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Authors

Van Dyke, Miriam E
Baumhofer, Nicole Kau'i
Slopen, Natalie
[et al.](#)

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Pervasive Discrimination and Allostatic Load in African-American and White Adults

Miriam E. Van Dyke, MPH¹, N. Kau'i Baumhofer, ScD², Natalie Slopen, ScD³, Mahasin S. Mujahid, PhD⁴, Cheryl R. Clark, MD, ScD⁵, David R. Williams, PhD⁶, Tené T. Lewis, PhD^{1,a}

¹Department of Epidemiology, Rollins School of Public Health, Emory University, Atlanta, GA, USA

²Department of Native Hawaiian Health, John A. Burns School of Medicine, University of Hawaii at Manoa, Honolulu, HI, USA

³Department of Epidemiology and Biostatistics, School of Public Health, University of Maryland, College Park, MD, USA

⁴Division of Epidemiology, School of Public Health, University of California at Berkeley, Berkeley, CA, USA

⁵Brigham and Women's Hospital, Harvard Medical School, Harvard University, Boston, MA, USA

⁶Department of Social and Behavioral Sciences, Harvard T.H. Chan School of Public Health, Harvard University, Boston, MA, USA

Abstract

Objective—To examine associations among race, the accumulation of multiple forms of discriminatory experiences (i.e., “pervasive discrimination”) and allostatic load (AL) in African-Americans and Whites in mid-life.

Methods—Using data collected in 2004–2006 from 226 African-American and 978 White adults (57% female; mean age=54.7 years (SD=0.11)) in the Midlife in the United States II (MIDUS II) Biomarker Project, a pervasive discrimination score was created by combining three discrimination scales, and an AL score was created based on 24 biomarkers representing 7 physiological systems. Linear regression models were conducted to examine the association between pervasive discrimination and AL, adjusting for demographics and medical, behavioral, and personality covariates. A race by pervasive discrimination interaction was also examined in order to determine whether associations varied by race.

Results—African-Americans had higher pervasive discrimination and AL scores than Whites. In models adjusted for demographics, socioeconomic status, medications, health behaviors, neuroticism and negative affect, a pervasive discrimination score of 2 vs. 0 was associated with a greater AL score (b=0.30; SE=0.07, p<.001). While associations appeared to be stronger among African-Americans as compared to Whites, associations did not statistically differ by race.

^aCorresponding Author: Tené T. Lewis, PhD, 1518 Clifton Rd NE, Atlanta, GA 30322, Claudia Nance Rollins Building, 3rd Floor, Emory University, Rollins School of Public Health, tene.t.lewis@emory.edu.

Conclusions—More pervasive discrimination was related to greater multisystemic physiological dysregulation in a cohort of African-American and White adults. Measuring discrimination by combining multiple forms of discriminatory experiences may be important for studying the health effects of discrimination

Keywords

African-Americans; discrimination; allostatic load; social determinants of health; health disparities

INTRODUCTION

On almost every major indicator of poor health, African-Americans fare worse than their White counterparts. From diabetes mellitus (1, 2) and stroke (3) incidence and mortality to pre-term birth and infant mortality (4), as well as mortality from heart disease (5) and cancer (1), racial disparities have been documented. Although factors including socioeconomic status (SES) (6), access to and receipt of medical care (7), and behavioral risk factors (1) have been cited as contributors to these disparities, they do not fully account for health gaps between African-Americans and Whites (6).

Researchers have proposed that experiences of discrimination may be important to consider in studies designed to advance understanding of racial disparities in health (8, 9). Across cohorts, African-Americans report more discrimination than Whites (10, 11), and self-reported experiences of discrimination have been associated with outcomes such as breast cancer (12), incident cardiovascular disease (13), incident diabetes (14) and metabolic syndrome (15), asthma (16), and poor sleep (17–19). However, although African-Americans typically report discrimination for different reasons than Whites (e.g. “race/ethnicity” versus “sex/gender” or “appearance”) (20, 21), to date, studies have not consistently found evidence to support stronger associations among African-Americans (or other minority groups) compared to Whites (9, 13, 18, 22, 23); nor have studies consistently found that one attribute (e.g. race) more negatively affects outcomes than another (15, 17, 24–26).

