorporated biologically, or embodied [23]. Self-reported experiences of racist events have been linked to AL [24,25]. Racial discrimination manifests in many forms, which may have differential impacts on health. However, the effects of different types of discrimination (e.g., interpersonal, institutional) have not been parsed analytically; and racial discrimination associations with AL among black women is underreported.

Scholars propose that institutional experiences of racial discrimination (vs. routine mistreatment) may be a key risk factor for developing chronic health conditions among blacks [10,11,26–30]. Institutional racial discrimination operates within societal organizations (e.g., universities, the workplace) to shape access to health-promoting resources and opportunities, whereas day-to-day experiences are more mundane interpersonal experiences that generally occur in public [31]. Both experiences can vary in frequency (acute vs. chronic). For black women, racial and gender inequalities interact to make avoiding high-risk exposures exceedingly more difficult over the life course regardless of socioeconomic position [19,32–35]. This may help explain, in part, mixed findings between racial discrimination and various health outcomes [2,36–47]. For instance, workplace discrimination was associated with 30% higher breast cancer risk in the Black Women's Health Study, whereas everyday exposure was weakly associated [44]. Should lifetime experiences within major social institutions be found to elicit deleterious physiological responses leading to elevated AL, interventions addressing institutional racism

may be more effective in combatting a variety of health disparities faced by black women and other socially marginalized groups.

Research shows that self-perceived health status is predictive of chronic disease risk and mortality [48–50]. but report discrepancies between self-perceived health status and more objective health indicators. For example, evidence suggests that discrepancies between self-perceived health status and underlying AL levels can widen disparities in disease prevention, detection, and treatment [17,51–53]. Those at higher subclinical disease risk, such as having elevated cholesterol or blood pressure, may not be aware of it. Self-perceived good health contributes to lower health care utilization, such as annual checkups [54,55]. Moreover, discrepancies between perceived and actual health are higher for blacks (vs. whites) [56,57]. This disconnect has considerable implications for the timely clinical detection of AL in black women, the highest at-risk group. Furthermore, associations between self-reported health and racial discrimination are mixed: everyday exposures more consistently predict poor self-reported health than institutional experiences [57–64]. Together these factors suggest a need to assess whether specific racial discrimination exposures are more predictive of health, and predict similar AL and self-perceived health status amid black women.

No studies, to date, have compared associations between everyday versus institution-specific racial discrimination with AL and self-perceived health. To address this gap, our objectives were to examine associations of (1) everyday racial discrimination with both AL and self-reported health and of (2) institution-specific racial discrimination with AL and self-reported health. We hypothesized that (1) everyday racial discrimination would be associated with worse health and that (2) institution-specific racial discrimination would be associated with worse health, and that associations would be stronger than everyday experiences due to the theoretical excess burden related to the embodiment of institutional discrimination [12,21–23].

Materials and methods

Study sample, recruitment, and participation

Data are from the African American Women's Heart & Health Study, a cross-sectional study examining the association between social-environmental stress and mental and physical health among a sample of 208 midlife black (i.e., African American) women. African American Women's Heart & Health Study methods for study sample recruitment and participation have been described in more detail elsewhere [25]. Briefly, a community sample was recruited from five San Francisco Bay Area counties using purposive sampling with multiple recruitment strategies to maximize variability across key variables of interest (e.g., racial discrimination, socioeconomic indicators). Participants completed an interviewer-administered questionnaire, computer-assisted self-interview, physical examination, and fasting venous blood draw. Study approval was provided by the Committee for the Protection of Human Subjects at the University of California, Berkeley.

Exposure variables

Everyday discrimination scale—We used a modified version of the everyday discrimination scale (EDS) asking respondents how often they experienced day-to-day unfair treatment based on their race, ethnicity, or skin color in 10 subtle yet routine life situations (e.g., receiving poorer service) ($\alpha = 0.95$) [65]. Responses were scored on a six-point Likert scale ranging from 1 = "Never" to 6 = "Almost every day", and were then added across items to generate a summary score (10–60) with higher scores reflecting greater frequency of everyday experiences. Although racial discrimination has been measured continuously to assess health associations [28,60,66], previous evidence suggests a potential curvilinear relationship [25,40]. Therefore the EDS variable was measured both continuously and categorically (5-level qualitative-based) to reflect gradual increases in annual exposure; none (≤20), low = few times/year (21–30), moderate = few times/month (31–40), high = at least once a week (41–50), and very high = ~everyday (51–60) [25].

