"Weathering" and Age Patterns of Allostatic Load Scores Among Blacks and Whites in the United States

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Objectives. We considered whether US Blacks experience early health deterioration, as measured across biological indicators of repeated exposure and adaptation to stressors.

Methods. Using National Health and Nutrition Examination Survey data, we examined allostatic load scores for adults aged 18–64 years. We estimated probability of a high score by age, race, gender, and poverty status and Blacks' odds of having a high score relative to Whites' odds.

Results. Blacks had higher scores than did Whites and had a greater probability of a high score at all ages, particularly at 35–64 years. Racial differences were not explained by poverty. Poor and nonpoor Black women had the highest and second highest probability of high allostatic load scores, respectively, and the highest excess scores compared with their male or White counterparts.

Conclusions. We found evidence that racial inequalities in health exist across a range of biological systems among adults and are not explained by racial differences in poverty. The weathering effects of living in a race-conscious society may be greatest among those Blacks most likely to engage in high-effort coping. (*Am J Public Health.* 2006;96:826–833. doi:10.2105/AJPH.2004.060749)

Racial/ethnic differences in chronic morbidity and excess mortality are pronounced by middle age.[1–4] Evidence of early health deterioration among Blacks and racial differences in health are evident at all socioeconomic levels.[5–7] To account for early health deterioration among Blacks, Geronimus[2] proposed the "weathering" hypothesis, which posits that Blacks experience early health deterioration as a consequence of the cumulative impact of repeated experience with social or economic adversity and political marginalization. On a physiological level, persistent, high-effort coping with acute and chronic stressors can have a profound effect on health.[8–10] The stress inherent in living in a race-conscious society that stigmatizes and disadvantages Blacks may cause disproportionate physiological deterioration, such that a Black individual may show the morbidity and mortality typical of a White individual who is significantly older. Not only do Blacks experience poor health at earlier ages than do Whites, but this deterioration in health accumulates, producing ever-greater racial inequality in health with age through middle adulthood.

In the absence of a direct measure of weathering, investigators have studied diverse health indicators such as pregnancy outcome,[11,12] excess mortality,[5,13] and disability,[3] and have found age patterns by race that are consistent with weathering. More broadly, scientists have sought to link biomarkers to social measures in an attempt to better understand the underlying physiological mechanisms of social disparities in health. Cortisol levels,[14] sympathetic nerve activity,[15] blood-pressure reactivity,[16,17] cytokine production,[18] waist-to-hip ratio,[19] and glycated hemoglobin levels[20,21] have been related to socioeconomic status,[19,22] occupation,[17] birth outcome,[23–25] and environmental risk.[26] Building on this idea, McEwen and colleagues[10,27] developed the concept of *allostatic load,* or the cumulative wear and tear on the body's systems owing to repeated adaptation to stressors.

McEwen and Seeman, and Seeman and colleagues[27,28] conceptualized allostatic load as the physiological burden imposed by stress, as indicated by 2 categories of biomarkers. The first category, primary mediators, comprises the substances the body releases in response to stress. It includes norepinephrine, epinephrine, cortisol, and dehydroepiandrosterone sulfate (DHEA-S). The second category comprises the effects that result from the actions of the primary mediators. Examples are elevated systolic and diastolic blood pressures, cholesterol levels, glycated hemoglobin levels, and waist-to-hip ratio. Allostatic load has been operationalized by algorithms that contain measurements of primary mediators or secondary effects, with associated risks assessed on a dichotomous scale and summed to produce a total allostatic load score. The first allostatic load algorithm developed by researchers, which had 10 components,[28] has been modified on the basis of available data to encompass 8,[29] 13,[30] 14,[21] or 16 components,[31] with certain components (e.g., diastolic and systolic blood pressures) common to all versions. In general, results from these studies suggest that higher allostatic load scores are associated with older age,[30] increased mortality,[31] lower socioeconomic status,[29] cognitive decline,[28] and unsupportive childhood[32] and adult[33] relationships.

An allostatic load algorithm is conceptually suited for the study of weathering. Because the stress response disrupts regulation of various systems throughout the body—for example, the cardiovascular, metabolic, and immune systems—the concept of weathering encompasses multiple systems and includes impacts on them that might not yet register clinically. Similarly, allostatic load is measured across physiological systems and includes subclinical indicators of the body's response to stress—responses that increase the risk of morbidity. For example, the algorithm includes elevated blood pressure below the clinical threshold for hypertension as well as at or above it. However, whether an allostatic load algorithm provides a useful summary measure of weathering remains an empirical question. Its usefulness for detecting differences among young through middle-aged adults across racial/ethnic populations has not been tested.

To date, studies using these algorithms in adults have been conducted primarily among selected populations homogeneous in age and race. Most research has used a subset of the MacArthur Studies of Successful Aging sample,[14,28,29,33] which only includes individuals aged 70 to 79 years who have high cognitive and physical functioning. In 2 studies, very small subsamples of medium- and low-functioning elderly people were included to compare them with high-functioning elderly people. Although 18% of the subset was Black, these individuals (aged 70–79 years old) had already exceeded the average life expectancy of Blacks.[34] Studies in Taiwan examined people aged 60 years and older,[22] as well as a group of individuals aged 54 to 70 years,[31] but this work provided limited insight into the racial disparities in US health. Similar research in younger adults—those aged 52 to 53 years from the Wisconsin Longitudinal Study,[32] those aged 20 to 80 years from the Normative Aging Study,[29] and those aged 21 to 60 years from a German manufacturing plant study[21]—included almost no racial/ethnic minorities.

