

Accelerated Health Declines among African Americans in the USA

Roland J. Thorpe, Ruth G. Fesahazion, Lauren Parker, Tanganiyka Wilder, Ronica N. Rooks, Janice V. Bowie, Caryn N. Bell, Sarah L. Szanton, and Thomas A. LaVeist

ABSTRACT *The weathering hypothesis, an explanation for race disparities in the USA, asserts that the health of African Americans begin to deteriorate prematurely compared to whites as a consequence of long-term exposure to social and environmental risk factors. Using data from 2000–2009 National Health Interview Surveys (NHIS), we sought to describe differences in age-related health outcomes in 619,130 African Americans and whites. Outcome measures included hypertension, diabetes, stroke, and cardiovascular disease. Using a mixed models approach to age-period-cohort analysis, we calculated age- and race-specific prevalence rates that accounted for the complex sampling design of NHIS. African Americans exhibited higher prevalence rates of hypertension, diabetes, and stroke than whites across all age groups. Consistent with the weathering hypothesis, African Americans exhibited equivalent prevalence rates for these three conditions 10 years earlier than whites. This suggests that African Americans are acquiring age-related conditions prematurely compared to whites.*

KEYWORDS *Weathering hypothesis, Health disparities, Chronic conditions, Mixed models, African Americans, Stress, Allostatic load*

BACKGROUND

There is overwhelming evidence of disparities in morbidity and mortality among African Americans and other ethnic groups compared to whites in the USA.^{1–6} The magnitude and consistency of these disparities have led scientists to move beyond mere documentation of differences to efforts that seek to explain the etiology of health disparities. Several potential explanations have been proposed and many have been empirically tested. These include individual-level factors, such as socioeconomic status (SES),^{5, 7} health behaviors,^{4, 8} and access to and quality of medical

Thorpe, Fesahazion, Bowie, Szanton, and LaVeist are with the Hopkins Center for Health Disparities Solutions, Johns Hopkins Bloomberg School of Public Health, 624 N. Broadway, Ste 708, Baltimore, MD 21205, USA; Thorpe, Parker, and Bowie are with the Department of Health, Behavior, and Society, Johns Hopkins Bloomberg School of Public Health, Baltimore, USA; Thorpe is with the Center for Biobehavioral Health Disparities Research, Duke University, Durham, USA; Wilder is with the Department of Biological Sciences, Florida A&M University, Tallahassee, FL, USA; Rooks is with the Department of Health and Behavioral Sciences, University of Colorado Denver, Denver, USA; Bell is with the Department of African American Studies, University of Maryland College Park, College Park, MD, USA; Szanton is with the Johns Hopkins School of Nursing, Johns Hopkins University, Baltimore, USA; LaVeist is with the Department of Health Policy and Management, Milken Institute School of Public Health, George Washington University, Washington, DC, USA.

Correspondence: Roland J. Thorpe, Jr, Hopkins Center for Health Disparities Solutions, Johns Hopkins Bloomberg School of Public Health, 624 N. Broadway, Ste 708, Baltimore, MD 21205, USA. (E-mail: rthorpe@jhu.edu)

care;⁹ societal influences ranging from government policies to race-based discrimination;^{10, 11} environmental factors (such as the workplace or living environment);¹²⁻¹⁴ and possible genetic influences.^{15, 16} However, a large proportion of observed racial differences in health remain unexplained.

African Americans tend to develop age-related diseases in which racial health disparities have been observed at earlier ages than whites.^{3, 17} Two key explanations exist. One explanation for this is the substantial evidence that African Americans experience differential exposure to chronic stressors such as discrimination, racism, and segregation,^{3, 5, 7, 11, 12, 18, 23} and have different life experiences compared to non-Hispanic whites.²⁴⁻²⁸ The second is stress—an social or environmental pressure that often requires at least one of the following responses: psychological, physiological, or behavioral.^{29, 50} Moreover, these factors can lead to alterations in physical and mental health.³⁰ Many diseases, especially those related to cardiovascular disease, have been linked to stress, adverse life events, and other factors related to stress such as allostatic load,³¹ social support,^{4, 22} and discrimination.³² Thus, because of race differences in the social and environmental risk exposures and life experiences facilitated by residential segregation,^{11, 12, 23, 33, 34} African Americans could be expected to have an earlier onset of disease and disability than non-Hispanic whites.^{6, 11, 35-37}

There is a burgeoning body of literature documenting the association between age, SES, and health indices.^{37, 56} House and colleagues³⁷ wrote a seminal paper that exhibits earlier onset of chronic disease and functional limitations associated with SES. Using data from the *Americans' Changing Lives* study, they demonstrated that both education and income were significantly and independently related to chronic conditions and functional status. More importantly, they showed the effect of age on health indices vary by level of social stratification. For example, among those with the least education, significant increases in rates of functional impairments were observed in middle adulthood. Among the most highly educated group, there was a delay in the onset of functional decline by 30 years compared to the least educated. Exposure to chronic stressors and adverse life experiences is inversely related to SES as well as race; indeed, African Americans are disproportionately represented in the lowest SES groups (^{5, 13}; LaVeist et al. 2007). This suggests the possibility that both differential exposures to stressors and the ability to cope with them may contribute to the earlier onset in the course of health declines among individuals with limited SES resources or African Americans.