Experiences of discrimination scale—Respondents were asked whether they have "ever been treated unfairly, judged differently than others, prevented from doing something, been hassled, or made to feel inferior because of their race, ethnicity or skin color" across eight institutional domains (e.g., at work) ($\alpha = 0.92$) [67]. One survey item from the original experiences of discrimination (EOD) scale ("getting services in a store or restaurant") was removed to avoid overlap with the same EDS item. Responses were scored on a five-point Likert scale ranging from 1 = "Never" to 5 = "6 or more times" over one's lifetime. Summary scores across the 8 EOD items ranged from 8 to 40 with higher scores reflecting greater frequency of institution-specific exposure. Like the EDS, summary scores were assessed continuously and as a 5-category qualitative-based variable representing conceptual increases of lifetime discriminatory experiences; never (=8), once (9–16), 2–3× (17–24), 4–5× (25–32), and ≥6× (33–40) [25]. To establish commensurability with the EDS, the EOD categories were then labeled as none, low, moderate, high, and very high.

Outcome variables

Allostatic load—AL comprised 15 biomarkers indicating functioning across four physiologic systems (Table 1). Following Seeman et al. and others [12,14,17,19,25,68–73], we used 75th-percentile distribution-based cut-points for biomarkers without established clinical risk criterion. All other biomarkers were coded according to established cut-points, consistent with the conceptual definition of AL as an indicator of subclinical risk. Each biomarker was coded dichotomously (0 = low risk,1 = high risk), then a summary score was created ranging from 0 to 15 with higher scores reflecting higher AL.

Self-reported health—Respondents were asked how they would rate their overall physical health at the present time. Responses were scored on a five-point Likert scale ranging from 1 = "Excellent" to 5 = "Poor" with higher scores representing worse health. Consistent with previous studies [48,62,74–78], self-reported health was assessed dichotomously (0 = "Excellent/Very good/Good", 1 ="Fair/Poor") [79].

Covariates

We assessed established empirical and theoretical confounders of the exposure—outcome association; age, education, employment, poverty, and marital/partnership status. Other covariates included medication use (cardiovascular and diabetes) (Information regarding the specific type of cardiovascular and diabetes medications taken by participants was not collected. Without knowing the specific medication and which biomarker(s) are targeted, we cannot account for it in our 15-biomarker AL measure (e.g., antihypertensives lower blood pressure; statins lower cholesterol). To preserve internal validity and account for medication use, we adjusted for medication use. We evaluated potential confounding by indication using stratified and bias analyses, alas cell sizes were too small (see [80,81]). However, stepwise regression showed medication use was significant in final models ($P < .05$) and that adjusting for it did not introduce bias or diminish the precision of the estimates.) Because research suggests that health behaviors and access to health-promoting resources may be on the pathway between racial discrimination and AL [1,10,19,66,69,82–87], health insurance and behaviors were identified as mediators and thus were not included in the analysis to avoid overcontrolling (see Supplemental Fig. 1). Except for age (measured in years), all other covariates were dichotomized at established risk levels (0 = low, 1 = high) to maximize power. These included 1 = ≤ high school diploma, 1 = not employed, 1 = ≤ 100% federal household poverty threshold, 1 = not married/domestically partnered, and 1 = current medication use. In addition, a composite socioeconomic position (SEP) measure was generated using the four dichotomous SEP variables. SEP summary scores ranged from 0 to 4 with higher values representing worse SEP.

Statistical analysis

STATA SE 13.1 was used for all statistical analyses. Data were missing at random ($P > .10$) which was ≤5%. Thus, we used multiple imputation ($m = 20$) to account for missingness [88]. In the final models, one observation had more than one missing variable within the linear combination of predictors and within the AL response variable resulting in computational problems, and was therefore dropped before imputation. Biomarkers were log-transformed before imputation to satisfy assumptions of normality, as needed. Statistical differences between exposure and outcome variables were assessed using bivariate analysis methods (e.g., *t* test), as applicable. Linear regression was used to estimate EDS and EOD associations with AL, whereas logistic regression was used to estimate self-reported health associated with each exposure, adjusted for covariates significant at $P < .10$. Similar to previous work [25,40], moderate discrimination was selected as the reference group of categorical regression models given that episodic stress exposure is self-regulatory and considered health-protective [13,89,90]. We conducted model diagnostic tests, as appropriate (e.g., heteroscedasticity). We assessed relative efficiency after imputation to ensure the simulated data did not inflate residual variance [88,91]. Sensitivity and bias analyses were performed on regression models [80,81]. We found no evidence of linear associations for AL or for self-reported health with either discrimination scale, as anticipated; hence, our final models are reported using the qualitative-based EDS and EOD variables described previously. Parameters for power calculations are reported in the final regression table [92].

Results

Sample characteristics

Table 2 presents the sample distribution of sociodemographic characteristics.

Everyday experiences of discrimination

Half of the sample reported low to moderate levels of everyday discrimination (i.e., 1–3×/year) (Table 2). Approximately, 1 in 5 (21%) reported high to very high levels (i.e., at least 1×/week to ~ everyday). Figure 1 shows the distribution of reported occurrences by each scale item. The majority reported racial discrimination in 6 of 10 “everyday” situations.