One study used National Health and Nutrition Examination Survey III (NHANES III) data[30] to examine allostatic load in an age- and race-diverse population. However, analysis was not conducted by race. We expanded on previous work by analyzing a national sample of young through middle-aged adults drawn from a data set containing biomarkers and proverty measures to describe age patterns of allostatic load among Blacks and Whites.

METHODS

We used data from the fourth and most current National Health and Nutrition Examination Survey (NHANES IV, 1999–2002)[35] to examine gender and race differences in age-related allostatic load scores. NHANES surveys, conducted by the National Center for Health Statistics, use stratified, multistage probability samples to provide national estimates of health and nutritional status for the civilian, noninstitutionalized population of the United States.[35] Data are collected using a questionnaire on various health and social factors and a clinical examination, during which measurements of height, weight, and blood pressure are taken and blood is drawn.

Our NHANES IV sample included men and women aged 18–64 years. Respondents who self-identified as non-Hispanic Black or non-Hispanic White were included in our "Black" and "White" analyses, respectively. Pregnant women were excluded because their biomarker readings may be in the high-risk category owing only to the pregnancy. Individuals with missing data for any component of the algorithm also were excluded. Comparisons of excluded and included respondents revealed no difference in the age distribution of respondents; however, the excluded group contained a slightly larger proportion of poor or Black respondents, raising the possibility that Blacks with the worst health may not have been fully represented.

On the basis of previous research and data availability, 10 biomarkers were selected for inclusion in the algorithm. Systolic and diastolic blood pressures and body mass index (BMI) were obtained from physical examinations. Glycated hemoglobin, albumin, creatinine clearance, triglycerides, C-reactive protein, homocysteine, and total cholesterol were collected from blood samples (see NHANES IV documentation[35] for greater detail on measurement and biomarker assays). For each biomarker, we empirically determined the high-risk threshold on the basis of the distribution of that biomarker in our sample following a standard approach.[28,30,36] We assigned each participant a biomarker reading beyond the threshold (defined as below the 25th percentile for creatinine clearance and albumin and above the 75th percentile for all others) a point for that biomarker. The points were then summed to obtain the allostatic load score, with a maximum score of 10 possible. In the NHANES IV sample, high-risk thresholds were as follows: albumin, 4.2 g/dL; BMI, 30.9; C-reactive protein, 0.41 mg/dL; creatinine clearance, 66 mg/dL; diastolic blood pressure, 80 mm Hg; glycated hemoglobin, 5.4%; homocysteine, 9 μmol/L; systolic blood pressure, 127 mm Hg; total cholesterol, 225 mg/dL; triglycerides, 168 mg/dL.

For participants taking medication for diabetes, hypertension, or high cholesterol, we assigned a point for glycated hemoglobin, blood pressure, or total cholesterol, respectively, even if these participants did not meet or pass the high-risk threshold for the biomarker in their NHANES examination. In previous research, participants taking these medications whose clinical examination readings fell below the high-risk quartile were not assigned a point for the biomarker in question, because those individuals had brought that biomarker out of the high-risk quartile. That approach is sensible in studies of individuals aged 70 to 79 years in which baseline rates of chronic disease conditions may be high and the distinction between managing and not managing chronic disease has key implications for healthy life expectancy. However, given our focus on younger adults and on the social epidemiology of chronic disease, we took a different approach, assuming that individuals taking medication (and thus, previously diagnosed with a chronic disease) have already experienced systemic deterioration. Furthermore, taking medication to control the condition does not alleviate any aspects of the social environment that may have helped precipitate the condition.

We calculated mean scores by age category (18–24, 25–34, 35–44, 45–54, and 55–64 years), race (Black or White), and gender. We also calculated age-standardized score distribution by race and gender and found that above any score threshold (i.e., 1 through 9), the fraction of Blacks exceeded the fraction of Whites for men and women. Using logit models, we estimated the probability of having a high score as a function of age, separately by race and gender. On the basis of these regressions, we estimated Blacks' and Whites' relative odds of having a high score. We defined a high score as 4 or above. We chose this cutoff because previous literature suggests that differences in morbidity and mortality arise between groups when allostatic load scores reach 3 or 4.

We further subdivided the Black and White samples according to the poverty income ratio (PIR), an income-to-needs variable measuring the ratio of household income to the US poverty threshold, which varies by family size and composition. We defined poor as a PIR of less than or equal to 1.85, based on the eligibility cutpoint for US Department of Agriculture food assistance

programs (Special Supplemental Nutrition Program for Women, Infants, and Children [WIC], Food Stamps, School Lunch).[37,38]

We tested the robustness of our results in several ways. First, we reanalyzed data using alternative functional forms, including ordinary least squares regression, probit models, and logit models with an age-squared variable. All approaches yielded similar results. Second, we used different cutoff scores to represent a "high score." Third, we performed analyses that excluded individuals taking medication in the high-risk group. Fourth, using NHANES III and IV data, we created a cohort of individuals born between 1935 and 1975 to examine the possible contribution of cohort effects to trends in scores. That is, in the absence of longitudinal data, and under the reasonable assumption that NHANES III and NHANES IV are each representative of the same population, we used repeated cross-sections to draw inferences about population means.[39,40] (Data on homocysteine are unavailable for NHANES III, so we do not include it in this sensitivity analysis.)

Following National Center for Health Statistics guidelines, we used Mobile Examination Center (MEC) sampling weights to account for NHANES complex sampling design for all analyses.[35] We used Stata version 8.0 (Stata Corp, College Station, Tex) to account for the sampling design when calculating standard errors.