The weathering hypothesis, developed by Geronimus,^{35, 40} has become a framework for observing accelerated health declines. This hypothesis states that the health status of African American adults begins to deteriorate prematurely compared to other American ethnic groups as a consequence of long-term exposure to social and environmental risk factors. An interaction exists between age and race/ethnicity such that African Americans acquire conditions normally associated with older age at younger ages than whites.^{36, 40} Thus, observed health disparities in the onset of age-related diseases by racial/ethnic groups are reflective of persistent weathering in African Americans. A number of studies have demonstrated age patterns by race that are consistent with the weathering hypothesis for a variety of health indicators such as reproductive health,⁴¹⁻⁴³ mortality,⁴⁴ hypertension,^{2, 45} allostatic load,⁴⁶ and functional status⁴⁷ and telomere length.⁶

Few studies testing the weathering hypothesis have addressed the methodological issue of determining if age effects represent age effects or cohort effects.^{2, 46} The objective of this study was to test the weathering hypothesis by investigating whether differences exist in patterns of age-related chronic conditions in a national

sample of African American and white adults. Specifically, we hypothesize that African Americans will experience earlier onset of disease, suggesting accelerated health declines and supporting the weathering hypothesis. This cross-sectional study adds to the body of literature by testing the weathering hypothesis using a mixed models approach that will provide an opportunity to tease apart age, period, and cohort effects as well as accounting for the potential violation of independence of errors assumption.⁴⁸

DATA AND METHODS

The National Health Interview Survey (NHIS) is an annual face-to-face survey of the civilian, non-institutionalized households in the USA. This survey, conducted by the US Census Bureau for the National Center for Health Statistics, uses a multistage stratified cluster sampling design. Detailed information regarding NHIS can be found elsewhere.^{49, 50} Briefly, NHIS is comprised of questions regarding demographic characteristics, health status, health behaviors, functional limitations, AIDS, cancer screening, and health-care access and utilization. While African Americans were oversampled in each survey to ensure reliable estimates for these groups, we combined 10 years of data to ensure a sufficient sample size for race and age group using data from the 2000–2009 NHIS Public Use Person and Sample Adult Files. For this project, our analyses consisted of 619,130 adults ages 18 and older with 20.3 % being African American.

Outcome measures included self-report of four health conditions: hypertension, stroke, diabetes, and cardiovascular disease (CVD). These conditions were selected because their prevalence is known to increase with age.^{9, 51, 52} Participants reported whether a “doctor or other health professional” told them they had hypertension, stroke, or diabetes. In addition, those participants who reported whether a “doctor or other health professional” told them they had coronary heart disease, angina, a myocardial infarction, or other heart disease were considered to have CVD. Each condition was specified as a binary variable. Race, the main independent variable, was self-reported. Race was determined by the response to “What race or races do you consider yourself to be?” Those who reported their race as white only or black/African American only and reported their ethnicity as non-Hispanic were included. Age was categorized into the following age groups: 18–24, 25–34, 35–44, 45–54, 55–64, 65–74, and 75 and older.

Covariates included demographic and health-related characteristics. Demographic variables included sex, education level, marital status, and income category. Male sex was specified as a binary variable. Educational level was dichotomized as high school graduate or not. Marital status was dichotomized as married or not. Three binary variables were used to categorize income: <\$35,000, \$35,000–\$74,999, and >\$75,000. Because we combined 10 years of data, a variable to represent survey year was included in our models to account for secular trends.

Health-related characteristics included obesity, health insurance status, and physical inactivity. Based on self-reported height and weight, body mass index (BMI) was calculated. We classified participants whose BMI ≥ 30 kg/m² as obese relative to participants whose BMI <30 kg/m². A dichotomous variable was created to classify those individuals who had health insurance. Physical inactivity was specified as a binary variable where respondents reported they did not participate in light, moderate, or vigorous leisure-time physical activities at least once a week for a minimum of 10 min.

Using Student's *t* and chi-square tests, we evaluated the mean and proportional differences across racial/ethnic groups for the demographic characteristics and the health-related factors. We used a mixed models approach to conduct the age-period-cohort analysis of repeated cross-section surveys outlined by Yang and Land⁴⁸ that account for the potential violation of the independence of errors assumption. We specified logistic regression models to examine the association between race group and each of the health conditions by age group. Covariates in the fully adjusted model included: male sex, educational level, income, marital status, obesity, physical inactivity, smoking status, and insurance status. Age group-specific and race-specific models were used to obtain the prevalence of the health outcome. All analyses have been adjusted by Taylor-linearization procedures to account for the multistage sampling design of the NHIS data. To account for the population size across the ten surveys used in this project, we created a weight variable by averaging across the weights of the ten survey years.⁴⁹ All estimates presented herein are weighted. *p* values <0.05 were considered statistically significant and all tests were two sided. Analyses were conducted using Stata version 10.