Experiences of institution-specific discrimination

Approximately 2 in 3 women (64%) reported experiencing low to moderate levels of institutional racial discrimination (i.e., 1–3× in ≥1 domain) (Table 2). Figure 2 presents the frequency of reported experiences within each domain. A quarter reported high to very high levels (i.e., ≥4–6× within ≥1 domain), and over half reported at least one racial discrimination experience in 7 of 8 domains.

Allostatic load

Mean AL score was 5.96 ± 2.24 (range 0–15) (Table 2).

Self-reported health

Precisely 3 in 4 women reported good to excellent health (75%) (Table 2).

Significance of exposure and outcome differences

Bivariate analyses compared exposure-to-exposure, outcome-to-outcome, and exposure-to-outcome differences (see Appendices A.3–4). Significant differences were found between EDS and EOD exposure measures ($\chi^2 = 151$; $P < .01$). Mean AL did not vary by self-reported health status ($t = -0.51$; $P = .69$). Estimates showed lower mean AL among “very high” (vs. “moderate”) EOD levels ($F = 2.56$; $P = .04$) and no variation for EDS ($F = 1.21$; $P = .31$). No differences were found between either EDS or EOD measure with self-reported health ($\chi^2 = 4.05$; $P = .40$ and $\chi^2 = 2.08$; 0.72, respectively).

Multivariable linear regression models

Figures 3 and 4 show the adjusted estimates for AL and self-reported health, respectively, for each EOD and EDS level. Compared with those reporting moderate discrimination (reference), there was a negative association between EOD and AL for those reporting very high discrimination ($\beta = -1.31$; 95% confidence interval = −2.41, −0.20) (Table 3). No significant association was found for EDS and AL. Self-reported health was not associated with EDS or EOD.

Discussion

Summary of findings

In this study, we examined whether self-reported experiences of everyday versus institution-specific racial discrimination showed differing associations with AL and with self-reported health in a community sample of midlife black women. There were four main findings: as hypothesized, we found (1) differential associations between everyday versus institution-specific discrimination and health and (2) divergent associations between institution-specific racial discrimination and AL versus self-reported health. However, contrary to our hypothesis, we found (3) that racial discrimination did not predict self-reported health and (4) a negative association between chronic (vs. moderate) exposure to institutional racial discrimination and AL. Women reporting “very high” levels of EOD had lower levels of AL whereas EDS showed no association. These findings suggest that factors associated with reporting a high burden of institutional racial discrimination may contribute to lower subclinical disease risk for black women, and that the underlying biological manifestation of chronic exposure within major institutions may diverge from associations with health perception.

EOD and AL

Similar to our initial study [25], AL was lower among those reporting chronic institution-specific racial discrimination. Likewise, previous studies have shown inverse associations between adverse life experiences and biological stress reactivity [93–95]. Carpenter et al. demonstrated that childhood maltreatment predicted decreased adrenocorticotropin hormone and cortisol reactivity in adults [94]. Lovallo et al found an inverse dose-dependent effect of adverse life events on cortisol levels and heart rate [93]. We cannot make inferences about stress reactivity patterns because of the cross-sectional nature of our study. However, our finding provides support for the notion that reporting chronic racial discrimination may promote a blunted stress-response for black women.

The paradoxical relationship between reporting high levels of racial discrimination and a reduced stress-response has been previously reported [25,28,40,46]. Krieger and Sidney showed that working-class blacks reporting the highest frequency of EOD had lower risk of elevated systolic blood pressure, and the effect was stronger for women [40]. This buffered response related to racial discrimination has also emerged for cardiovascular disease risk among whites reporting an implicit bias connecting themselves to being a target of racial discrimination [28]. Scholars suggest that positive perceptions of within-group racial identity, as well as attributing negative experiences to systemic racism versus self-blame, may be health-protective [96–102].

Conversely, stress theory posits a biopsychosocial mechanism by which one’s appraisal of a recurrent threat, perceived resources, and working memory of previous exposures can result in a reduced (i.e., maladaptive) stress response [13,89,103–105]. Inadequate reactivity to stressors can be health-damaging long term via hyperactivity of supplementary mediators [13]. Indeed, in comparing the distributions of at-risk biomarkers within our sample, women taking cardiometabolic medications were at higher risk than nonmedication users (Appendix

A.6). This interpretation proposes a deleterious biopsychosocial pathway. Because black women show the highest predicted AL than other racial/gender groups, an attenuated value could erroneously appear normal or “healthy”, masking the harmful changes in regulatory systems [12,13,89]. Further within-racial group longitudinal studies may help disentangle healthy versus unhealthy stress-response mechanisms associated with racial discrimination.

