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Allostatic Load Burden and Racial Disparities in Mortality

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Abstract

Background—Black-white disparities in mortality persist after adjustment for socioeconomic status and health behaviors. We examined whether allostatic load, the physiological profile influenced by repeated or chronic life stressors, is associated with black-white mortality disparities independent of traditional sociobehavioral risk factors.

Methods—We studied 4515 blacks and whites aged 35 to 64 years from the third National Health and Nutrition Examination Survey (1988–1994), using the linked mortality file, to ascertain participant deaths through 2006. We estimated unadjusted sex-specific black-white disparities in cardiovascular/diabetes-related mortality and noninjury mortality. We constructed baseline allostatic load scores based on 10 biomarkers and examined attenuation of mortality disparities in 4 sets of sex-stratified multivariate models, sequentially adding risk factors: (1) age/clinical conditions, (2) socioeconomic status (SES) variables, (3) health behaviors, and (4) allostatic load.

Results—Blacks had higher allostatic load scores than whites; for men, 2.5 vs 2.1, $p < .01$; and women, 2.6 vs 1.9, $p < .01$. For cardiovascular/diabetes-related mortality among women, the magnitude of the disparity after adjustment for other risk factors (hazard ratio [HR], 1.63; 95% confidence interval [CI], 0.96–2.75) decreased after adjustment for allostatic load (HR, 1.15; 95% CI, 0.70–1.88). For noninjury mortality among women, the magnitude of the disparity after adjustment for other risk factors (HR, 1.43; 95% CI, 1.00–2.04) also decreased after adjustment for allostatic load (HR, 1.26; 95% CI, 0.90–1.78). For men, disparities were attenuated but persisted after adjustment for allostatic load.

Conclusions—Allostatic load burden partially explains higher mortality among blacks, independent of SES and health behaviors. These findings underscore the importance of chronic physiologic stressors as a negative influence on the health and lifespan of blacks in the United States.

Keywords

stress; mortality; African Americans

Non-Hispanic blacks in the United States suffer increased all-cause, noninjury, and cardiovascular-related mortality rates compared to non-Hispanic whites.[1–4] These black-white disparities in mortality are attributed to several chronic conditions among both men and women, increase progressively from age 20 through 64 years, and decline but remain

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present through age 85 years.[3] Racial differences in life expectancy are typically somewhat attenuated but persist after statistical adjustment for demographic factors, including indicators of socioeconomic status (SES) and health insurance.[2,5,6]

The concept of *allostatic load*, which refers to the accumulation of physiological perturbations as a result of repeated or chronic stressors in daily life,[7–9] could partially explain the residual black-white disparity in mortality rates. The specific measurement of allostatic load varies between research studies, but it has generally included levels of hormones secreted in response to stress (primary, direct mediators) and/or biomarkers that reflect the effects of these hormones on the body (secondary, indirect mediators).[10] This stress may accumulate from early life through the working years and manifest as cumulative physiologic dysregulation, leading to an eventual increase in allostatic load as well as an increase in premature morbidity and mortality from chronic diseases.[11]

Allostatic load differences by race may explain why black-white disparities in mortality are observed even among Americans with high SES.[5] Although the burden of allostatic load is greater overall among patients of low SES, Geronimus and colleagues found that black-white differences in allostatic load are larger for nonpoor vs poor individuals, particularly among women.[12,13] Chronic stressors such as food insecurity, living in substandard housing, inadequate access to health care, and greater exposure to violence are greater among persons with low SES, regardless of race. However, both poor and nonpoor blacks may share other stressors not generally experienced by whites, such as interactions with institutionalized racism, which could lead to increased allostatic load.[13–15]

Using longitudinal data from the National Health and Nutrition Examination Survey (NHANES III) linked mortality file, we investigated whether allostatic load at baseline was associated with racial differences in subsequent mortality rates among middle-aged adults. We hypothesized that after adjusting for SES measures, health insurance status, and health behaviors, further adjustment for a 10-component secondary measure of allostatic load would substantially reduce the magnitude of subsequent black-white mortality disparities.