RESULTS

Select characteristics of 2000–2009 NHIS participants by race are displayed in Table 1. Fewer African Americans were males and reported being married compared to whites. African Americans exhibited lower SES profiles relative to whites with lower percentages of each group being high school graduates and higher percentages of each group having incomes less than \$35,000. As it relates to health-related factors, a smaller proportion of African Americans compared to whites had health insurance. A larger proportion of African Americans were physically inactive or obese than whites.

The adjusted prevalence of hypertension by race and age is shown in Table 2. African Americans had significantly higher prevalence of hypertension than whites in all age groups. Although the prevalence of hypertension increased with age for

TABLE 1 Distribution of selected characteristics by race/ethnicity, National Health Interview Survey, 2000–2009

	Total	White	Black
Characteristics	<i>n</i> = 619,130	<i>n</i> = 495,108	<i>n</i> = 124,022
Age (years), mean (S.E.)	37.6 (0.03)	38.9 (0.03)	32.7 (0.1)*
Male, %	47.9	48.7	45.0*
Married, %	59.3	63.6	40.7*
High school grad, %	86.7	87.6	83.0*
Income			
<\$35,000	25.5	22.0	39.5*
\$35,000–\$75,000	26.3	27.1	22.9*
>\$75,000	21.6	24.4	10.4*
Missing	26.7	26.6	27.2
Insurance, %	88.3	89.5	83.4*
Obese, %	44.4	41.2	58.2*
Physically inactive, %	40.2	37.5	51.8*

*Significant difference at $p \leq 0.05$ comparing Black to whites

both race groups, the increase was much higher for African Americans. For example, the prevalence of hypertension for African Americans ages 35–44 was 21.6 %. The prevalence of hypertension for whites ages 45–54 was 23.1 %. Thus, a similar prevalence of hypertension among whites is achieved 10 years later than that of African Americans.

The adjusted prevalence of stroke by race and age is shown in Table 3. African Americans had significantly higher prevalence of stroke between age 45–54 (2.4 vs. 1.1 %) and age 55–64 (5.9 vs. 2.7 %) compared to whites. Similar to hypertension for these age groups, the rate of strokes in whites occurred 10 years later than African Americans. The prevalence of stroke for African Americans age 45–54 is 2.4 %; the prevalence of stroke for whites does not reach this level until age 55–64 (2.7 %). There were no observed differences for African Americans and whites in the following age groups: 18–24, 25–34, 35–44, 65–74, and 75+.

The adjusted prevalence of diabetes by race and age is shown in Table 4. With the exception of participants ages 18–34, African Americans had higher prevalence of diabetes compared to whites. In the age groups between 35 and 75+, the prevalence of diabetes was nearly double for African Americans and increased at a faster rate compared to whites. For example, in the 45–54 age group, African Americans had a prevalence of 10.1 % and whites had a prevalence of 4.5 %.

The adjusted prevalence of CVD by race and age is shown in Table 5. African Americans age 35–44 had a lower prevalence of CVD (4.4 vs. 5.9 %) compared to whites. Among older adults aged 65+, African Americans also had a lower prevalence of CVD relative to whites. African Americans had a prevalence of 22.8 % in the 65–74 age group and 27.9 % in the 75+ age group, compared to 26.1 and 36.7 %, respectively, among whites. There were no observed differences in the prevalence of CVD between African Americans and whites in the three younger age groups.

DISCUSSION

The purpose of the study was to examine the effect of weathering between African Americans and whites. In this national sample of non-institutionalized adults, African Americans consistently exhibited higher prevalence rates of hypertension, diabetes, and stroke across all age groups. Moreover, African American prevalence rates in a given age group for all health conditions were not reached in whites until at least 10 years later. As it relates to CVD, whites displayed a higher prevalence rate across all age groups except ages 55–64. This differential in prevalence increased with age and is consistent with accelerated health declines among African Americans.

The results from this study share findings with previous ones that have tested the weathering hypothesis.^{2, 6, 36, 41-45, 47} However, most of the previous work has largely focused on “weathering” between races and ethnicities, namely African Americans, Mexican Americans, and whites. This work is similar in this respect but extends to other highly prevalent chronic conditions such as diabetes, stroke, and CVD that these populations typically face. Despite examining prevalence by race and age groups across a few chronic conditions, African Americans exhibit the worse health even when accounting for demographic and health-related factors. This suggests that there may be something inherent to the African American experience that underlies these disparities in health that was not measured in this study.