TABLE 1—Mean Allostatic Load (AL) Score and Proportion With High Score, by Race and Gender: National Health and Nutrition Examination Survey IV, 1999–2002

	Total		Men		Women	
	No.	Mean Score (% With AL≥4)	No.	Mean Score (% With AL≥4)	No.	Mean Score (% With AL≥4)
			18–24 y			
Total[b]	1560	1.23 (6.32)	825	1.21 (6.49)	735	1.25 (6.14)
Black	356	1.59 (8.33)	198	1.45 (7.92)	158	1.75 (8.80)
White	475	1.14 (5.74)	249	1.14 (6.31)	226	1.15 (5.13)
P[a]		<.01 (<.26)		<.04 (<.68)		<.01 (<.22)
			25–34 y			
Total[b]	1170	1.80 (15.00)	616	1.76 (14.19)	554	1.86 (15.94)
Black	221	2.17 (21.45)	102	1.96 (18.26)	119	2.34 (24.16)
White	510	1.76 (14.78)	272	1.67 (13.43)	238	1.86 (16.37)
P[a]		<.02 (<.11)		<.08 (<.27)		<.07 (<.19)
			35–44 y			
Total[b]	1416	2.48 (26.07)	719	2.51 (26.33)	697	2.45 (25.79)
Black	306	2.96 (37.45)	151	2.92 (34.18)	155	3.00 (40.28)
White	613	2.37 (23.53)	322	2.38 (23.92)	291	2.36 (23.12)
P[a]		<.01 (<.01)		<.01 (<.04)		<.01 (<.01)
			45–54 y			
Total[b]	1305	3.44 (45.26)	645	3.37 (45.73)	660	3.51 (44.84)
Black	248	4.04 (58.27)	130	3.78 (54.45)	118	4.34 (61.81)
White	646	3.32 (42.50)	323	3.28 (43.03)	323	3.36 (42.01)
P[a]		<.01 (<.01)		<.02 (<.07)		<.01 (<.01)
			55–64 y			
Total[a]	1135	4.12 (62.25)	568	3.84 (57.42)	567	4.39 (66.99)
Black	209	4.79 (77.28)	94	4.51 (69.49)	115	4.99 (82.68)
White	556	4.03 (59.61)	291	3.79 (55.84)	265	4.29 (63.59)
P[a]		<.01 (<.01)		<.01 (<.01)		<.01 (<.01)

[a]*P* value for comparison of Black and White mean scores (*P* value for comparison of Black and White percentages with high score).
[b]Total includes participants of all races/ethnicities.

RESULTS

In Table 1, mean scores and the percentage of respondents with high scores are reported for 5 age groups, by race and gender. In all cases, mean scores for Blacks were statistically significantly higher than mean scores for Whites. Black women consistently had higher scores than Black men, whereas scores of White men and women were similar except in the oldest age group, within which the female-to-male ratio exceeded 1.

Figure 1 shows results of the logistic regression for the full sample by race and by race and gender. Blacks had a higher probability than did Whites of a high score (≥4) at all ages. Furthermore, the size of the Black–White gap increased with age from 18 to 64 years. For Blacks aged 50 years, the probability of having a high score was approximately 60%; this probability was not reached by Whites until about age 60 years.

Black men and women also were more likely than their White counterparts to have a high score (Figure 1b). Black women had the greatest probability of having a high score and, when compared with either Black men or White women, this gap in scores grew with age from 18 to 64 years, becoming especially pronounced after age 30 years. Among Whites older than 45 years, women appeared to be slightly more likely than men to have high scores, but the differences were small and not statistically significant. By age 45 years, 50% of Black women had a high score; by age 64 years, more than 80% did. In contrast, Whites reached the 50% level only as they approached age 60 years, and they never reached levels much above 60%.

Table 2 displays the Black–White relative odds of having a high score, overall and by age group and gender, through use of different models. For individuals aged 18 to 24 years, the estimated odds of a Black person having a high score was 1.49 times that of a White person having a high score (column 1). By ages 55–64 years, the estimated Black–White relative odds rose to 2.31. These estimates were statistically significant at $P<.10$ for persons aged 25–34 years and at $P<.01$ for the age groups 35–44, 45–54, and 55–64 years.