METHODS

Survey Design and Data Collection

The NHANES is conducted by the National Center for Health Statistics, using a stratified multistage probability design to obtain a representative sample of the civilian, noninstitutionalized US population. Details on the sampling strategy and weighting methods are available in electronic form.[16] The NHANES includes household interviews that collect sociodemographic and clinical information; standardized physical examinations, including height, weight, and blood pressure; and collection of blood samples in special mobile examination centers. As the NHANES data are publicly available and subjects can never be identified, these analyses are not considered human subjects research and are exempt from institutional board review at the institutions of the coauthors.

We used data from NHANES III (1988–1994), which included a sample of approximately 40 000 persons from 89 randomly selected locations throughout the United States. Using a longitudinal study design, we examined the association between allostatic load at baseline and subsequent mortality as well as black-white disparities in mortality rates. For this analysis, we included participants aged 35 to 64 years at the time of interview who self-identified as either non-Hispanic black or non-Hispanic white (n = 5478). Pregnant women and patients who were interviewed but not examined were excluded. We focused on the age range of 35 to 64 years in order to include participants who were old enough to have

developed physiologic dysregulation yet had variable health insurance coverage (ie, not yet eligible for Medicare).

Variable Definitions

Based on prior literature, we constructed a summed allostatic load score based on values for 10 secondary biomarkers, available in the NHANES data set, that represent physiologic dysregulation.[13,17] These biomarkers included metabolic markers (waist to hip ratio, glycated hemoglobin), cardiovascular markers (systolic blood pressure, diastolic blood pressure, total cholesterol, triglycerides, homocysteine), inflammatory markers (albumin, C-reactive protein), and a marker of organ dysfunction (estimated glomerular filtration rate [eGFR]). These 10 biomarkers are not a comprehensive measure of physiologic regulation and are unlikely to capture alterations in immune function, inflammatory responses, or in neuroendocrine systems such as the hypothalamic-pituitary axis.

For each biomarker in our study, we stratified the sample by gender and identified the participants with values in the highest-risk quartile (<25th percentile for eGFR and albumin, >75th percentile for all others). Participants received 1 point toward their allostatic load score for each value in the highest-risk quartile, with a maximum score of 10. As there is little difference in the predictive ability of simple count scores compared to more complex weighted measures, we used the former approach for ease of interpretation.[10] We excluded participants who were missing data for 2 or more components of the score (n = 963). However, we imputed data for participants missing only a single value (n = 2504), based on the mean value for their age, gender, and race.

We used the NHANES III Linked Mortality File to calculate race-specific death rates for NHANES III participants through 2006, up to 18 years later. Since we hypothesized that elevated allostatic load would lead to increased mortality through biologic mechanisms, we specifically focused on noninjury mortality, excluding accidents, suicide, and homicide. We also examined cardiovascular- and diabetes-related mortality, combining deaths from heart disease, cardiovascular disease, and diabetes, in a separate analysis. In the analysis examining cardiovascular- and diabetes-related mortality, we controlled for multiple self-reported noncardiovascular comorbidities, including chronic obstructive pulmonary disease (COPD), cancers (other than skin cancer), thyroid disease, rheumatoid arthritis, systemic lupus erythematosus, and asthma. We controlled for additional covariates in both sets of models, including education (<9 years, 9–12 years, >12 years), health insurance (yes/no), and poverty to income ratio (PIR) at the time of NHANES III. *PIR* is an income-to-needs variable measuring the ratio of household income to the US poverty threshold for each respondent's family size and composition. We also controlled for health behaviors, namely the Healthy Eating Index,[18] which is scored from 0 to 100,smoking status (current, former, never), physical activity (any vs none), and alcohol use (nondrinker, 1–30 drinks/month, >30 drinks/month).