TABLE 2 Adjusted prevalence of hypertension by race and age among 2000–2009 NHIS participants

	White American (%)	95 % confidence interval (%)	African American (%)	95 % confidence interval (%)
18–24	3.3	(2.7–3.9)	6.0*	(4.4–7.5)
25–34	6.4	(5.7–7.0)	11.6*	(10.3–13.7)
35–44	11.6	(10.8–12.3)	21.6*	(19.9–24.1)
45–54	23.1	(22.0–24.1)	45.2*	(42.7–48.0)
55–64	40.7	(39.2–41.9)	65.9*	(63.7–69.9)
65–74	52.0	(50.5–53.5)	73.6*	(70.5–77.2)
75+	55.3	(53.8–56.7)	70.7*	(67.4–75.3)

*Significant difference at $p \leq 0.05$ in the adjusted prevalence between whites and African Americans. Models were adjusted for male sex, educational level, income, marital status, obesity, physical inactivity, smoking status, and insurance status

One unmeasured factor that may contribute to the accelerated health declines particularly among African Americans is residential segregation, a form of racial discrimination. The weathering hypothesis contends that the early onset of health declines in African Americans is a consequence of long-term exposure to harmful social and environmental conditions often facilitated by residential segregation. Moreover, the history of slavery, reconstruction, the Jim Crow era, and perceived racism and discrimination have set the stage for many structural limitations and increased stressors faced by African Americans (^{53, 54}; Kreiger et al. 2013). These stressors and structural factors have enduring effects that might be explained by the weathering hypotheses. For example, African Americans may experience weathering because they are more likely to live in high risk and under-resourced environments with fewer opportunities to reach higher levels of education and build wealth.^{12, 33, 34, 55, 56} Additionally, living in a segregated environment may lead to African Americans facing barriers to care and receiving inferior quality of care when they are ill or injured. Minority communities are more likely to be physician shortage areas^{57, 18} and residents of minority communities use fewer health-care services.⁵⁸ Hospitals that primarily serve minority patients are known to have worse outcomes for some medical and surgical conditions.^{54, 60} Even for acute conditions such as trauma, recent data suggests that hospitals with a higher proportion of African American

TABLE 3 Adjusted prevalence of stroke by race and age among 2000–2009 NHIS participants

	White American (%)	95 % confidence interval (%)	African American (%)	95 % confidence interval (%)
18–24	0.0	(0.0–0.0)	0.0	(0.0–0.0)
25–34	0.2	(0.1–0.3)	0.1	(0.0–0.4)
35–44	0.6	(0.4–0.8)	0.8	(0.3–1.2)
45–54	1.1	(0.8–1.3)	2.4*	(1.8–3.3)
55–64	2.7	(2.2–3.2)	5.9*	(4.5–7.8)
65–74	6.1	(5.4–6.8)	8.0	(6.1–10.6)
75+	9.7	(8.8–11.1)	12.1	(9.5–15.4)

*Significant difference at $p \leq 0.05$ in the adjusted prevalence between whites and African Americans. Models were adjusted for male sex, educational level, income, marital status, obesity, physical inactivity, smoking status, and insurance status

TABLE 4 Adjusted prevalence of diabetes by race and age among 2000–2009 NHIS participants

	White American (%)	95 % confidence interval (%)	African American (%)	95 % confidence interval (%)
18–24	0.6	(0.4–0.9)	0.6	(0.3–1.2)
25–34	1.1	(0.9–1.5)	1.8	(1.3–2.7)
35–44	2.0	(1.6–2.4)	4.5*	(3.5–5.8)
45–54	4.5	(3.9–5.0)	10.1*	(8.5–11.8)
55–64	9.1	(7.9–9.7)	22.5*	(19.7–25.5)
65–74	13.1	(11.8–13.9)	26.7*	(23.4–30.3)
75+	11.6	(10.4–12.4)	21.6*	(17.9–25.8)

*Significant difference at $p \leq 0.05$ in the adjusted prevalence between whites and African Americans. Models were adjusted for male sex, educational level, income, marital status, obesity, physical inactivity, smoking status, and insurance status

and minority patients have increased mortality compared to hospitals with a primarily white trauma patient population.⁶¹ Moreover, overt interpersonal racial discrimination as daily, chronic stressors have also been associated with precursors of age-related diseases such as inflammation (Lewis, Aiello, and Leurgans et al.⁶² 2010) and oxidative stress.⁶³ Thus, residential segregation as a form of racial discrimination may facilitate weathering or the early onset of age-related diseases in response to chronic stress.