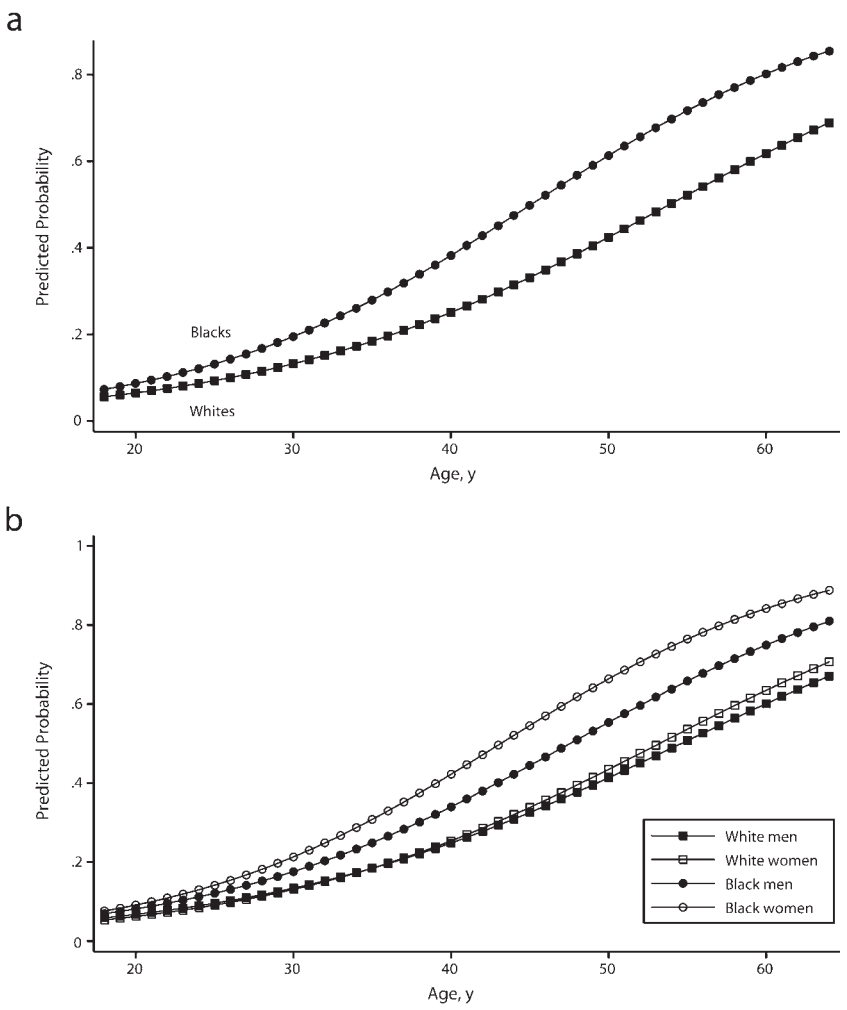


FIGURE 1—Probability of having an allostatic load of 4 or higher, as predicted by race (a) and race and gender (b).

Black men and Black women had higher estimated odds relative to their White counterparts at all age groups, and these relative odds increased in size with increasing age. For men, the Black–White odds were statistically significant at $P<.10$ at ages 45–54 years and at $P<.05$ for ages 35–44 and 55–64 years. For women, the Black–White odds of a high score were substantial, at 2.24, 2.23, and 2.73 for ages 35–44, 45–54, and 55–64 years, respectively, and highly statistically significant ($P<.01$).

Black women were consistently more likely than were Black men to have a high score. Black women aged 55 to 64 years were estimated to have more than twice the relative odds of a high score compared with Black men ($P<.05$). Among Whites, little gender difference was observed except among individuals aged 55 to 64 years, in which women were estimated to have higher relative odds (1.38; $P<.10$).

Poverty, Race, and Gender

As shown in Figure 2a, the probability by age of having a high score was greater for poor respondents (PIR≤1.85) than for nonpoor respondents (PIR>1.85). However, when the Black–White relative odds of having a high score were adjusted for PIR (Table 2, column 2), they remained strong and showed essentially the same patterns as when they were unadjusted (column 1). This result indicates that excess rates of high scores among Blacks were not accounted for by the higher proportion of Blacks who were poor. To check the robustness of this finding and to rule out residual confounding, we performed analyses with a continuous PIR variable, which confirmed our findings. Moreover, we found that *poor* Whites were less likely than *nonpoor* Blacks to have high scores (Figure 2b).

Among the poor, the relative odds of having a high score continued to favor Whites (Table 2, column 4), but the point estimates were substantially lower than for the overall sample: close to par at ages younger than 35 years and not statistically significant at conventional levels for any age group. However, among the nonpoor (Table 2, column 5), estimated relative odds were larger than for the overall estimates and were highly statistically significant for women older than 24 years and men older than 44 years. Among nonpoor respondents, Black women of all ages had at least twice the relative odds of high scores compared with White women. Nonpoor Black women aged between 55 and 64 years had 5 times the odds of high scores compared with their White counterparts.

Sensitivity Analyses

We reanalyzed the data using alternative functional forms. When we excluded respondents on medication from the high-risk group, all age groups had lower scores, but the drop in scores was greatest for Blacks aged 35 to 64 years. Even so, significant differences between Blacks and Whites persisted. We found that the pattern of results was not sensitive to the choice of allostatic load score cutoff. Indeed, we found that when a cutoff of 3 was used, Black–White differences were substantially more dramatic than those we report, which used 4 as the cutoff. Our analysis of the cohort data confirmed that for both Blacks and Whites, allostatic load scores increased between the ages of 18 and 65 years. Over the 10- to 14-year period representing the move from NHANES III to NHANES IV, mean scores (on a 9-point scale) for Blacks aged 15 to 59 years in NHANES III increased from 2.59 to 3.15 ($P<.05$). Mean scores for Whites aged 15 to 59 years in NHANES III increased from 2.11 to 2.71 ($P<.05$). This finding refutes the alternative

TABLE 2—Relative Odds (With Confidence Intervals) of Having an Allostatic Load Score of 4 or Higher: National Health and Nutrition Examination Survey IV, 1999–2002