Statistical Analyses

We calculated allostatic load scores by gender and race. We also calculated mean values for the demographic and clinical covariates of interest for black men, black women, white men, and white women. All estimates were weighted to adjust for the differential probabilities of sampling and nonresponse to represent the total civilian, noninstitutionalized US population. Estimates derived from a sample size smaller than the recommended lower limit in the NHANES analytic guidelines were considered unreliable.[16]

We constructed several sets of logistic regressions, comparing the black-white risks of noninjury and cardiovascular- and diabetes-related mortality separately for men and for

women. The regressions comparing noninjury mortality included 4 models, the first adjusting for age; the second, adding education, PIR, and health insurance status; the third, adding health behaviors; and the fourth, adding allostatic load score. The regressions comparing cardiovascular- and diabetes-related mortality also included 4 models: first, adjusting for age together with several noncardiac clinical conditions and then adding education, PIR, health insurance status, health behaviors, and allostatic load. Of note, because of low disease prevalence, we did not include systemic lupus erythematosus in the regression models predicting cardiovascular-and diabetes-related mortality among men. Results are expressed as hazard ratios (HRs) with 95% confidence intervals (CIs). All analyses were performed with the use of SUDAAN (Research Triangle Park, North Carolina), a statistical package that adjusts all estimates for the complex NHANES survey design. Because the observations contributed by each participant in the sample are weighted for the differential probabilities of selection and nonresponse, actual sample sizes are not reported along with percentages.

In order to assess whether medication use affected our results by lowering blood pressure and/or cholesterol levels, in sensitivity analyses, we assigned 1 point toward the allostatic load score if a participant was taking antihypertensives but had well-controlled blood pressure below the 75th percentile threshold. We also assigned 1 point toward the allostatic load score if a participant was taking cholesterol-lowering medications but had a total cholesterol value lower than the 75th percentile threshold. As the results from these sensitivity analyses did not appreciably alter our findings, we report only the results from the main analyses.

RESULTS

The final analytic sample included 4515 NHANES participants between 35 and 64 years of age. Within the included sample, black men and women had fewer years of education, were less likely to have health insurance, and were more likely to have a high PIR compared to white men and women (Table 1). Black men and women also had higher mean allostatic load scores compared to white men (2.5 vs 2.1, $p < .01$) and women (2.6 vs 1.9, $p < .01$).

As expected, we observed statistically significant black-white differences in cardiovascular- and diabetes-related and noninjury mortality among both women and men (Tables 2 and 3). The disparity in cardiovascular- and diabetes-related disease mortality for women (HR, 2.00; 95% CI, 1.31–3.06) was slightly less than the corresponding disparity for men (HR, 2.24; 95% CI, 1.59–3.16) after adjustment for age and comorbid conditions. As shown in Table 2, after sequential adjustment for baseline education and poverty status and health behaviors, the mortality disparity for women was somewhat attenuated and no longer statistically significant (HR, 1.63; 95% CI, 0.96–2.75). The magnitude of the disparity declined much further after adjustment for allostatic load (HR, 1.15; 95% CI, 0.70–1.88). The disparity in men was also somewhat attenuated but persisted after adjustment (HR, 1.55; 95% CI, 1.04–2.32).

We observed a similar pattern for noninjury mortality. As shown in Table 3, after adjustment for both baseline education and poverty status variables and baseline allostatic load scores, the black-white disparity in mortality at follow-up for women was attenuated and no longer statistically significant (HR, 1.26; 95% CI, 0.90–1.78), while the mortality disparity for men remained marginally significant (HR, 1.39; 95% CI, 1.00–1.92).

In both sexes, each 1-point increase in allostatic load score was associated with increased mortality at follow-up in all models, ranging from an HR of 1.22 (95% CI, 1.13–1.30) for

noninjury mortality among men, to an HR of 1.65 (95% CI, 1.44–1.90) for cardiovascular- and diabetes-related mortality among women (Tables 2 and 3).

DISCUSSION

Our findings indicate that baseline racial differences in indicators of physiologic dysregulation, as measured by secondary markers of allostatic load, help to explain black-white disparities in mortality among middle-aged adults followed up to 18 years later. This effect is additive to that of health behaviors and basic measures of SES, including education, poverty, and health insurance status. Our work expands on prior studies that linked allostatic load with mortality among adults 70 years of age and older[12,13,19] and raises the possibility that decreasing allostatic load burdens among black persons earlier in life may reduce racial disparities in mortality—particularly cardiovascular- and diabetes-related mortality—in later years.