Some have argued that the lack of observed racial differences in the association between discrimination and health is largely due to the possibility that interpersonal mistreatment impacts individuals similarly, irrespective of their racial/ethnic backgrounds (8). However, it could also potentially be due to the fact that the majority of prior studies have focused on discrimination assessed using a single scale. Both national and self-reported data indicate that relative to Whites, African-Americans are exposed to discrimination across a wider variety of situations and settings in life, including while shopping (27), at work (28) and when seeking housing (29), employment (30) and healthcare (31), as well as in interactions with the criminal justice system (32). For African-Americans in particular, the pervasiveness of discrimination in everyday life may be stressful, unavoidable and ultimately detrimental to health. This is consistent with the “weathering hypothesis,” which posits that cumulative exposure to chronic stressors such as discrimination in the context of overall racial disadvantage leads to accelerated physiological aging, or “weathering” among African-Americans, relative to their White counterparts (33, 34). However, the impact of pervasive

discrimination on health, and any potential racial differences in its association with outcomes, has been underexplored.

In the present study, we examined associations between self-reported “pervasive” discrimination—assessed by combining multiple scales that assess experiences of discrimination across a range of settings and situations-- and an indicator of impaired physiological functioning, allostatic load (AL), among African-Americans and Whites. We chose AL because it summarizes overall systemic dysregulation, i.e. physiological “wear and tear” on the body (35), and may be one pathway through which discrimination affects a variety of health outcomes. Prior studies have documented positive associations between self-reported discrimination measured via a single scale and AL (36–42); however experiences of discrimination assessed using a combination of multiple scales may better capture the level of pervasiveness by which it permeates the lives of certain groups. Additionally, to our knowledge, only one of these prior studies of discrimination and AL examined racial differences (39), but that study focused on weight discrimination specifically, which is actually underreported by African-Americans, relative to Whites (43). Consequently, we further examined whether the association between pervasive discrimination and AL was stronger among African-Americans than Whites, given the particularly ubiquitous nature of discrimination in the lives of African-Americans as compared to Whites and the potential role of weathering.

METHODS

Sample

The sample was drawn from the Midlife in the United States II (MIDUS II) Biomarker Project, a national assessment of long-term change in the relationships between sociodemographic and psychosocial variables with biological functioning among non-institutionalized adults living in the 48 contiguous states. The original MIDUS I participants, aged 25–74 (N=7,108), were interviewed using random digit dialing between 1995–1996 and were contacted again for MIDUS II between 2004–2006 (N=5,895). The response rate in the MIDUS II follow-up survey was 70%. A subset of 1,255 MIDUS II participants provided further information through a physical exam, medical history, medication regime, sleep assessment, laboratory challenge of physical functioning, and a comprehensive array of biomarkers during a 2-day clinic visit. A more detailed description of the procedures and methods in the MIDUS II Biomarker project has been previously published (44). The current study focused on data from the 2004–2006 study visit as it was the only study visit where information on both biomarkers and psychosocial factors was collected. Of the original 1,255 participants included in the MIDUS II Biomarkers Project, 51 were excluded from this analysis because they were not either White or African American. A total sample of 1,204 was used in this analysis and was based on 10 multiple imputed data sets. Multiple imputation was used to minimize bias from the missing data for a few variables (i.e., among variables with missing data, missingness ranged from <1% –8.5% of the sample).

Measures

Pervasive Discrimination—Pervasive discrimination was assessed using a combination of three discrimination scales: Everyday Discrimination Scale (45), Lifetime Discrimination Scale (20), and Workplace Discrimination Scale (46).

Everyday Discrimination and Lifetime Discrimination Scales: The introduction to the self-administered questions for both the Everyday and Lifetime Discrimination scales asked respondents “How many times in your life have you been discriminated against in each of the following ways because of such things as your race, ethnicity, sex, age, religion, physical appearance, sexual orientation, or other characteristics?” Participants were instructed to only report experiences due to discrimination and not experiences due to other reasons.

Everyday discrimination, defined as “chronic, routine, and relatively minor experiences of unfair treatment” was measured using a nine-item self-administered scale (Cronbach’s $\alpha=0.91$) (45). The scale asked participants: “In your day-to-day life, how often do any of the following things happen to you?” The following list included nine situations, such as having been treated with less courtesy than other people or having received poorer service than other people at restaurants or stores. Participants could respond if they “often,” “sometimes,” “rarely,” or “never” experienced these situations. The coded responses for each of the nine items were summed so that a higher score indicated a higher level of everyday discrimination.