One plausible explanation for the association between discrimination and accelerated health declines among African Americans is allostatic load. This concept refers to the cumulative wear and tear on many physiological systems in the body as a result of repeatedly adapting to persistent chronic, psychosocial stressors.⁶⁴ This adaptation leads to the activation of the HPA axis which leads to increased blood pressure, cortisol, epinephrine, and other neurohormonal substances. In the setting of perceived or real discrimination, chronic and/or hyper-activation of many of cellular response systems can lead to maladaptive health consequences such as increased rates of hypertension, depression, infectious diseases, cancers, and cardio-metabolic conditions.⁶⁵ For example, studies suggest that the perception of discrimination ultimately leads to higher diastolic blood pressures^{66–69}; ⁶¹ and other

TABLE 5 Adjusted prevalence of cardiovascular disease by race and age among 2000–2009 NHIS participants

	White American (%)	95 % confidence interval (%)	African American (%)	95 % confidence interval (%)
18–24	3.5	(2.8–4.0)	1.8	(1.2–2.7)
25–34	4.3	(3.8–4.8)	3.6	(2.8–4.6)
35–44	5.9	(5.4–6.4)	4.4*	(3.5–5.6)
45–54	10.1	(9.4–10.8)	11.7	(10.1–13.6)
55–64	17.3	(16.2–18.3)	19.7	(17.2–22.6)
65–74	26.1	(24.7–27.4)	22.8*	(19.6–26.3)
75+	36.7	(35.4–38.2)	27.9*	(24.1–32.2)

*Significant difference at $p \leq 0.05$ in the adjusted prevalence between whites and African Americans. Models were adjusted for male sex, educational level, income, marital status, obesity, physical inactivity, smoking status, and insurance status

systemic responses that destabilize the natural stress fighting environment and alternatively hyper-responds to single or multiple insults of stress, irrespective of source. This cellular hyper-responsivity, over time, leads to increased expression and systemic serum levels of substances like interleukins, TNF-alpha, and C-reactive protein, which are inflammatory mediators and strong predictors of coronary heart failure, coronary heart disease, stroke, diabetes, and hypertension.^{70, 71} Indeed, allostatic load has even been reported to partially explain higher mortality rates among blacks, independent of SES and health behaviors.⁷²

There are some aspects of our study that deserve comment. First, while residential segregation is a plausible explanation for the weathering hypothesis particularly among African Americans, the data to test this proposition is not available in the NHIS public use dataset. It is possible that other social and physical environmental factors independent of and in conjunction with residential segregation may be associated with weathering. Second, our measure of prevalence was based on physician diagnosis. Underserved populations are often under diagnosed which may lead to a mistakenly small prevalence for minorities who are less likely to have health insurance. Although we controlled for health insurance in our analyses, it is also well documented that racial minorities, even with health insurance, have less access to health care than do whites⁹; this may cause residual confounding. This same fact that whites have more access to health-care services may also partially explain the higher prevalence of CVD reported by white participants in NHIS. Because this study uses cross-sectional data, the ability to make inferences regarding causality is limited. Despite the limitations, this study has a number of strengths. First, this study uses 10 years of NHIS data that ensured a sufficient sample size for all race and age groups. Second, there were several variables included in the models to account for potential confounding. Third, when examining age effects using cross-sectional data, it is possible that the effects observed represent cohort effects. A mixed models statistical technique was used to disentangle the age-period-cohort effects that often occur with repeated cross-section surveys.⁴⁸ Another advantage of this approach is that this technique accounts for the potential violation of the independence of errors assumption. This is advancement on previous cross-sectional studies that have examined the weathering hypothesis.

Patterns of hypertension, diabetes, and stroke are consistent with accelerated health declines in African Americans. This suggests that African Americans are acquiring conditions normally associated with aging at younger ages than other race groups. These accelerated health declines are likely due to African Americans experiencing the synergistic effects of multiple health challenges and comorbid conditions, chronic psychological stress, and high effort coping because of residential segregation, interpersonal racism, and discrimination. Policies should target opportunities to remove structural barriers that may impede the improvement of overall population health and the reduction of health disparities,^{11, 22, 23, 34} possibly focusing on cohort effects, or health events for younger African Americans, or period effects targeting all ages. For example, a period effect policy focused on structurally improving communities and the health of community members might include reducing food insecurity, via the Healthy Food Financing Initiative at the federal level and many similar state initiatives, to decrease health disparities in the nutrition-related diseases we examined (<http://healthyfoodaccess.org/find-money/hffi?destination=node/304>^{73, 74}). Efforts to develop interventions and health promoting strategies to delay or postpone the onset of health events among African Americans at younger ages will also require the public health sector to go

beyond its traditional boundaries and concentrate its attention on building collaborations with those focusing on key social determinants of health such as education, housing, and employment.

ACKNOWLEDGMENTS

The research conducted by the first author was supported by a grant from the National Institute for Minority Health and Health Disparities (P60MD000214). The first author is a visiting scholar in the Center for Biobehavioral Health Disparities Research in the Social Science Research Institute at Duke University. The first author and fourth author are supported by the National Institute on Heart, Lung, and Blood Institute (R25HL12614).