The relations among stress, increased allostatic load, and disease are multifactorial and complex. McEwen conceptualizes the development of allostatic load as the relationship of an individual to their particular environmental stressors, which is modified by person-level differences (genetic variation, life experiences), different perception of environmental stressors, and different behavioral responses (including variation in both positive health behaviors such as physical exercise as well as negative health behaviors such as tobacco use) to these environmental stressors.[8] Elevated allostatic load and organ dysfunction can result from more frequent environmental stressors, an inability to adapt to constant or repeated stressors over time, and both anticipation of stressors (eg, worry about a stressful event in the future that may or may not take place) and memories of stressors that took place in the past (eg, posttraumatic stress disorder).[8] Cohen and colleagues describe a similar mechanism—namely, that stress in the environment results in negative emotional states and psychological distress as well as the adoption of unhealthy behaviors as a coping mechanism. These psychological and behavioral responses ultimately result in long-term physiologic changes that increase allostatic load and lead to organ dysfunction.[20] Recent empirical evidence is supportive of these concepts, showing that environmental stressors, particularly financial strain and relationship stressors, are more common among blacks as compared to whites and are also strongly linked to poor health.[21]

While racial differences in allostatic load may be influenced to some extent by genetic differences between racially designated groups, this is unlikely to be the sole or predominant explanatory factor for observed black/ white disparities in the United States. Adults in sub-Saharan Africa have much lower rates of hypertension, diabetes, and obesity than do blacks in the United States.[22–24] All of these conditions are likely to involve many genes, each of which has multiple possible variants. In addition, genomic studies indicate that as few as 3 to 5 common haplotypes include the bulk of allelic variation at any specific location in the genome. These haplotypes are well represented in the populations of all continents, so any specific “susceptibility” alleles that exist must be shared across all racial groups.[24]

Of note, interactions between genes and the environment may still contribute to black-white disparities, if blacks and whites have the same high-risk genes but have varying levels of environmental exposures that differentially affect expression of these genes. The National Institutes of Health recently funded an Epigenomics Program to conduct research on the influence of gene promoters, gene suppressors, and other key determinants of gene expression.[25] Ongoing work in this area may provide interesting and important evidence supporting the concept that differing patterns of social exposures produce changes in gene regulators that ultimately contribute to racial disparities in health, as described by Williams et al.[26]

In addition to person-level genetic variation and different behavioral responses to environmental stressors, psychological stressors that disproportionately affect blacks may help explain racial differences in allostatic load as well as racial differences in mortality. As an example, perceived racial discrimination as experienced in interpersonal interactions or as a result of institutional racism could potentially result in elevation of primary (eg, cortisol) and secondary (eg, systolic blood pressure) biomarkers in this population, leading in turn to subclinical disease, overt disease, and, ultimately, death from a variety of conditions. Furthermore, internalized racism and the acceptance of negative societal beliefs about oneself may lead to similar outcomes.[27]

Several recently published studies have examined the association between perceived racism and individual biomarkers among blacks, with the majority finding a positive link. This literature includes both overall and health care–specific perceptions of race-specific discrimination, which have been linked to higher systolic and diastolic blood pressure and glycated hemoglobin, as well as to an increase over time in waist to hip ratio (or waist circumference).[27–33] Each of these physiologic measures independently predicts mortality and is included in the allostatic load score operationalized in our study. Additional studies examining the link between perceived racism and other inflammatory/organ dysfunction markers (eg, albumin, eGFR), would provide further information about the possible relationship between race-related stress and other biomarkers that predict morbidity and mortality.

The concept of allostatic load as a lifelong cumulative measure of physiologic dysfunction resulting from stress suggests that efforts within the health care system to reduce secondary biomarkers (eg lower blood pressure and glycated hemoglobin) with medications and diet is one approach to reduce racial disparities in mortality. Numerous studies have shown that interactions between patients and physicians are complicated. Unintentional misinterpretation by physicians of patient wishes and patient-related information may lead to lower rates of potentially beneficial therapeutic interventions for minorities.[5] Systemic influences such as differences in health care accessibility and adverse financial incentives may further exacerbate disparities in health care and in outcomes.[5] Broad-based efforts to improve the equity of health care delivery may help to attenuate racial disparities in allostatic load and, ultimately, in mortality.