Lifetime discrimination, defined as events that occurred over the life course, but had greater potential consequences for the individual’s socioeconomic position, was measured using an 11-item self-administered index (Cronbach’s $\alpha=0.85$) (20). Participants were asked if they had ever experienced major events such as being “discouraged by a teacher or advisor,” “not hired for a job,” “prevented from renting or buying a home in the neighborhood [they] wanted,” or “denied or provided inferior medical care.” Each time the respondent answered “yes” to one of the 11 questions a score of 1 was assigned. Scores were summed, with a higher score indicating a higher level of lifetime discrimination.

Workplace Discrimination Scale: Workplace discrimination, defined as job harassment and unfair treatment at work, was measured using a 6-item self-administered scale (Cronbach’s $\alpha = 0.79$) (46), with Likert-style items such as: “how often are you watched more closely than other workers?” or “how often has a co-worker with less experience and qualifications gotten promoted before you?” Participants who reported working in the last 10 years were asked to indicate whether they experienced one of the six scenarios “once a week or more,” “a few times a month,” “a few times a year,” “less than once a year,” or “never.” The scale was scored by summing the responses of each of the 6 items and coded so that a higher score indicated a higher level of workplace discrimination.

Pervasive Discrimination Score: The Everyday Discrimination Scale, Lifetime Discrimination Scale, and Workplace Discrimination Scale were moderately correlated among African-Americans (ρ range: 0.22–0.48) and Whites (ρ range: 0.21–0.42) (all $p<0.0001$). Because we were particularly interested in the unavoidable aspects of pervasive discrimination, we wanted to capture those who reported relatively high exposure to

discrimination across multiple contexts (rather than high in one context, but moderate in others). Consequently, we created a pervasive discrimination score by combining the three discrimination scales, using the following procedure. First, for each of the three scales, respondents were categorized into tertiles based on the sample distribution. Second, the pervasive discrimination score was computed as follows: 1) participants in the lowest tertile on all three discrimination scales were given a “0” for the pervasive discrimination score; 2) participants in the highest tertile for only one of the three discrimination scales were given a “1”; and 3) participants in the highest tertile for two or three of the discrimination scales were given a “2.” This last group was combined given the small number (8.4%) of participants in the highest tertile for all three of the scales. In supplemental analyses, we also examined each individual discrimination scale continuously in separate models.

Allostatic load

Using established criteria, an overall AL score was created from seven AL subscales based on a set of 24 biomarkers collected from individuals in the MIDUS II Biomarker Project. The inter-assay and intra-assay variation of the set of biomarkers ranged from 0.85% to 13.0% and from 0.8% to 7.9%, respectively (47). The 24 biomarkers were chosen to describe physiological dysregulation across multiple systems (47–50). The seven AL subscales created from these 24 biomarkers included: sympathetic system functioning (urine epinephrine and norepinephrine, both adjusted for urine creatinine), parasympathetic system functioning (heart rate variability measures), hypothalamic pituitary adrenal-axis functioning (urine cortisol adjusted for urine creatinine and blood DHEAS), inflammation (Interleukin-6, fibrinogen, C-reactive protein, E-selectin, and ICAM), cardiovascular system functioning (systolic blood pressure, pulse pressure, heart rate), glucose metabolism (HbA_{1c}, blood glucose, insulin resistance), and lipid metabolism (body mass index (BMI), waist to hip ratio, triglycerides, high-density lipoproteins and low-density lipoproteins). The distributions of each of the 24 biomarkers were split into risk quartiles. Values in the quartile with highest risk received a risk-score of 1 and all other values received a risk-score of 0. For each participant with valid measurements for at least half of the biomarkers in a given subscale, the risk-scores for each biomarker in the seven subscales were averaged to create a summary score. For example, risk-scores for systolic blood pressure, heart rate, and pulse pressure were averaged together to create a cardiovascular summary score. If an individual ranked in the highest quartile of risk for only one of these three biomarkers, the cardiovascular summary score was 0.33; if two of the three biomarkers fell within the highest quartile of risk, the cardiovascular summary score was 0.66; all three biomarkers within the highest quartile of risk would produce a cardiovascular summary score of 1.00. The seven subscale scores were summed to compute a final overall AL score that ranged between 0 and 7 for all participants with valid scores for at least six of the subscales (47–50).

Demographics

Age, sex, marital status, self-reported race, employment status, education, and total household income were chosen as demographic covariates based on previous literature (36–42). Age was determined by participant birth date. Sex was coded as male or female. Marital status was either currently married, formerly married, or never married. Race was non-

Hispanic White or non-Hispanic African-American. Employment status was coded as currently employed or not currently employed. SES was measured using education coded as a high school diploma or less, some college, and an Associate's or Bachelor's degree or more, and total household income was reported in dollars per year.