REFERENCES

1. Ferraro KF, Farmer MM. Double jeopardy to health hypothesis for African Americans: analysis and critique. *J Health Soc Beh.* 1996; 64: 303–15.
2. Geronimus AT, Bound J, Keene D, Hicken M. Black-white differences in age trajectories of hypertension prevalence among adult women and men, 1999–2002. *Ethn Dis.* 2007; 17(1): 40–8.
3. LaVeist TA, Bowie JV, Cooley-Quille M. Minority health status in adulthood: the middle years of life. *Health Care Financ Rev.* 2000; 21(4): 9–21.
4. Sudano JJ, Baker DW. Explaining US racial/ethnic disparities in health declines and mortality in late middle age: the roles of socioeconomic status, health behaviors, and health insurance. *Soc Sci Med.* 2006; 62: 909–22.
5. Williams DR, Collins C. US socioeconomic and racial differences in health: patterns and explanations. *Annu Rev Sociol.* 1995; 21: 391–431.
6. Geronimus AT, Pearson JA, Linnenbringer E, Schulz AJ, Reyes AG, Epel ES, Lin J, Blackburn E. Race-ethnicity, poverty, urban stressor, and telomere length in a Detroit community-based sample. *J Health Soc Behav.* 2015; 56(2): 199–224.
7. Hayward MD, Miles TP, Eileen MC, Yang Y. The significance of socioeconomic status in explaining the racial gap in chronic health conditions. *Am Sociol Rev.* 2000; 65(6): 910–30.
8. Lantz PM, Golberstein E, House JS, Morenoff J. Socioeconomic and behavioral risk factors for mortality in a national 19-year prospective study of U.S. adults. *Soc Sci Med.* 2010; 70(10): 1558–66.
9. Committee on understanding and eliminating racial and ethnic disparities in health care. *Unequal treatment: confronting racial and ethnic disparities in health care.* 1st ed. Washington, D.C.: National Academies Press; 2002: 782.
10. LaVeist TA, Gaskin D, Trujillo AJ. Segregation spaces, risky places: the effects of racial segregation on health inequalities. Report. Joint Center for Political and Economic Studies, 2011. Available on at <http://www.jointcenter.org/research/seggregated-spaces-risky-places-the-effects-of-racial-segregation-on-health-inequalities>.
11. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep.* 2001; 116(5): 404–16.
12. LaVeist T, Pollack K, Thorpe RJ, Fesahazion R, Gaskin D. Place, not race: disparities dissipate in southwest Baltimore when blacks and whites live under similar conditions. *Health Aff.* 2011; 30(10): 1880–7.
13. LaVeist TA, Thorpe RJ Jr, Bowen-Reid T, et al. Exploring health disparities in integrated communities: overview of the EHDIC study. *J Urban Health.* 2008; 85(1): 11–21.