EDS and AL

Contrary to previous work, we did not find an association between everyday discrimination and AL, which could be explained by our EDS scale explicitly attributing the unfair treatment to one’s race/ethnicity [106]. Four recent publications found positive associations between everyday discrimination and AL using similar versions of the EDS [19,69,107,108]. However, these studies measured general unfair treatment as opposed to experiences of discrimination attributed to race/ethnicity. A meta-analysis including 30 years of studies examining chronic psychosocial factors and acute physiologic responses found that stress reactivity is contingent on the specific nature of the psychosocial exposure [109]. Research shows that most blacks attribute their discrimination experiences to race/ethnicity (vs. other social identities) [28,107,110,111], report racial discrimination as a predominant psychosocial stressor [40,108,110,112,113], and report more chronic experiences (vs. whites) [28,111]. Thus, our result may be partly representing the unique embodiment of routine race-based discrimination for black women as opposed to more general discriminatory exposures. In addition, commonplace stressors become highly predictable and less stressful resulting in a diminished stress response (e.g., military parachute training) [114]. This may help explain why daily racial discrimination showed no association with AL in our study versus studies measuring less predictable unfair treatment, such as getting housing.

EOD, EDS, and self-reported health

Our divergent finding from the literature of a null racial discrimination—self-reported health association for both EOD and EDS could be explained by our restricted sample of middle-aged black women. Racial discrimination levels likely did not vary enough between self-reported health categories to detect adjusted associations. Previous studies have demonstrated positive EDS associations with self-reported health comparing blacks with whites, comparing men with women, and based on estimates adjusted for age and gender [63,65,115]. Findings related to institution-specific racial discrimination are more disparate: EOD has predicted worse self-reported health among U.S. black CARDIA study participants [62] yet showed no association among U.S.-born nor foreign-born blacks in Boston [60]. Subtle differences in study sample composition likely contribute to such varied results. Our finding adds to this literature by demonstrating no within-group differences among Bay Area black women reporting any exposure to everyday or institutional racial discrimination. More importantly, findings suggest that racism-related self-perceived health may differ from actual disease risk, which, if further validated, could contribute substantially to our understanding of racial health disparities.

Limitations and strengths

There are several methodological considerations for this research. First, our cross-sectional design limits causal inference yet provides important evidence that may help inform future work in this area. We recruited a nonprobability sample intended to maximize variability in the exposure. Findings are not generalizable. However, our sample's distribution of covariates was largely comparable with midlife black women living in the same counties in the 2013 American Community Survey [116]. Next, our utilization of two well-validated, reliable discrimination scales for exposure assessment strengthened the study's internal validity while also allowing for comparability of our results with other studies. The EDS and EOD measures were highly but not perfectly correlated ($r = 0.74$) [117] showing that, conceptually, they are capturing different experiences and are not interchangeable (see Appendix A.4). Moreover, collapsing summary scores of discrimination responses into 5 categories across multiple life domains risks misclassifying those with highly frequent experiences in just one or two domains (e.g., being called names "almost everyday") as low risk when such exposure frequency could be considered chronic. Nevertheless, constructing discrimination categories was supported by our sensitivity analysis comparing continuous and quintile-based exposure variables, which provided evidence that there were no linear or meaningful distribution-based associations (see Supplemental Tables 1–3). Patterns of qualitative-based versus distribution-based EDS and EOD measures showed no agreement (0% and 13%, respectively; see Appendix A.5), suggesting that qualitative categories may better represent conceptual increases in exposure. Furthermore, exposure and outcome misclassification due to poor recall is a fundamental limitation to any observational design using self-report. Finally, we greatly reduced potential misclassification of physical health by using biomarkers for AL assessment.

Conclusion

This study provides preliminary evidence that institutional racial discrimination may contribute to physiologic stress-regulation for midlife black women regardless of self-perceived health status. These findings introduce the potential utility of allostatic load as a clinical tool to assess black women's underlying health risk. Furthermore, policy and program interventions addressing institutional racism may help mitigate chronic disease disparities. Additional research is needed to elucidate mediators and moderators that buffer the physiologic consequences of racial discrimination, particularly within major social institutions with a focus on black women taking medication to manage high cardiometabolic risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by research grants from University of California, Berkeley Hellman Fund, USA; UCB Population Center, USA; UCB Research Bridging Grant, USA; Robert Wood Johnson Health and Society Scholars Program (UCB site), USA; UC Center for New Racial Studies, USA; and the UCB Institute for the Study of Societal Issues (ISSI), USA. M.D.T. was partially supported by NIGMS grant UL1GM118985, USA, and by a Ford

Foundation Predoctoral Fellowship administered by the National Academies of Sciences, Engineering, and Medicine; E.K.M. was partially supported by grant GTDR14301469 from the Susan G. Komen Foundation; A.M.A. was also partially supported by NIMHD grant P60MD006902, USA. Study sponsors did not participate in study design, data collection, data analysis, interpretation of study results, or drafting of the manuscript.