Addressing some of the systemic disparities in areas other than health care delivery that contribute to increased stressors among black adolescents and young adults is another potential approach. Examples of these systemic racial disparities include disproportionately punitive treatment from the justice system for black adolescents vs white adolescents[34,35] or racial discrimination in the apartment rental and home mortgage markets.[36] Eliminating discriminatory policies and practices has been shown to result in improved health among blacks; several studies have demonstrated reductions in black-white disparities in life expectancy and infant mortality after passage of the 1964 Civil Rights Act.[37] Yet another approach could be trying to alter the response to environmental stressors among young racial/ethnic minorities (eg, by increasing psychosocial reserve capacity to cope with stress), which could also potentially reduce allostatic load. Small feasibility studies of this approach could be an important and viable first step in efforts to reduce persistent and unacceptable racial disparities in mortality.[38]

Our study had several limitations. First, the NHANES does not include information on primary hormonal mediators of stress (eg, markers of the hypothalamic-pituitary axis), and we were therefore unable to include them in our measure of allostatic load. Second, our measures of SES were limited to basic compositional measures (years of education, poverty status), and we were unable to adjust for either detailed individual SES measures or

contextual measures such as neighborhood- and community-level SES indicators. Third, although we used a simple summary score, it is likely that some biomarkers contributed more than others to the overall allostatic load measure. However, comparisons of a simple count with more complex weighted measures have not found major differences in predictive ability, and simpler measures are more easily defined and interpreted across populations.[10] Finally, as with any observational study, we cannot definitively infer causality, although our longitudinal study design greatly minimizes the effect of time-dependent confounding and reverse causality.

In summary, a composite measure of allostatic load partially explains black-white disparities in mortality, particularly cardiovascular- and diabetes-related mortality, after adjustment for education, poverty status, and health insurance status. These findings underscore the potential importance of chronic physiologic stressors as a negative influence on the health and lifespan of blacks in the United States. Eliminating black/white disparities in mortality across different conditions will require additional efforts to understand and mitigate the stress-induced physiologic deterioration experienced by black Americans.

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Table 1

Baseline Characteristics of NHANES III Population, Stratified by Race and Gender

	Black Men (n = 832)	White Men (n = 1226)	*P* Value	Black Women (n = 1056)	White Women (n = 1401)	*p* Value
Demographics						
Mean age (SD)	46 (0.3)	47 (0.3)	<.01	46 (0.3)	47 (0.3)	.01
Education, y						
<9 (%)	14.9	6.7	<.01	11.1	5.8	<.01
9–12 (%)	52.8	38.1		56.6	47.4	
>12 (%)	32.3	55.3		32.3	46.8	
Poor (poverty to income ratio <2), %	49.8	17.0	<.01	54.9	20.7	<.01
Has health insurance, %	85.9	92.9	.01	85.0	91.8	<.01
Comorbidities (noncardiovascular-related)						
Lung disease, %	3.9[a]	6.0	.04	9.2[a]	10.0	.62
Cancer, %	1.1[a]	2.1[a]	.10	3.8[a]	5.7[a]	.05
Thyroid disease, %	0.7[a]	1.3[a]	.2	6.4[a]	11.4	<.01
Rheumatoid arthritis, %	3.1[a]	2.6[a]	.6	6.2[a]	5.1[a]	.36
Systemic lupus erythematosus, %	0.2[a]	0.4[a]	.5	0.2[a]	0.5[a]	.18
Asthma, %	6.8[a]	7.7	.5	9.9	9.1	.54
Health behaviors						
Current smokers, %	47.7	30.7		30.4	25.0	
Former smokers, %	24.7	38.8	<.01	16.4	26.0	<.01
Never smokers, %	27.6	30.5		53.2	49.1	
Physically active, %	73.9	86.9	<.01	56.4	77.8	<.01
Nondrinkers, %	35.5	34.5		61.1	49.4	
1–30 alcoholic drinks/mo, %	51.5	53.3	.69	36.5	46.1	<.01
>30 alcoholic drinks/mo, %	13.0	12.2		2.5	4.5	
Healthy Eating Index score	57.8 (0.6)	63.0 (0.6)	<.01	60.9 (0.7)	65.1 (0.5)	<.01
Allostatic Load Components (% of each subgroup with "high-risk" values) [b]						