14. Thorpe RJ Jr, Jackson J, LaVeist TA. Social context as an explanation for race disparities in hypertension: findings from the Exploring Health Disparities in Integrated Communities (EHDIC) study. *Soc Sci Med*. 2008; 67: 1604–11.
15. Sankar P, Cho MK, Condit CM, et al. Genetic research and health disparities. *JAMA*. 2004; 291(24): 2985–9.
16. Kuzawa CW, Sweet E. Epigenetics and the embodiment of race: developmental origins of U.S. racial disparities in cardiovascular health. *Am J Hum Biol*. 2009; 21: 2–15.
17. Nuru-Jeter A, Thorpe RJ Jr, Fuller-Thomson E. Black-white differences in self-reported disability outcomes in the U.S.: early childhood to older adulthood. *Public Health Rep*. 2011; 126(6): 834–43.
18. Crimmins EM, Hayward MD, Seeman TE. Race/ethnicity, socioeconomic status and health. In: Anderson NB, Bulatao RA, Cohen B, eds. *Critical perspective on racial and ethnic differences in health in later life*. Washington D. C: National Academies Press; 2004: 310–52.
19. Hummer RA, Benjamins MR, Rogers RG. Racial and ethnic disparities in health and mortality among US elderly. In: Anderson NB, Bulatao RA, Cohen B, eds. *Critical perspective on racial and ethnic differences in health in later life*. Washington D. C: National Academies Press; 2004: 53–94.
20. Hummer RA, Chinn JJ. Race/ethnicity and U.S. adult mortality: progress, prospects, and new analyses. *Du Bois Rev*. 2011; 8(1): 5–24.
21. Link BG, Phelan J. Social conditions as fundamental causes of disease. *J Health Soc Behav*. 1995; Spec No: 80–94.
22. Williams DR, Mohammed SA. Racism and health I: pathways and scientific evidence. *Am Behav Sci*. 2013;57(8).
23. Thorpe RJ Jr, Richard P, Bowie JV, LaVeist TA, Gaskin DJ. Economic burden of men's health disparities in the United States. *Int J Mens Health*. 2013; 12(3): 195–212.
24. Ferraro KF, Kelley-Moore JA. Cumulative disadvantage and health: long-term consequences of obesity? *Am Sociol Rev*. 2003; 68(5): 707–29.
25. Ferraro KF, Thorpe RJ Jr, McCabe GP, Kelly-Moore JA, Jiang Z. The color of hospitalization over the adult life course: cumulative disadvantage in black and white? *J Gerontol B Psychol Sci Soc Sci*. 2006; 61(6): S299–306.
26. O'Rand AM. The precious and the precocious: understanding cumulative disadvantage and cumulative advantage over the life course. *Gerontologist*. 1996; 36(2): 230–8.
27. Whitfield KE, Weidner G, Clark R, Anderson NB. Sociodemographic diversity and behavioral medicine. *J Consult Clin Psychol*. 2002; 70(3): 463–81.
28. Bruce MA, Beech BM, Sims M, et al. Social environmental stressors, psychological factors, and kidney disease. *J Invest Med*. 2009; 57(4): 583–9.
29. Bruce M, Griffith DM, Thorpe RJ Jr. Stress and kidney disease. *Adv Chronic Kidney Dis*. 2015; 22(1): 46–54.
30. Szanton SL, Gill J, Allen JK. Allostatic load: a mechanism of socioeconomic health disparities? *Biol Res Nurs*. 2005; 7(1): 7–15.
31. Bell CN, Thorpe RJ Jr, LaVeist TA. Race/ethnicity and hypertension: the role of social support. *Am J Hypertens*. 2010; 23(5): 534–40.
32. Landrine H, Corral I. Separate and unequal: residential segregation and black health disparities. *Ethn Dis*. 2009; 19: 179–84.
33. Williams DR. The health of men: structured inequalities and opportunities. *Am J Public Health*. 2003; 93(5): 724–31.
34. Geronimus AT. The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis*. 1992; 2(3): 207–21.
35. Geronimus AT. Black/white differences in the relationship of maternal age to birth weight: a population-based test of the weathering hypothesis. *Soc Sci Med*. 1996; 42(4): 589–97.
36. House JS, Kessler RC, Herzog AR. Age socioeconomic status and health. *Milbank Q*. 1990; 68(3): 383–411.

37. House JS, Lantz PM, Herd P. Continuity and change in the social stratification of aging and health over the life course: evidence from a nationally representative longitudinal study from 1986 to 2001/2002 (Americans' Changing Lives Study). *J Gerontol B Psychol Sci Soc Sci.* 2005; 60(Spec No. 2): 15–26.
38. House JS, Lepkowski JM, Kinney AM, et al. The social stratification of aging and health. *J Health Soc Behav.* 1994; 35: 213–34.
39. 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 Women's Assoc.* 2001; 56(4): 133–6.
40. Geronimus AT. The effects of race, residence, and prenatal care on the relationship of maternal age to neonatal mortality. *Am J Public Health.* 1986; 76(12): 1416–21.
41. Geronimus AT. The effects of race, residence, and prenatal care on the relationship of maternal age to neonatal mortality. *Am J Public Health.* 1986; 76(12): 1416–21.
42. Rauh VA, Andrews HF, Garfinkel RS. The contribution of maternal age to racial disparities in birth weight: a multilevel perspective. *Am J Public Health.* 2001; 91(11): 1815–24.
43. Wildsmith EM. Testing the weathering hypothesis among Mexican-origin women. *Ethn Dis.* 2002; 12(4): 470–9.
44. Astone NM, Ensminger M, Juon HS. Early adult characteristics and mortality among inner-city African American women. *Am J Public Health.* 2002; 92(4): 640–5.
45. Geronimus AT, Andersen HF, Bound J. Differences in hypertension prevalence among U.S. black and white women of childbearing age. *Public Health Rep.* 1991; 106(4): 393–9.
46. Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health.* 2006; 96(5): 826–33.
47. 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(2): 227–51.
48. Yang Y, Land KC. A mixed models approach to the age-period-cohort analysis of repeated cross-section surveys: trends in verbal test scores. *Sociol Methodol.* 2006; 36: 75–97.
49. National Health Interview Survey. *Public use data release.* Hyattsville: Division of Health Interview Statistics, National Center for Health Statistics; 2008.
50. National Center for Health Statistics. National Health Interview Survey: research for the 1995–2004 redesign. *Vital Health Stat.* 1999; 2: 1–119.
51. Burt VL, Whelton P, Roccella EJ, et al. Prevalence of hypertension in the US adult population. Results from the third National Health and Nutrition Examination Survey, 1988–1991. *Hypertension.* 1995; 25(3): 305–13.
52. Hayward MD, Herron M. Racial inequality in active life among adult Americans. *Demography.* 1999; 36: 77–91.
53. Feagin J, McKinney K. *The many costs of racism.* Lanham: Rowman & Littlefield; 2003.
54. Krieger N, Chen J, Coull B, Waterman P, Beckfield J. The Unique Impact of Abolition of Jim Crow Laws on Reducing Inequities in Infant Death Rates and Implications for Choice of Comparison Groups in Analyzing Societal Determinants of Health. *American Journal of Public Health* 2013; 103(12); 2234–2244.
55. Thorpe Jr. RJ, Kelley E*, Bowie J, Griffith D, Bruce M, LaVeist T. Explaining Racial Disparities in Obesity Among Men: Does Place Matter? *American Journal of Men's Health*, 9(6):464–472.
56. Morello-Frosch R, Lopez R. The riskscape and the color line: examining the role of segregation in environmental health disparities. *Environ Res.* 2006; 102(2): 181–96.
57. Gaskin DJ, Dinwiddie GY, Chan K, McCleary RR. Residential segregation and the availability of primary care physicians. *Health Serv Res.* 2012; 47(6): 2353–76.