The authors wish to thank Holly Berryman Stern from the UCB Tang Center for support related to laboratory specimens, Roger Hoffman of Westportal for in-kind support in building the CASIC platform, Miguel Dorta from StataCorp, Maureen Lahiff for comments on statistical analysis, Kirsten Bibbins-Domingo for comments related to physiologic dysregulation among vulnerable populations, and the many undergraduate, graduate, and postdoctoral students working as volunteer research assistants. They also thank the UCB epidemiology graduate students and members of the HEART Research Group (Health Effects Associated with Racism Threat) at the UCB ISSI for comments regarding the refinement of this article. They would also like to thank the anonymous reviewers for comments on previous drafts of this article.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Appendix

Table A1

Frequency (%) of racial discrimination (EDS) among AAWHHS participants ($n = 207$)

Everyday discrimination domain	Never	<1/year	Few times/ year	Few times/ month	≥1/week	Almost everyday	≥1/year
You are treated with less courtesy than other people	24 (11.82)	25 (12.08)	66 (31.88)	39 (18.84)	27 (13.30)	26 (12.56)	158 (76.33)
You are treated with less respect than other people	35 (16.91)	30 (14.49)	57 (27.54)	32 (15.46)	25 (12.08)	28 (13.53)	142 (68.60)
You receive poorer service than other people at restaurants or stores	28 (13.53)	43 (20.77)	57 (27.54)	37 (17.87)	19 (9.18)	23 (11.11)	136 (65.70)
People act as if they think you are not smart	49 (23.67)	35 (16.91)	48 (23.19)	21 (10.14)	28 (13.53)	26 (12.56)	123 (59.42)
People act as if they are afraid of you	69 (33.33)	33 (15.94)	32 (15.46)	26 (12.56)	22 (10.63)	25 (12.08)	105 (50.73)
People act as if you are dishonest	64 (30.92)	43 (20.77)	38 (18.36)	20 (9.66)	14 (6.76)	28 (13.53)	100 (48.31)
People act as if they are better than you are	26 (12.56)	24 (11.59)	45 (21.74)	40 (19.32)	25 (12.08)	47 (22.71)	157 (75.85)
You are called names or insulted	95 (45.89)	41 (19.81)	34 (16.43)	12 (5.80)	12 (5.80)	13 (6.28)	71 (34.30)
You are threatened or harassed	118 (57.00)	33 (15.94)	23 (11.11)	10 (4.83)	9 (4.35)	14 (6.76)	56 (27.05)
You are followed around in stores	46 (22.22)	37 (17.87)	61 (29.47)	18 (8.70)	17 (8.21)	28 (13.53)	124 (59.90)

AAWHHS=African American Women's Heart & Health Study

Table A2

Frequency (%) of racial discrimination (EOD scale) among AAWHHS participants ($n = 207$)

Experience of discrimination domain	Never	Once	2–3 times	4–5 times	≥6 times	≥1 experiences
At school	82 (39.61)	23 (11.11)	55 (26.57)	17 (8.21)	30 (14.49)	125 (60.39)
Getting hired or getting a job	73 (35.27)	33 (15.94)	52 (25.12)	22 (10.63)	27 (13.04)	134 (64.73)
At work	69 (33.33)	34 (16.43)	56 (27.05)	22 (10.63)	26 (12.56)	138 (66.67)

Experience of discrimination domain	Never	Once	2–3 times	4–5 times	≥6 times	≥1 experiences
Getting housing	102 (49.28)	23 (11.11)	40 (19.32)	14 (6.76)	28 (13.53)	105 (50.72)
Getting medical care	127 (61.35)	15 (7.25)	30 (14.49)	13 (6.28)	22 (10.63)	80 (38.65)
Getting credit, bank loans, or a mortgage	95 (45.89)	15 (7.25)	47 (22.71)	13 (6.28)	37 (17.87)	112 (54.11)
On the street or in a public setting	57 (27.54)	32 (15.46)	70 (33.82)	17 (8.21)	31 (14.98)	150 (72.46)
From the police or in the courts	69 (33.33)	45 (21.74)	44 (21.26)	22 (10.63)	27 (13.04)	138 (66.67)

AAWHHS = African American Women's Heart & Health Study.

Table A3

χ^2 test of homogeneity for qualitative-based reports of EDS and EOD among AAWHHS participants ($n = 207$)

Discrimination	EOD					
EDS	**None**	**Low**	**Moderate**	**High**	**Very High**	**Total**
None	19	31	8	1	0	59
Low	2	31	23	8	1	65
Moderate	0	6	19	8	5	38
High	1	2	9	10	4	26
Very high	0	1	4	2	12	19
Total	22	71	63	29	22	207

Pearson χ^2 (16) = 151.5538; $P = .000$.