Age Group, y	Relative Odds (95% Confidence Interval)			
	Unadjusted	Adjusted for PIR	PIR ≤ 1.85	PIR > 1.85
Blacks vs Whites				
18–24	1.49 (0.73, 3.06)	1.42 (0.69, 2.94)	1.18 (0.45, 3.06)	1.92 (0.68, 5.38)
25–34	1.57 (0.91, 2.73)*	1.41 (0.83, 2.39)	0.98 (0.53, 1.83)	2.03 (1.01, 4.09)**
35–44	1.95 (1.41, 2.67)***	1.69 (1.16, 2.46)***	1.55 (0.78, 3.08)	1.78 (1.10, 2.87)**
45–54	1.89 (1.33, 2.69)***	1.79 (1.26, 2.54)***	1.41 (0.75, 2.65)	2.00 (1.40, 2.87)***
55–64	2.31 (1.60, 3.32)***	2.22 (1.48, 3.32)***	1.36 (0.70, 2.66)	2.97 (1.52, 5.79)***
Black Women vs White Women				
18–24	1.78 (0.76, 4.12)	1.67 (0.68, 4.10)	1.21 (0.41, 3.57)	2.33 (0.60, 8.94)
25–34	1.62 (0.80, 3.30)	1.39 (0.68, 2.84)	0.95 (0.37, 2.47)	2.32 (1.02, 4.88)**
35–44	2.24 (1.39, 3.63)***	2.17 (1.31, 3.57)***	1.89 (0.76, 4.71)	2.39 (1.19, 4.79)**
45–54	2.23 (1.37, 3.64)***	1.98 (1.19, 3.28)**	1.76 (0.73, 4.26)	2.08 (1.20, 3.61)**
55–64	2.73 (1.53, 4.89)***	2.94 (1.64, 5.27)***	1.44 (0.51, 4.02)	5.12 (1.83, 14.35)***
Black Men vs White Men				
18–24	1.28 (0.40, 4.10)	1.18 (0.39, 3.58)	1.07 (0.31, 3.72)	1.50 (0.22, 10.49)
25–34	1.44 (0.76, 2.73)	1.41 (0.71, 2.78)	1.00 (0.36, 2.78)	1.86 (0.74, 4.66)
35–44	1.65 (1.06, 2.57)**	1.29 (0.81, 2.04)	1.22 (0.53, 2.77)	1.32 (0.70, 2.49)
45–54	1.58 (0.96, 2.61)*	1.57 (0.92, 2.70)*	1.02 (0.43, 2.44)	1.89 (1.01, 3.56)**
55–64	1.80 (1.12, 2.89)**	1.69 (1.00, 2.84)**	1.24 (0.46, 3.34)	1.98 (1.00, 3.91)**
Black Women vs Black Men				
18–24	1.12 (0.33, 3.80)	0.93 (0.24, 3.47)	0.53 (0.14, 2.00)	2.09 (0.29, 15.02)
25–34	1.43 (0.65, 3.15)	1.35 (0.62, 2.95)	1.68 (0.46, 6.05)	1.10 (0.46, 2.65)
35–44	1.30 (0.72, 2.33)	1.42 (0.89, 2.55)	1.02 (0.42, 2.46)	1.79 (0.74, 4.31)
45–54	1.35 (0.77, 2.39)	1.46 (0.77, 2.77)	3.88 (1.31, 11.58)**	0.87 (0.38, 1.99)
55–64	2.10 (1.01, 4.36)**	2.75 (1.27, 5.95)**	1.94 (0.44, 8.59)	3.78 (1.76, 8.20)***
White Women vs White Men				
18–24	0.80 (0.41, 1.56)	0.77 (0.40, 1.46)	0.47 (0.16, 1.42)	1.35 (0.49, 3.74)
25–34	1.26 (0.88, 1.82)	1.14 (0.78, 1.66)	1.75 (0.84, 3.67)	0.92 (0.58, 1.43)
35–44	0.96 (0.67, 1.37)	0.90 (0.65, 1.23)	0.65 (0.37, 1.17)	0.99 (0.69, 1.41)
45–54	0.96 (0.72, 1.27)	0.96 (0.73, 1.26)	2.24 (1.27, 3.97)**	0.79 (0.56, 1.11)
55–64	1.38 (0.96, 1.98)*	1.49 (0.97, 2.29)*	1.68 (0.64, 4.35)	1.46 (0.94, 2.28)*

Note. PIR = poverty income ratio.
*$P < .10$; **$P < .05$; ***$P < .01$.

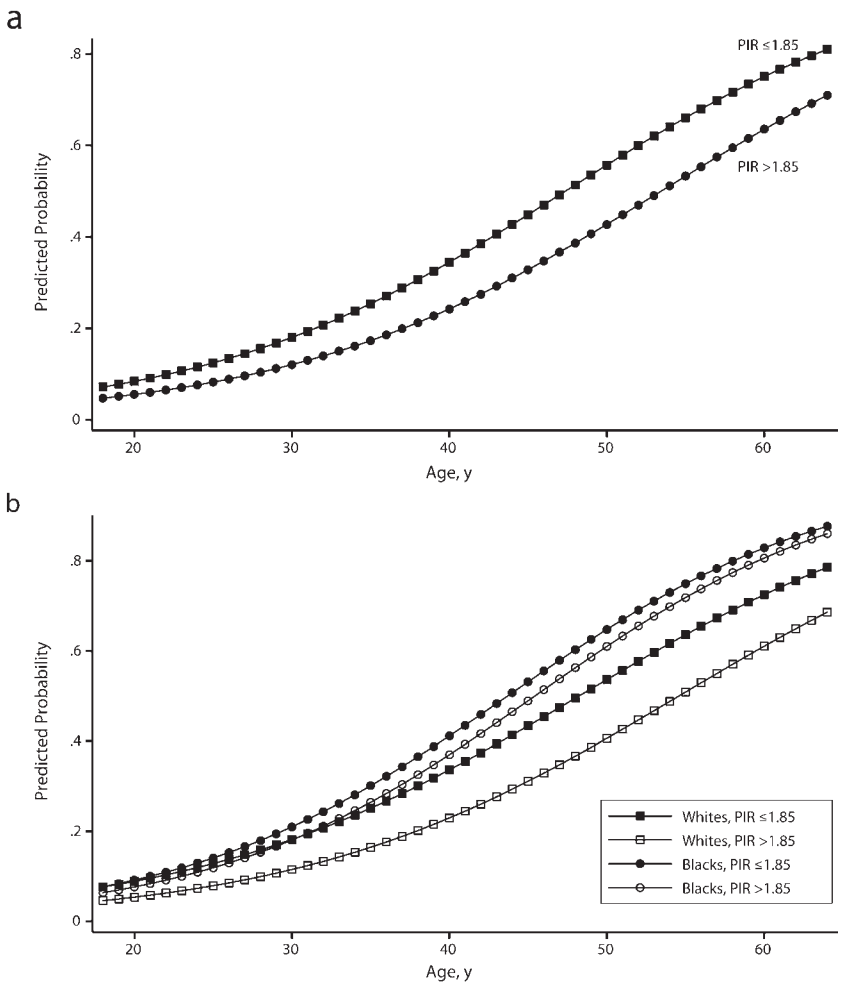
hypothesis that the cross-sectional differences we found by age and race arise from younger cohorts' being healthier than older cohorts *and* younger cohorts having smaller racial disparities than older cohorts.