	Black Men (n = 832)	White Men (n = 1226)	*P* Value	Black Women (n = 1056)	White Women (n = 1401)	*p* Value
Systolic blood pressure	28.0	17.8	<.01	26.2	13.2	<.01
Diastolic blood pressure	38.8	30.2	<.01	42.4	26.6	<.01
Glycated hemoglobin	49.9	19.6	<.01	48.0	20.7	<.01
Glomerular filtration rate	10.8	19.0	<.01	8.0[a]	18.5	<.01
Albumin	27.5	13.6	<.01	28.5	12.0	<.01
Triglycerides	16.2	26.4	<.01	13.1	22.4	<.01
C-reactive protein	33.7	22.6	<.01	37.0	23.1	<.01
Homocysteine	12.1	9.2[a]	.11	9.1[a]	8.8[a]	.85
Total cholesterol	20.6	26.5	.02	19.0	21.9	.01
Waist to hip ratio	14.2	24.4	<.01	31.9	22.4	.08
Mean (SE) allostatic load score, range 0–10	2.5 (0.1)	2.1 (0.1)	<.01	2.6 (0.1)	1.9 (0.1)	<.01
Deaths						
Cardiovascular-/diabetes-specific mortality rate (per 100 patient-years)	0.63	0.33		0.36	0.21	
All-cause mortality rate (per 100 patient-years)	1.42	0.79		0.91	0.63	

[a]Estimate is unreliable, as the sample size was smaller than that recommended in the National Health and Nutrition Examination Survey analytic guidelines for the design effect and estimated proportion.

[b]*High-risk values* were defined as <25th percentile by gender for estimated glomerular filtration rate and albumin, and >75th percentile by gender for all other allostatic load components.

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Table 2

Black-White Disparities in Cardiovascular- and Diabetes-Related Mortality, by Sex[a]

	Model 1	Model 2	Model 3	Model 4
Women				
Black race	2.00 (1.31–3.06)	1.93 (1.18–3.17)	1.63 (0.96–2.75)	1.15 (0.70–1.88)
Allostatic load (per point)	–	–		1.65 (1.44–1.90)
Men				
Black race	2.24 (1.59–3.16)	2.18 (1.43–3.34)	1.93 (1.27–2.92)	1.55 (1.04–2.32)
Allostatic load (per point)				1.50 (1.34–1.68)

[a]Reference group is white race. Model 2 adjusts for education, health insurance, and poverty to income ratio. Model 3 adds smoking status, physical activity, and alcohol use to the covariates in model 2. Model 4 adds allostatic load to the covariates in model 3. All models are age-adjusted and also adjust for the Healthy Eating Index score, asthma, chronic obstructive pulmonary disease, nonskin cancer, thyroid disease, and rheumatoid arthritis. Regression models for women, but not men, also adjust for systemic lupus erythematosus.

Table 3

Black-White Disparities in Noninjury Mortality, by Sex[a]

	Model 1	Model 2	Model 3	Model 4
Women				
Black race	1.66 (1.27–2.17)	1.50 (1.07–2.11)	1.43 (1.00–2.04)	1.26 (0.90–1.78)
Allostatic load (per point)				1.23 (1.14–1.32)
Men				
Black race	2.11 (1.61–2.76)	1.73 (1.07–2.78)	1.54 (1.10–2.17)	1.39 (1.00–1.92)
Allostatic load (per point)				1.22 (1.13–1.30)

[a]Reference group is white race. Model 2 adjusts for education, health insurance, and poverty to income ratio. Model 3 adds smoking status, physical activity, and alcohol use to the covariates in model 2. Model 4 adds allostatic load to the covariates in model 3. All models are age-adjusted.