AAWHHS=African American Women's Heart & Health Study.

Table A4

Bivariate analyses for exposure-to-exposure, outcome-to-outcome, and exposure-to-outcome differences and correlations among AAWHHS participants ($n = 207$)

Variable	Allostatic load		Self-reported health			Pearson's *r*	
Discrimination	**β 95% CI**	***P*-value**	**Good *n* (%)**	**Not Good *n* (%)**	***P*-value**	**AL**	**EDS**
EDS		.307			.400	−0.0127	—
None	−0.126 (−1.048, 0.796)		41 (26)	18 (35)			
Low	−0.839 (−1.738, 0.060)		52 (33)	13 (25)			
Moderate (ref)	—		30 (19)	8 (16)			
High	−0.585 (−1.725, 0.556)		21 (14)	5 (10)			
Very high	−0.595 (−1.841, 0.650)		12 (8)	7 (14)			
EOD		.040			.721	−0.0950	0.7430
None	0.729 (−0.362, 1.819)		16 (10)	6 (12)			
Low	−0.517 (−1.279, 0.244)		55 (35)	16 (31)			

Variable	Allostatic load		Self-reported health			Pearson's *r*	
Discrimination	**β 95% CI**	***P*-value**	**Good *n* (%)**	**Not Good *n* (%)**	***P*-value**	**AL**	**EDS**
Moderate (ref)	—		49 (31)	14 (27)			
High	−0.260 (−1.249, 0.728)		22 (14)	7 (14)			
Very High	−1.226 (−2.316, −0.135)		14 (9)	8 (16)			
Self-reported health	(μ)	.694					
Good (*n* = 156)	5.917 (5.566, 6.268)		—	—	—	—	—
Not good (*n* = 51)	6.098 (5.481, 6.715)		—	—	—	—	—

AAWHHS = African American Women's Heart & Health Study; β = beta coefficient; CI = confidence interval; SRH = self-reported health; μ = mean.

Table A5

Kappa tests for qualitative-based and distribution-based reports of EDS and EOD among AAWHHS participants (*n* = 207)

Discrimination	% Agreement	Kappa	*P*-value
EDS	0.00	−0.17	1.00
EOD	12.56	−0.08	1.00

AAWHHS = African American Women's Heart & Health Study.

Table A6

Sample distribution of at-risk biomarkers by medication (Med) use among AAWHHS participants (*n* = 207)

At-risk	Full (*n* = 207)	No Meds (*n* = 157)	Meds (*n* = 50)	*P*-value
System	***n* (%)**			
CV				
DBP	104 (50)	58 (37)	46 (92)	.001
SBP	112 (54)	66 (42)	46 (92)	.001
Inflammatory				
IL6	57 (28)	46 (29)	11 (22)	.314
CRP	103 (50)	72 (46)	31 (62)	.047
Neuroendocrine				
Cortisol	54 (26)	35 (22)	19 (38)	.028
Epi	51 (25)	40 (25)	11 (22)	.619
Norepi	51 (25)	36 (23)	16 (32)	.165
Metabolic				
HDL	87 (42)	61 (39)	19 (38)	.508
LDL	84 (41)	62 (40)	22 (44)	.572
Tri	15 (7)	8 (5)	5 (10)	.388
Cholesterol	138 (67)	106 (68)	33 (66)	.842
BMI	179 (86)	135 (86)	43 (86)	.911

At-risk System	Full (*n* = 207) *n* (%)	No Meds (*n* = 157)	Meds (*n* = 50)	*P*-value
Waist	151 (73)	109 (70)	41 (82)	.098
Glucose	40 (19)	23 (15)	17 (34)	.003
A1c	40 (19)	27 (17)	15 (30)	.028

AAWHHS = African American Women's Heart & Health Study; CV = cardiovascular.

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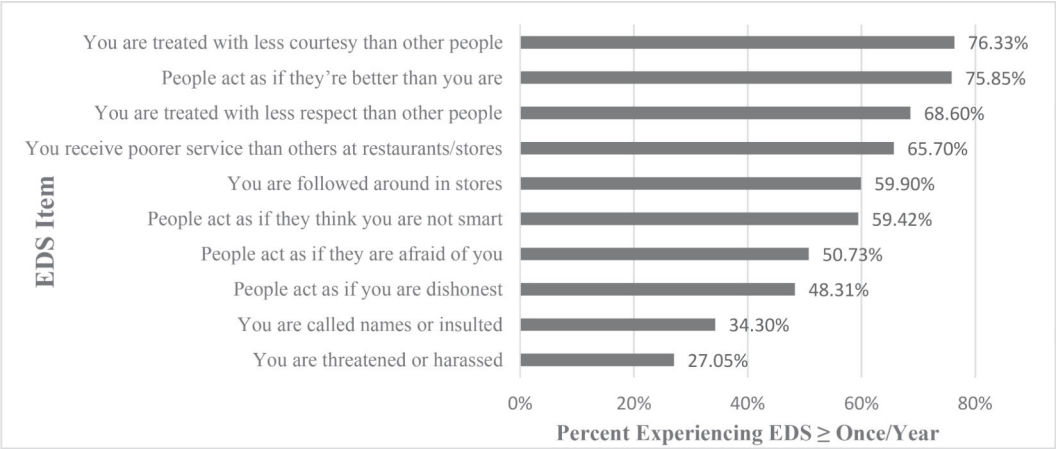