Covariates

Oral steroid, beta-blocker, SSRI (selective serotonin reuptake inhibitor), and cholesterol medications were self-reported and included as covariates. Based on previous literature (47), health behavior covariates included self-reported smoking status, past month alcohol usage, and physical activity. Current smoking was coded as either current smoker or not currently a smoker. Alcohol usage was split into three categories of past month usage: never or not in the past month, less than once per week, at least once per week in the past month. Physical activity was measured using a summary weighted score based on weekly frequency and intensity of household, leisure, and occupational physical activity (47). The scores ranged from 9 to a possible maximum of 54. To minimize bias that could potentially be linked to personality factors, neuroticism and negative affect were included as covariates. Neuroticism was measured using four self-report items (51). Negative affect was measured using six self-report Likert style items combined into a scale, taken from the Positive and Negative Affect Scale (52).

Statistical Analyses

All statistical analyses were performed using SAS 9.4 (Cary, NC). Descriptive statistics were utilized to summarize participant characteristics for the overall sample and by race. Linear regression analyses were conducted to examine associations between pervasive discrimination and overall AL. Models were sequentially adjusted for demographics (race, age, sex, marital status, and employment status), SES (educational attainment and total household income), medications (oral steroid, SSRI, beta-blocker, and cholesterol use), health behaviors (current smoking, physical activity score, past month alcohol use), and personality covariates (neuroticism and negative affect). We also ran sensitivity analyses to adjust for anti-hypertensives and diabetes medication. To be consistent with prior studies, these were not included in our primary models. Similarly, models were run with and without BMI as a covariate (only the lipid metabolism subscale included BMI). Sensitivity analyses with anti-hypertensives, diabetes medication, and BMI did not alter our findings, thus they were excluded from final models.

The interaction between race and pervasive discrimination was formally assessed in minimally and fully-adjusted non-stratified linear regression models by creating cross-product terms. We did not observe a significant interaction (p-values >.05); nonetheless, we ran race-stratified models in exploratory analyses. This was done for two reasons: 1) to obtain race-specific effect sizes of the association between pervasive discrimination and AL in our cohort, given our original hypothesis, and emerging consensus across fields for the importance of effect sizes over p-values;(53–55) as well as 2) conceptual arguments for the importance of stratifying by race in order to account for differential confounding of associations of interest *within* each racial group (56).

Following these primary and exploratory analyses, supplementary analyses were conducted to: 1) examine the association between pervasive discrimination and each of the seven AL subscales separately; 2) examine the association between each individual discrimination scale (modeled continuously) and overall AL; and 3) examine the associations between pervasive discrimination and AL using a continuous version of the pervasive discrimination score, in order to retain those individuals who scored in the moderate range across the three scales. The continuous version of the pervasive discrimination score was created by standardizing aggregated z-scores from the three individual discrimination scales. Our analyses of continuous pervasive discrimination z-scores were conducted in both the overall sample and then separately for African-Americans and Whites, in order to more effectively compare any observed results to those in our primary and exploratory analyses.

RESULTS

Participant Characteristics

Descriptive statistics of the analytic sample are presented in Table 1. African-Americans reported more pervasive discrimination than Whites ($p < 0.001$), with 42.7% of African-Americans receiving a score of 2 (i.e. reporting discrimination in the highest tertile for 2 or 3 of the 3 discrimination scales) relative to 20.1% of Whites. On average, African-Americans also had a higher AL score (mean=1.93, standard deviation (SD)=1.01) than Whites (mean=1.71, SD=1.05) ($p = .005$). Additionally, in comparison to Whites, African-Americans were younger, less likely to be male, less likely to be currently employed or married, and reported less education and lower total household incomes. Although African-Americans reported less alcohol use than their White counterparts, they were more likely to be current smokers than Whites.

Primary Analyses

Among the full sample, in models adjusted for demographics and SES only, a pervasive discrimination score of 1 as compared to 0 was not associated with AL ($b = 0.07$, standard error (SE) = 0.07, $p = 0.33$), although a score of 2 compared to 0 was associated with greater AL ($b = 0.35$, SE = 0.07, $p < .001$) (Table 2). These relationships were observed in models further adjusted for medications, health behaviors, and personality characteristics (pervasive discrimination score of 1 vs. 0 $b = 0.04$, SE = 0.06, $p = 0.50$; score of 2 vs. 0 $b = 0.30$, SE = 0.07, $p < .001$).