DISCUSSION

Our research confirms the existence of stark racial disparities in health in clinical *and* subclinical conditions across a range of biological systems among young through middle-aged adults. Among both men and women, Blacks have higher mean allostatic load scores than do Whites at all ages, and the differential in scores increases with age. Although both poor Blacks and poor Whites have higher scores than their nonpoor counterparts, the greater poverty rates among Blacks do not account for the Black–White difference. Furthermore, nonpoor Blacks have a greater probability of high scores than do poor Whites.

When different functional forms or high score cutoff points were used in the analyses, our findings remained robust. Our sensitivity analyses suggest that our findings represent increased allostatic load with aging, rather than cohort effects. In addition, we found little difference in allostatic load scores for respondents younger than 35 years, regardless of whether medication usage was included in the algorithm. For individuals aged 35 to 64 years, allostatic load scores for Blacks were smaller when medication usage was not included in the algorithm and the size of the Black–White gap was reduced, but it remained sizable and statistically significant. The reductions observed when medicine usage was not counted in the algorithm reflect the higher burden of chronic disease experienced by Black individuals aged 35 to 64 years compared with White individuals of the same age.

Black women, in particular, bear a large burden of allostatic load compared with either Black men or White women. We found little difference among Whites in mean score by gender until age 55 years. However, Black women had higher scores than Black men at all ages studied. Differences were particularly pronounced among nonpoor Black women compared with nonpoor White women. These findings are consistent with a growing body of literature suggesting that higher economic status in Blacks is more protective against early mortality than it is against early morbidity and that racial differences in health reflect more than differences in economic resources alone.[3,12,41] The finding of larger racial disparities among the nonpoor than the poor, and among women than men, suggests that persistent racial differences in health may be influenced by the stress of living in a race-conscious society. These effects may be felt particularly by Black women because of "double jeopardy" (gender *and* racial discrimination).[12,42,43] In addition, gendered aspects of public sentiment on race may have limited Black men's role in providing social and economic security for their families, while raising expectations of Black women. For example, less-educated Black men have experienced a long secular decline in employment rates, continuing even through the labor market expansion of the 1990s.[44] In contrast, Black women bear much of the responsibility for the social and economic survival of Black families, kinship networks, and communities.[12,43,44] In fulfilling these responsibilities,



Note. PIR = poverty income ratio.

FIGURE 2—Probability of having an allostatic load of 4 or higher, as predicted by poverty income ratio (a) and poverty income ratio and race (b).

Black women may face greater exposure than Black men to stressors that require sustained and high-effort coping, along with the wear and tear on biological systems such repeated adaptation implies.[12,43,44]

Consistent with the weathering hypothesis, our findings also suggest that Blacks experience earlier deterioration of health than do Whites. In each age group, the mean score for Blacks was roughly comparable to that for Whites who were 10 years older. The predicted probabilities of having a high allostatic load score showed the same pattern. Recent research by Epel and colleagues[46] suggests that stress is related to accelerated cellular aging in young through middle-aged women because of decreased telomerase activity and shortened telomeres, the stabilizing ends of chromosomes. They found that these markers were associated with both increased perceived stress in their entire sample and with length of caregiving in their subsample of women who were caring for a chronically ill child. Their study sample was young to middle-aged women, a fact that suggests their findings may provide some insight into the cellular process of weathering, augmenting its biological plausibility. The stresses associated with living in a race-conscious society may lead to early health deterioration in Black women through a complex mechanism that includes telomere shortening.