Fig. 1.

Percentage reporting EDS more than once per year by item (n = 207).

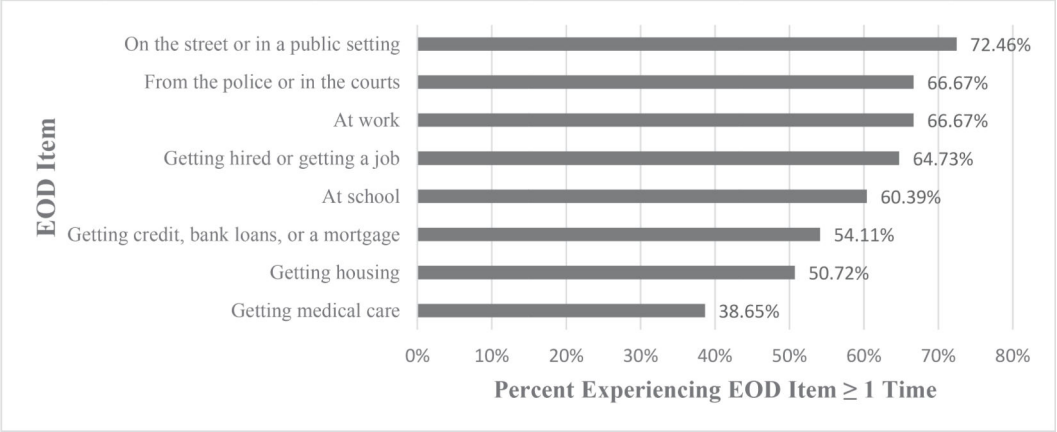

Fig. 2.
Percentage reporting EOD one or more times by item (n = 207).

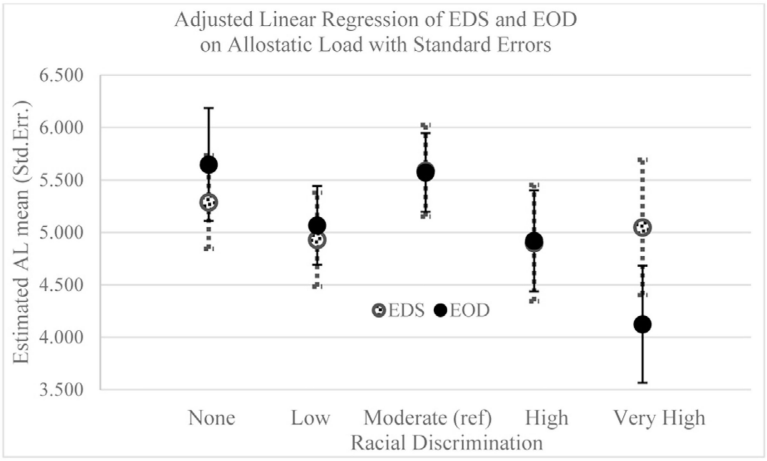

Fig. 3.

Linear regression of allostatic load by EDS and EOD measures adjusted for age, socioeconomic position, and medication use.

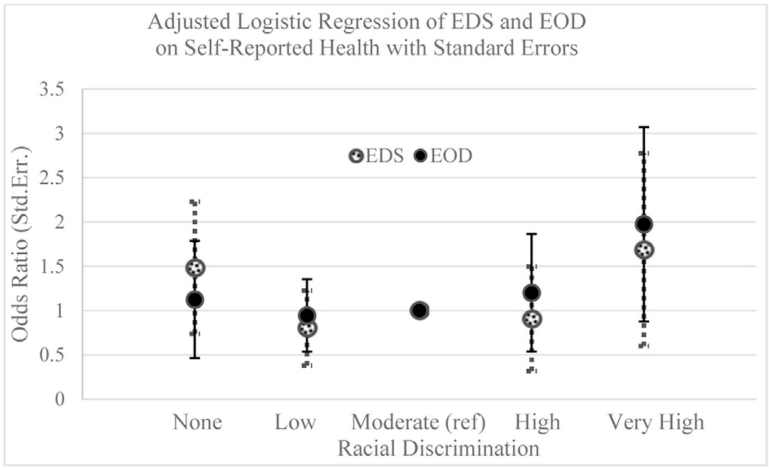

Fig. 4.