Exploratory Analyses

Table 3 presents exploratory, race-stratified models. In fully adjusted linear regression models among Whites, a pervasive discrimination score of 1 as compared to 0 was not significantly associated with AL ($b = 0.02$, SE = 0.07, $p = 0.74$), although a score of 2 compared to 0 was significantly associated with greater AL ($b = 0.29$, SE = 0.09, $p < .001$). Similarly, among African-Americans in fully-adjusted models, a pervasive discrimination score of 1 as compared to 0 was not significantly associated with AL ($b = 0.26$, SE = 0.17, $p = 0.12$), although a score of 2 compared to 0 was significantly associated with greater AL ($b = 0.44$, SE = 0.15, $p = 0.004$). The race by pervasive discrimination interaction was not statistically significant ($p = .18$ for 0 vs 1 and $p = .19$ for 0 vs 2), although the effect size

among African-Americans comparing a score of 2 to 0 was 52% larger than that among Whites. Figure 1 displays the race-specific least square means for African-Americans and Whites separately, based on the fully-adjusted models. In addition to the magnitude of the association between pervasive discrimination and AL appearing larger among African-Americans as compared to Whites, the association between reports of pervasive discrimination seemed to be dose-response in nature among African-Americans, but not Whites. Of note, within group associations also differed. SES variables appeared to strengthen the association between pervasive discrimination and AL for African-Americans, but not for Whites (Model 2, Table 3). In fully-adjusted models, among African-Americans only, sex (female), SSRI use and negative affect were also significantly associated with a higher AL score; whereas among Whites, alcohol and beta-blocker use were significantly associated with a higher AL score.

Supplementary Analyses

In supplementary analyses, we examined associations between pervasive discrimination and each of the seven AL subscales (Table S1, Supplemental Digital Content), to document whether pervasive discrimination was associated with each of the individual system subscales included in the overall AL score. In the full cohort, positive associations were observed for glucose metabolism, lipid metabolism, and inflammation summary scores.

Supplementary analyses were also conducted examining associations between each individual discrimination scale modeled continuously and overall AL. Among the full sample in minimally and fully-adjusted models, Everyday Discrimination and Lifetime Discrimination (but not Workplace Discrimination) were associated with the AL score (Fully adjusted models: Everyday Discrimination: $b=0.02$, $SE=0.01$, $p<.001$; Lifetime Discrimination: $b=0.05$, 0.02 , $p=0.001$; Workplace Discrimination: $b=0.01$, $SE=0.01$, $p=0.27$).

Greater pervasive discrimination, in the form of a continuous score created by standardizing aggregated z-scores from the three individual discrimination scales, was also associated with a higher AL score in the full sample in minimally- and fully-adjusted models ($b=0.14$, $SE=0.03$, $p<.001$; $b=0.12$, $SE=0.03$, $p=0.001$; Table S2, Supplemental Digital Content). In fully-adjusted race-stratified models, associations were observed among both African-Americans ($b=0.13$, $SE=0.05$, $p<.001$), and Whites ($b=0.12$, $SE=0.04$, $p=.003$), and the effect sizes appeared to be comparable. There were also no significant race by pervasive discrimination interactions using the continuous pervasive discrimination score.

DISCUSSION

In this cohort of African-American and White adults, reporting higher levels of pervasive discrimination—i.e. the experience of multiple forms of discriminatory experiences— was more common among African-Americans compared to Whites. Among both African-Americans and Whites, reports of pervasive discrimination were independently associated with greater overall AL after adjusting for sociodemographics, health behaviors and psychosocial risk factors. Findings from this study are a contribution to a literature that includes studies that have simultaneously examined multiple scales of discrimination (11,

13, 57), but have done so by examining these scales individually, thereby potentially not fully realizing the effects of the pervasiveness of discrimination for some groups. Additionally, because few studies have examined racial differences in the association between overall discrimination (pervasive or individual scales) and AL, these analyses further add to our understanding of whether and how various forms of discrimination might be contextualized by race to contribute to adverse health outcomes.

Although we did not observe a statistically significant race by pervasive discrimination interaction, the magnitude of the pervasive discrimination and AL association in our exploratory race-stratified analyses appeared to be stronger and had more of a dose-response pattern among African-Americans in comparison to Whites. Although not conclusive, the race-specific findings in this exploratory analysis suggests that the measurement of discrimination using a combination of multiple scales instead of a single scale could be particularly important for studying the health effects of discrimination *within* specific racial groups. This could be one potential reason for why prior studies that focused on a single scale of discrimination did not observe more pronounced, or in some instances any, associations between discrimination and health among African-Americans (23, 58, 59).