Use of allostatic load scores to measure weathering expands on previous research that described weathering age patterns in single health indicators, because these scores encompass multiple measures across biological systems and include subclinical cases. However, the extent to which the algorithm we used can be said to capture weathering is open to question. As with all studies using allostatic load algorithms, biomarker selection was driven by data as well as theory. Although we observed Black–White differences in scores under several models and are confident that a disparity exists, no absolute score exists that can be compared across studies using different components. Most prominently, to estimate scores across racial/ethnic populations in young through middle adulthood, we had to use a data set that did not include measures of what McEwen referred to as "primary mediators." Researchers have found that these primary mediators are important contributors to the allostatic load measure, at least for predicting clinical decline or death among the elderly.[14,47]

We also note that the allostatic load algorithm used here and others similar to it in the literature have other potential limitations. First, in reality, it is unlikely that each biomarker used contributes equally to allostatic load. However, evidence suggests that if an equiweighted algorithm does not reflect physiological reality, it may provide a conservative estimate of the size of the relationship between allostatic load and poor health outcomes.[47] Second, each score is derived through use of a threshold demarcating the traditional high-risk end of the distribution for each biomarker (75th or 25th percentile). Biomarkers at this end of the distribution are associated with stress-related diseases: heart disease (C-reactive protein, homocysteine, total cholesterol, triglycerides), liver disease (albumin), obesity (BMI), hypertension (blood pressure), kidney disease (creatinine clearance), and diabetes (glycated hemoglobin). It is possible that for some of these biomarkers, having a value at the other end of the distribution may confer high-risk status for disease outcomes that are not stress related. For example, although hypotension and low cholesterol are clinically significant conditions, they are not stress related.

In sum, racial differences in allostatic load scores are small in the late teens and early 20s, but they quickly widen beginning in young adulthood through middle age and are largest between the ages of 35 and 64 years. Black women of these ages suffer the highest probability of having a high allostatic load score whether compared with Black men or with White men or women. These findings provide evidence that the impact of chronic stress on health has important implications not only for individuals but also for the population as a whole and suggest ways that dynamic social relationships between racial and ethnic groups may shape health in a race-conscious society. The findings suggest that progress in understanding and eliminating racial health inequality may require paying attention to the ways that American public sentiment on race, including its gendered aspects, exacts a physical price across multiple biological systems from Blacks who engage in and cope with the stressful life conditions presented to them. ■

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Contributors

A.T. Geronimus conceptualized the article, oversaw the analysis, interpreted the findings, and wrote significant portions of the text. M. Hicken contributed to the conceptualization of the allostatic load algorithm, performed statistical analyses, and wrote portions of the text. D. Keene contributed to the conceptualization of the allostatic load algorithm, performed statistical analyses, and wrote portions of the text. J. Bound conceptualized key statistical analyses, helped direct their operation, and participated in their interpretation.

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References

1. Elo IT, Preston SH. Educational differentials in mortality: United States, 1979–85. *Soc Sci Med.* 1996; 42:47–57.

2. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis.* 1992;2:207–221.

3. Geronimus AT, Bound J, Waidmann TA, Colen CG, Steffick D. Inequality in life expectancy, functional status, and active life expectancy across selected black and white populations in the United States. *Demography.* 2001;38:227–251.

4. Wong MD, Shapiro MF, Boscardin WJ, Ettner SL. Contribution of major diseases to disparities in mortality. *N Engl J Med.* 2002;347:1585–1592.

5. Geronimus AT, Bound J, Waidmann TA, Hillemeier MM, Burns PB. Excess mortality among blacks and whites in the United States. *N Engl J Med.* 1996; 335:1552–1558.

6. Williams DR. Race, socioeconomic status, and health. The added effects of racism and discrimination. *Ann N Y Acad Sci.* 1999;896:173–188.

7. Schoendorf KC, Hogue CJ, Kleinman JC, Rowley D. Mortality among infants of black as compared with white college-educated parents. *N Engl J Med.* 1992; 326:1522–1526.

8. James SA, Keenan NL, Strogatz DS, Browning SR, Garrett JM. Socioeconomic status, John Henryism, and blood pressure in black adults. The Pitt County Study. *Am J Epidemiol.* 1992;135:59–67.

9. Sapolsky RM. *Why Zebras Don't Get Ulcers—A Guide to Stress, Stress-Related Disorders and Coping.* 2nd ed. New York, NY: WH Freeman Publishers; 1998.

10. McEwen BS. Protective and damaging effects of stress mediators. *N Engl J Med.* 1998;338:171–179.

11. Rauh VA, Andrews HF, Garfinkel RS. The contribution of maternal age to racial disparities in birthweight: a multilevel perspective. *Am J Public Health.* 2001;91:1783–1789.

12. Geronimus AT. Understanding and eliminating racial inequalities in women's health in the United States: the role of the weathering conceptual framework. *J Am Med Womens Assoc.* 2001;56:133–136, 149–150.

13. Astone NM, Ensminger M, Juon HS. Early adult characteristics and mortality among inner-city African American women. *Am J Public Health.* 2002;92: 640–645.

14. Seeman TE, Singer B, Wilkinson CW, McEwen B. Gender differences in age-related changes in HPA axis reactivity. *Psychoneuroendocrinology.* 2001;26: 225–240.

15. Uchino BN, Cacioppo JT, Kiecolt-Glaser JK. The relationship between social support and physiological processes: a review with an emphasis on underlying mechanisms and implications for health. *Psychol Bull.* 1996;119:488–531.

16. Roy MP, Steptoe A, Kirschbaum C. Life events and social support as moderators of individual differences in cardiovascular and cortisol reactivity. *J Pers Soc Psychol.* 1998;75:1273–1281.

17. Steptoe A, Feldman PJ, Kunz S, Owen N, Willemsen G, Marmot M. Stress responsivity and socioeconomic status: a mechanism for increased cardiovascular disease risk? *Eur Heart J.* 2002;23:1757–1763.

18. Cohen S, Doyle WJ, Skoner DP. Psychological stress, cytokine production, and severity of upper respiratory illness. *Psychosom Med.* 1999;61:175–180.