Logistic regression of self-reported health by EDS and by EOD measures adjusted for age, socioeconomic position, and medication use.

Table 1

Allostatic load biomarker cut-points

Biomarker	Guideline used	AL cut-points
Metabolic system		
HDL (mg/dL)	ATPIII	<50
LDL (mg/dL)	ATPIII	≥100
Waist (in)	ATPIII	>35
Glucose (mg/dL)	ATPIII	≥100 or <70
HbA1c (mmol/mol)	ADA	≥5.7
Total cholesterol (mg/dL)	ATPIII	≥160
Triglycerides (mg/dL)	ATPIII	≥150
BMI (kg/m^2)	ATPIII	≥25 or <18.5
Cardiovascular system		
Systolic BP (mm Hg)	AHA (JNC 7)	≥120
Diastolic BP (mm Hg)	AHA (JNC 7)	≥80
Neuroendocrine system		
*Epinephrine (pg/mL)	n/a	>77.70
*Norepinephrine (pg/mL)	n/a	>686.30
*Cortisol (μg/dL)	n/a	>12.69
Inflammatory system		
*Il-6 (pg/mL)	n/a	>7.85
hsCRP (mg/L)	AHA	>3

*75th-percentile cut-points used for biomarkers that do not have clinical guidelines; subclinical cut-points used for rest.

Table 2

Study sample characteristics (n = 207)

Covariates	*n*	%
Age mean (±SD.)	41.72 (5.90)	
SEP mean (±SD) (range 0–4)	1.67 (0.96)	
Educational attainment		
> High school diploma	138	66.67
≤ High school diploma	69	33.33
Employment status		
Employed	114	55.07
Not employed	93	44.93
Poverty status		
> 100% federal poverty	168	81.16
≤ 100% federal poverty	39	18.84
Marital/partnership status		
Married/domestic partnership	61	29.47
Not married/domestic partnership	146	70.53
Cardiovascular medication		
Not currently taking	164	79.23
Currently taking	43	20.77
Diabetes medication		
Not currently taking	195	94.20
Currently taking	12	5.80
Racial discrimination exposures	***n***	**%**
Everyday discrimination ccale (EDS)		
None (EDS score: less than/equal 20)	59	28.50
Low (EDS score: 21–30)	65	31.40
Moderate (EDS score: 31–40)	38	18.36
High (EDS score: 41–50)	26	12.56
Very high (EDS score: 51–60)	19	9.18
Experiences of discrimination (EOD)		
None (EOD score: 8)	22	10.63
Low (EOD score: 9–16)	71	34.30
Moderate (EOD score: 17–24)	63	30.43
High (EOD score: 25–32)	29	14.01
Very high (EOD score: 33–40)	22	10.63
Health outcomes		
Allostatic load (range 0–15)		
mean (±SD)		5.96 (2.24)
Self-reported overall physical health		
Excellent/very good/good	156	75.36

Fair/poor	51	24.64

SD = standard deviation; SEP = socioeconomic position.

Table 3

Linear regression of allostatic load and logistic regression of self-reported health by EDS and by EOD ($n = 207$)

Discrimination	Allostatic load		Self-reported physical health	
	EDS	EOD	EDS	EOD
	β[*] 95% CI	β[†] 95% CI	OR[‡] 95% CI	OR[§] 95% CI
None	−0.166 (1.044, 0.713)	0.216 (−0.847, 1.279)	1.483 (0.554, 3.973)	1.124 (0.354, 3.565)
Low	*−0.524* (−1.408, 0.360)	−0.364 (−1.104, 0.377)	0.804 (0.286, 2.256)	0.945 (0.403, 2.213)
Moderate (ref)	//5.454	//5.431	1.000	1.000
High	−0.556 (−1.651, 0.539)	−0.512 (−1.464, 0.441)	0.907 (0.253, 3.248)	1.200 (0.405, 3.552)
Very high	−0.407 (−1.682, 0.869)	**−1.307 (−2.411, −0.203)**	1.686 (0.476, 5.969)	1.974 (0.664, 5.865)

Models adjusted for age, socioeconomic position, and medication use. Bolded value represents $P < .05$.

β = beta coefficient; CI = confidence interval; OR = odds ratio.

[*](Power) = 1-β = 0.99 ($R^2 = 0.16$, α = 0.05).

[†]1-β = 0.99 ($R^2 = 0.18$, α = 0.05).

[‡]1-β = 0.80 ($r^2 = 0.07$, α = 0.10).

[§]1-β = 0.80 ($r^2 = 0.06$, α = 0.10).

//estimated mean allostatic load.