However, it is important to note that the apparently stronger within-group findings for African-Americans were only observed with the pervasive discrimination score that compared relatively high levels of discrimination across scales to relatively low levels. Our supplemental analyses that focused on continuous scores found similar within group associations for both African-Americans and Whites. This suggests that experiencing discrimination past a “threshold” or at a relatively higher level than others across multiple domains in life may be particularly detrimental for health among African-Americans as compared to Whites. It is possible that the qualitative experience of pervasive discrimination is different for these two groups, such that the consequences of experiencing such discrimination may be more severe for African-Americans than Whites. For example, a primary stressor related to discrimination (i.e., discrimination at work, school, or when seeking housing, etc.) could give rise to other types of secondary stressors (i.e., financial strain, relational strain, etc.) which African-Americans may have fewer additional resources to manage (60, 61). Further, it is plausible that African-Americans in particular who experience high levels of pervasive discrimination develop heightened levels of vigilance or anticipatory stress (62, 63) around discrimination which may result in differential health effects by race.

In the current study, both neuroticism and negative affect were included as covariates in models. Yet, associations between pervasive discrimination and AL among both African-Americans and Whites in our cohort persisted after adjusting for these factors. Thus, our findings were independent of two important psychosocial risk factors that may be important to consider in the relationship between self-reported experiences of discrimination and health (8). However, there may be other personality characteristics not included in this study with known linkages to discrimination, such as hostility, that should be considered in future research (64).

In supplementary analyses, we examined the relationship between pervasive discrimination and the individual system subscales included in the overall AL score to gain additional insight into whether certain indicators of impaired physiological functioning were more strongly associated with our measure of pervasive discrimination (65). In our study, pervasive discrimination was positively associated with the glucose metabolism and the lipid metabolism scores. These findings are in line with another study using the MIDUS cohort which found that weight discrimination was associated with the dysregulation of lipid and glucose metabolism (39). In addition to these system subscales, pervasive discrimination was also positively associated with the inflammation summary score. Additional research is needed to understand the mechanisms through which pervasive discrimination negatively affects physiological systems.

There are limitations to consider in the interpretation of this study. First, we had a relatively small number of African-Americans compared to Whites in our cohort, which may have limited our power to detect a race by pervasive discrimination interaction. The results of our exploratory analyses are suggestive of the possibility of an association among African-Americans that could potentially be stronger than that observed in Whites. However, additional research in cohorts with larger numbers of African-Americans is needed to draw definitive conclusions. Second, Whites in our cohort were of higher SES than Whites nationally, and the African-Americans in our cohort were primarily from a community-based sample of African-Americans residing in one of the most highly segregated cities in the United States. Thus, our data are not completely generalizable. However, while the African American sample is not representative, it nonetheless captures the experience of a large segment of African-Americans who reside in urban, economically disadvantaged communities. Future studies should examine the relationship between pervasive discrimination and AL among other racial/ethnic groups in varying geographic areas. Third, the cross-sectional nature of our study limits the ability of causal inference of the effect of pervasive discrimination on AL. Fourth, our overall AL score included BMI, which might temporally precede the development of many physiological disorders captured by AL. However, BMI was only included in the lipid metabolism subscale, and we observed associations with two additional subscales (inflammation and glucose metabolism). This suggests that BMI alone is not the primary driver of our associations. Fifth, the three scales used to assess pervasive discrimination were measured by self-report questionnaires, which could be subject to multiple forms of bias (i.e., recall and reporting bias). Finally, because these data were collected in the mid-2000's, it is unclear whether our findings would generalize to populations from older or more recent cohorts.

Despite these limitations, this study has a number of strengths. This study, which combined multiple scales to assess the pervasive nature of discrimination, adds to a literature that has primarily focused on capturing experiences of discrimination using a single scale; and while this study relied on self-report questionnaires to assess discrimination, this methodology is the most widely used. Moreover, findings in this study were robust to important personality confounders, including negative affect and neuroticism. Lastly, the examination of AL and its individual system components as health outcomes provides an opportunity to examine the effects of pervasive discrimination using a multi-system approach which recognizes that the

cumulative toll of the adaptation to stressful life experiences (i.e., discrimination) may manifest in several physiological systems (35).