19. Marmot MG, Ruhrer R, Ettner SL, Marks NF, Bumpass LL, Ryff CD. Contribution of psychosocial factors to socioeconomic differences in health. *Milbank Q.* 1998;76:403–448.

20. Daniel M, O'Dea K, Rowley KG, McDermott R, Kelly S. Social environmental stress in indigenous populations: potential biopsychosocial mechanisms. *Ann N Y Acad Sci.* 1999;896:420–423.

21. Schnorpfeil P, Noll A, Schulze R, Ehlert U, Frey K, Fischer JE. Allostatic load and work conditions. *Soc Sci Med.* 2003;57:647–656.

22. Weinstein M, Goldman N, Hedley A, Yu-Hsuan L, Seeman T. Social linkages to biological markers of health among the elderly. *J Biosoc Sci.* 2003;35: 433–453.

23. Wadhwa PD, Culhane JF, Rauh V, Barve SS. Stress and preterm birth: neuroendocrine, immune/inflammatory, and vascular mechanisms. *Matern Child Health J.* 2001;5:119–125.

24. Stancil TR, Hertz-Picciotto I, Schramm M, Watt-Morse M. Stress and pregnancy among African American women. *Paediatr Perinat Epidemiol.* 2000;14: 127–135.

25. Maes M, Son C, Lin A, et al. The effects of psychological stress on humans: increased production of pro-inflammatory cytokines and a Th1-like response in stress-induced anxiety. *Cytokine.* 1998;10:313–318.

26. Evans GW. A multimethodological analysis of cumulative risk and allostatic load among rural children. *Dev Psychol.* 2003;39:924–933.

27. McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci.* 1999;896:30–47.

28. Seeman TE, Singer BH, Rowe JW, Horwitz RI, McEwen BS. Price of adaptation—allostatic load and its health consequences. *Arch Intern Med.* 1997;157: 2259–2268.

29. Kubzansky LD, Kawachi I, Sparrow D. Socioeconomic status, hostility, and risk factor clustering in the Normative Aging Study: any help from the concept of allostatic load? *Ann Behav Med.* 1999;21: 330–338.

30. Crimmins EM, Johnston M, Hayward M, Seeman T. Age differences in allostatic load: an index of physiological dysregulation. *Exp Gerontol.* 2003;38: 731–734.

31. Seeman T, Glei D, Goldman N, Weinstein M, Singer B, Yu-Hsuan L. Social relationships and allostatic load in Taiwanese elderly and near elderly. *Soc Sci Med.* 2004;59:2245–2257.

32. Singer B, Ryff CD. Hierarchies of life histories and associated health risks. *Ann N Y Acad Sci.* 1999;896: 96–115.

33. Seeman TE, Singer BH, Ryff CD, Love GD, Levy-Storms L. Social relationships, gender, and allostatic

load across two age cohorts. *Psychosom Med.* 2002;64: 395–406.

34. Arias E. United States life tables, 2001. *Natl Vital Stat Rep.* 2004;52(14):1–38.

35. NHANES home page. CDC Web site. Available at: http://www.cdc.gov/nchs/nhanes.htm. Accessed August 28, 2004.

36. Seeman TE, McEwen BS, Rowe JW, Singer BH. Allostatic load as a marker of cumulative biological risk: MacArthur studies of successful aging. *Proc Natl Acad Sci U S A.* 2001;98:4770–4775. E-publication April 3, 2001.

37. NHANES 1999–2000 addendum to the NHANES III analytic guidelines. Updated August 30, 2002. Available at: http://www.cdc.gov/nchs/data/nhanes/guidelines1.pdf. Accessed August 28, 2004.

38. Kramer-LeBlanc CS, Mardis A, Gerrior S, Gaston N. Review of the Nutritional Status of WIC Participants. Final Report. Washington, DC: USDA Center for Nutrition Policy and Promotion; December 1999. Report CNPP-8. Available at: http://www.cnpp.usda.gov/Pubs/Wic/wicrprt2.pdf. Accessed August 28, 2004.

39. Deaton A. T*he Analysis of Household Surveys: A Microeconometric Approach to Development Policy.* Baltimore, Md: Johns Hopkins University Press; 1997.

40. Deaton A. Panel data from time series of cross-sections. *J Econom.* 1985:109–126.

41. Williams DR, Collins C. US socioeconomic and racial differences in health: patterns and explanations. *Annu Rev Sociol.* 1995;21:349–386.

42. James SA. John Henryism and the health of African-Americans. *Cult Med Psychiatry.* 1994;18: 163–182.

43. Mullings L, Wali A. *Stress and Resilience: The Social Context of Reproduction in Central Harlem.* New York, NY: Kluwer Academic/Plenum Publishers; 2001.

44. Holzer, HJ, Offner P, Sorenson E. Declining employment among young Black less-educated men: the role of incarcertation and child support. *J Policy Anal Manage.* 2005;24:329–350.

45. Geronimus AT, Thompson JP. To denigrate, ignore, or disrupt: racial inequality in health and the impact of a policy-induced breakdown of African American communities. *Du Bois Rev.* 2004;1:247–279.

46. Epel ES, Blackburn EH, Lin J, et al. Accelerated telomere shortening in response to life stress. *Proc Natl Acad Sci U S A.* 2004;101:17312–17315.

47. Karlamangla AS, Singer BH, McEwen BS, Rowe JW, Seeman TE. Allostatic load as a predictor of functional decline. MacArthur studies of successful aging. *J Clin Epidemiol.* 2002;55:696–710.