58. Gaskin DJ, Dinwiddie GY, Chan K, McCleary RR. Residential segregation and the use of healthcare services. *Med Care Res Rev.* 2012; 69(2): 158–75.
59. Gaskin DJ, Spencer CS, Richard P, Anderson G, Powe NR, LaVeist TA. Do hospitals provide lower quality care to minorities compared to whites? *Health Aff.* 2008; 27(2): 518–27.
60. Hasnain-Wynia R, Baker DW, Nerenz D, et al. Disparities in health care are driven by where minority patients seek care: examination of the hospital quality alliance measures. *Arch Intern Med.* 2007; 167(12): 1233–9.
61. Haider AH, Ong’uti S, Efron DT, et al. Association between hospitals caring for a disproportionately minority trauma population demonstrate increased trauma mortality: a nationwide analysis of 434 hospitals [published online ahead of print September 19, 2013]. *Arch Surg.* 2012. <http://archsurg.jamanetwork.com/article.aspx?articleid=1107780>.
62. Lewis T, Aiello A, Leurgans S, Kelly J, Barnes L. Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African American adults. *Brain Behavior, and Immunity,* 2010; 24(3): 438–443.
63. Szanton SL, Rifkind JM, Mohanty JG, Miller ER, Thorpe RJ, Nagababu E, Epel ES, Zonderman AB, Evans MK. Racial discrimination is associated with a measure of red cell oxidative stress: a potential pathway for racial health disparities. *Int J Behav Med.* 2012; 19(4): 489–95.
64. McEwen BS, Stellar E. Stress and the individual mechanisms leading to disease. *Arch Intern Med.* 1993; 153(18): 2093–101.
65. Djuric Z, Bird CE, Furumoto-Dawson A, Rauscher GH, Ruffin MTT, Stowe RP, Masi CM. Biomarkers of psychological stress in health disparities research. *Open Biomark J.* 2008; 1: 7–19. doi:10.2174/1875318300801010007.
66. Din-Dzietham R, Nembhard WN, Collins R, Davis SK. Perceived stress following race-based discrimination at work is associated with hypertension in African-Americans. The Metro Atlanta Heart Disease Study, 1999–2001. *Soc Sci Med.* 2004; 58(3): 449–61.
67. Krieger N, Sidney S. Racial discrimination and blood pressure: the CARDIA study of young black and white adults. *Am J Public Health.* 1996; 86(10): 1370–8.
68. Steffen PR, McNeilly M, Anderson N, Sherwood A. Effects of perceived racism and anger inhibition on ambulatory blood pressure in African Americans. *Psychosom Med.* 2003; 65(5): 746–50.
69. Thomas KS, Nelesen RA, Malcarne VL, Ziegler MG, Dimsdale JE. Ethnicity, perceived discrimination, and vascular reactivity to phenylephrine. *Psychosom Med.* 2006; 68(5): 692–7.
70. Bastard JP, Maachi M, Lagathu C, Kim MJ, Caron M, Vidal H, Feve B. Recent advances in the relationship between obesity, inflammation, and insulin resistance. *Eur Cytokine Netw.* 2006; 17(1): 4–12.
71. Upadhyay RK. Emerging risk biomarkers in cardiovascular diseases and disorders. *J Lipids.* 2015; 2015: 971453. doi:10.1155/2015/971453.
72. Duru OK, Harawa NT, Kermah D, Norris KC. Allostatic load burden and racial disparities in mortality. *J Natl Med Assoc.* 2012; 104(1–2): 89–95.
73. Bell J, Mora G, Hagan E, Rubin V, Karpyn A. Access to healthy food and why it matters: a review of the research. PolicyLink and the Food Trust. 2013.
74. Healthy Food Financing Initiative. Healthy Food Access Portal sponsored by PolicyLink, The Food Trust, and The Reinvestment Fund, last accessed on 5/30/14 at <http://healthyfoodaccess.org/find-money/hffi?destination=node/304>.