In conclusion, to our knowledge, this is the first study to examine the association between pervasive discrimination-- measured by combining multiple scales that assess experiences of discrimination across a range of settings and situations-- and AL. In this sample of African-American and White adults, more pervasive discrimination was associated with greater multisystemic physiological dysregulation in both African-Americans and Whites. Future research on the measurement of pervasive discrimination and its effects on health is warranted.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Conflicts of Interest and Source of Funding:

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Abbreviations

AL	allostatic load
MIDUS II	Midlife in the United States II
SSRI	selective serotonin reuptake inhibitor
BMI	body mass index

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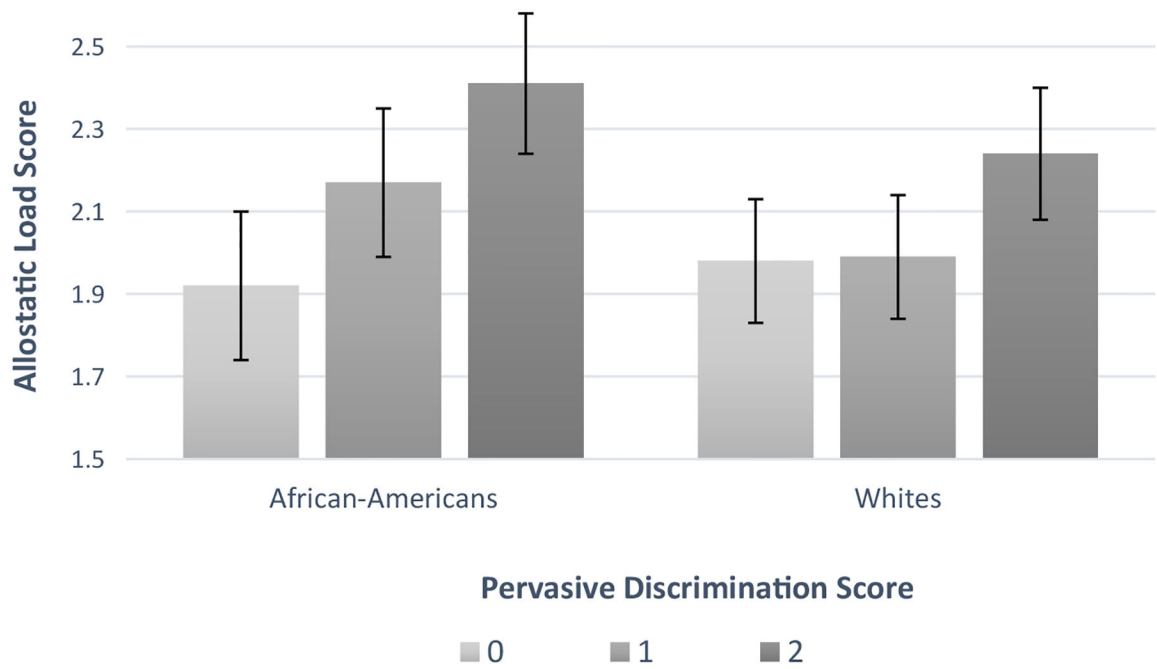


Figure 1. Associations between pervasive discrimination and allostatic load by race in the MIDUS II Biomarker Project (n=1,204)
MIDUS=Midlife in the United States; Values are estimated marginal means from linear regression models adjusted for demographics (age, sex, marital status, and employment status), socioeconomic status (educational attainment and total household income), medications (oral steroid, SSRI, beta-blocker, and cholesterol use), health behaviors (current smoking, physical activity score, past month alcohol use), and personality covariates (neuroticism and negative affect). Error bars represent standard errors. Sample based on 10 multiple imputed data sets.

Table 1. Characteristics of African-American and White adults in the MIDUS II Biomarker Project (n=1,204)

	All (1,204)	African-Americans (n=226)	Whites (n=978)	p-value
		% or mean (SD)	% or mean (SD)	
Age (years)	54.68 (11.77)	50.90 (10.59)	55.54 (11.87)	<.001
Male (%)	43.02	32.30	45.50	<.001
Marriage Status (%)				
Currently married	64.83	28.32	73.27	
Formerly married	22.96	39.38	19.16	<.001
Never married	12.21	32.30	7.57	
Currently Employed (%)	70.62	62.39	72.53	.003
Education (%)				
HS or less	28.07	46.90	23.72	
Some College	22.48	27.88	21.24	<.001
At least Associates/Bachelors	49.44	25.22	55.04	
Total Household Income	69960.70 (58260.01)	38192.50 (34567.70)	77301.80 (60129.00)	<.001
Pervasive Discrimination Score (%)				
0	44.18	30.75	47.28	
1	31.51	26.59	32.65	<.001
2 or 3	24.31	42.65	20.07	
Allostatic Load Score	1.75 (1.05)	1.93 (1.01)	1.71 (1.05)	.005
Neuroticism ¹	2.03 (0.63)	2.10 (0.69)	2.02 (0.62)	.11
Negative affect ²	1.53 (0.62)	1.77 (0.83)	1.48 (0.54)	<.001
Oral Steroid Use (%)	1.16	2.21	0.92	<.001
Beta Blocker Use (%)	14.53	15.04	14.42	.45
SSRI Use (%)	8.14	2.65	9.41	<.001
Cholesterol Med Use (%)	28.41	21.24	30.06	<.001
Current Smoker (%)	13.54	27.88	10.22	<.001
Alcohol Use (%)				

	All (1,204)	African-Americans (n=226) % or mean (SD)	Whites (n=978) % or mean (SD)	p-value
No alcohol in past month	34.14	44.48	31.78	
<Once per week in past month	31.15	27.57	31.98	.002
Once per week in past month	34.70	28.05	36.24	
Physical Activity Score³	29.72 (10.90)	29.72 (12.65)	29.72 (10.45)	.98
Body Mass Index	29.78 (6.62)	32.80 (8.33)	29.08 (5.94)	<.001

Percentages or mean (SD=standard deviation) presented.

P-values determined from chi-square and t-tests across racial groups.

¹ Out of a possible 4 points

² Out of a possible 6 points

³ Weighted score based on 3 questions (range: 9 to 54).

SSRI= selective serotonin reuptake inhibitor; MIDUS=Midlife in the United States.

Table 2. Associations between pervasive discrimination and allostatic load in the MIDUS II Biomarker Project (n=1,204)

	Model 1 ^a		Model 2 ^b		Model 3 ^c		Model 4 ^d		Model 5 ^e	
	Beta (SE)	p	Beta (SE)	p	Beta (SE)	p	Beta (SE)	p	Beta (SE)	p
0 (ref)	--		--		--		--		--	
1	0.07 (0.07)	0.33	0.06 (0.07)	0.32	0.07 (0.07)	0.28	0.05 (0.06)	0.44	0.04 (0.06)	0.50
2	0.35 (0.07)	<.001	0.34 (0.07)	<.001	0.33 (0.07)	<.001	0.33 (0.07)	<.001	0.30 (0.07)	<.001

SE= standard error; MIDUS=Midlife in the United States.

^aAdjusted for demographics (race, age, sex, marital status, employment status)

^bModel 1 + socioeconomic status (education, total household income)

^cModel 2 + medications (oral steroid, beta-blocker, selective serotonin reuptake inhibitor, cholesterol)

^dModel 3 + health behaviors covariates (current smoker, physical activity score, past month alcohol usage)

^eModel 4 + psychosocial characteristics (neuroticism, negative affect)

Table 3.

Associations between pervasive discrimination and allostatic load by race in the MIDUS II Biomarker Project

	Model 1 ^a		Model 2 ^b		Model 3 ^c		Model 4 ^d		Model 5 ^e	
	Beta (SE)	p	Beta (SE)	p	Beta (SE)	p	Beta (SE)	p	Beta (SE)	p
African-Americans (n=226)										
0 (ref)	--	--	--	--	--	--	--	--	--	--
1	0.19 (0.17)	0.26	0.26 (0.17)	0.12	0.29 (0.17)	0.08	0.25 (0.17)	0.14	0.26 (0.17)	0.12
2	0.41 (0.16)	0.01	0.52 (0.16)	0.002	0.48 (0.16)	0.002	0.46 (0.16)	0.003	0.44 (0.15)	0.004
Whites (n=978)										
0 (ref)	--	--	--	--	--	--	--	--	--	--
1	0.04 (0.07)	0.56	0.03 (0.07)	0.62	0.04 (0.07)	0.57	0.03 (0.07)	0.72	0.02 (0.07)	0.74
2	0.33 (0.09)	<.001	0.31 (0.09)	<.001	0.30 (0.09)	<.001	0.30 (0.09)	<.001	0.29 (0.09)	<.001

SE= standard error; MIDUS=Midlife in the United States.

^a Adjusted for demographics (age, sex, marital status, employment status)

^b Model 1 + socioeconomic status (education, total household income)

^c Model 2 + medications (oral steroid, beta-blocker, selective serotonin reuptake inhibitor, cholesterol)

^d Model 3 + health behaviors covariates (current smoker, physical activity score, past month alcohol usage)

^e Model 4 + psychosocial characteristics (neuroticism, negative affect)