Discrimination, Racial Bias, and Telomere Length in African-American Men

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Background: Leukocyte telomere length (LTL) is an indicator of general systemic aging, with shorter LTL being associated with several chronic diseases of aging and earlier mortality. Identifying factors related to LTL among African Americans may yield insights into mechanisms underlying racial disparities in health.

Purpose: To test whether the combination of more frequent reports of racial discrimination and holding a greater implicit anti-black racial bias is associated with shorter LTL among African-American men.

Methods: Cross-sectional study of a community sample of 92 African-American men aged between 30 and 50 years. Participants were recruited from February to May 2010. Ordinary least squares regressions were used to examine LTL in kilobase pairs in relation to racial discrimination and implicit racial bias. Data analysis was completed in July 2013.

Results: After controlling for chronologic age and socioeconomic and health-related characteristics, the interaction between racial discrimination and implicit racial bias was significantly associated with LTL (b=-0.10, SE=0.04, p=0.02). Those demonstrating a stronger implicit antiblack bias and reporting higher levels of racial discrimination had the shortest LTL. Household income-to-poverty threshold ratio was also associated with LTL (b=0.05, SE=0.02, p < 0.01).

Conclusions: Results suggest that multiple levels of racism, including interpersonal experiences of racial discrimination and the internalization of negative racial bias, operate jointly to accelerate biological aging among African-American men. Societal efforts to address racial discrimination in concert with efforts to promote positive in-group racial attitudes may protect against premature biological aging in this population.

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Introduction

frican-American men experience disproportionately greater chronic disease burden and accelerated declines in health compared to other racial and gender groups in the U.S.¹⁻⁴ Overall life expectancy

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0749-3797/\$36.00 http://dx.doi.org/10.1016/j.amepre.2013.10.020 for African-American men is 69.7 years, compared to 75.7 years for white men.⁴ African-American men experience aging-related diseases earlier in life and suffer greater severity and worse consequences of disease compared to other groups.^{5,6} These racial disparities in health may be traced to disproportionately greater psychosocial stressors experienced by African-American men, particularly those uniquely tied to racial minority status.^{7–10}

Racial discrimination constitutes a qualitatively distinct stressor, which continues to be salient and pervasive in the lives of African Americans. 11,12 Several studies have found that experiences of racial discrimination, in domains such as employment, housing, education, and legal contexts, as well as more routine experiences of being treated with less courtesy or respect are perceived as being stressful. 12-14 These experiences may affect disease risk via mental health pathways as well as through maladaptive behavioral coping mechanisms. 15-20 Racial

discrimination can also have more direct effects on health through its impact on biological systems engaged in the stress response.²¹ Self-reports of racial discrimination have been associated with a range of biological markers of stress, including neuroendocrine risk markers for poor health outcomes, glucocorticoids, and proinflammatory cytokines. 15,21-24 Further, negative psychological responses coupled with the experience of racial discrimination may have particularly deleterious effects on disease vulnerability.²⁵ Holding a negative evaluation of one's own racial group may constitute an additional source of psychosocial stress and may moderate the effect of racial discrimination. 26-29 Adopting negative in-group racial attitudes may lead to poor self-concept by impeding positive racial identity formation. These characteristics may compromise the ability to cope with stressors, particularly those associated with race, and increase vulnerability to psychosocial challenges. 30-34

Along these lines, experiences of racial discrimination and in-group racial bias may have negative implications for aging at the biological level. In particular, there is growing interest in studying telomere length in the development and progression of aging-related diseases.^{35–37} Telomeres are repetitive sequences of DNA at the ends of chromosomes that protect against DNA degradation. In eukaryotes, the DNA sequences at the terminal end of the lagging strand are lost during replication.^{38,39} By capping the ends of chromosomes, telomere attrition occurs in most adult somatic cells with each mitotic cycle, resulting in an annual loss of 50-100 base pairs. In this respect, telomeres are important in supporting chromosomal stability, and critically short telomeres are associated with cellular senescence. Accordingly, telomere length has been posited to be a marker of replicative history and aging at the cellular level. 40,41 Telomere length from leukocytes are generally preferred to telomere length from other cell types as they may reflect overall immune health, and has been posited to be a marker of general systemic aging of the organism. Leukocyte telomere length (LTL) has been associated with several aging-related health outcomes such as cardiovascular disease, diabetes, dementia, Alzheimer's disease, and arthritis, as well as earlier mortality, in addition to their associated risk factors (e.g., biological, behavioral, and environmental). 42-46 Importantly, studies suggest that psychosocial and physiologic stressors can lead to accelerated LTL shortening and may be a mechanism that helps to explain differences in the onset of chronic diseases. 47-49 For example, depression, financial stressors, strains associated with caregiving, and health behaviors such as exercise and smoking have all been associated with LTL. $^{50-54}\,$

Together, research suggests that identifying factors that affect LTL can be informative in examining variations in disease risk and understanding racial disparities in health.^{55–57} Although some cross-sectional studies have found no racial differences in LTL or that African Americans may in fact have longer LTL,^{58–60} other recent studies have found that African Americans have shorter telomeres and/or undergo a faster rate of LTL shortening compared to whites.^{55–57,60} For example, although perhaps initially having longer LTL than whites, a recent study found a steeper inverse association between chronologic age and LTL for African Americans.⁶⁰ Similarly, a longitudinal cohort study found that African Americans had a faster rate of telomere shortening compared to whites.⁵⁷

As a marker of cumulative stress and physiologic wear and tear, LTL may be a particularly relevant biological marker of health to study in relation to racial minority stressors that are experienced throughout the lifecourse.⁶¹ The current study is the first to examine whether experiences of racial discrimination and ingroup racial bias are associated with LTL in a community sample of African-American men. In-group racial bias was measured using the black-white Implicit Association Test (IAT), an experimental technique that assesses unconscious racial attitudes. 62,63 The IAT measures the speed with which participants match images of faces and words with positive and negative valence with their respective categories (African American/white and good/bad). Faster pairings are posited to be more closely associated with representations in memory. National studies have found that 70% of people in the U.S. display an implicit anti-black bias, including about half of African Americans, making on average faster categorizations when mapping the African American-bad and white-good pairing condition in comparison to the African American-good and white-bad condition.⁶⁴ The IAT may be a more valid measure of in-group racial bias compared to explicit reports given that it is not susceptible to environmental or other extraneous factors, and because performance is not influenced by the provision of socially desirable responses.⁶⁴

Consonant with racial identity frameworks, this study tested whether there would be a significant interaction between racial discrimination and implicit racial bias, with racial discrimination having a steeper negative association with LTL among those holding a greater implicit anti-black bias. Specifically, it was hypothesized that those reporting high levels of racial discrimination and who display an implicit anti-black bias would have the shortest LTL among black middle-aged men.

Methods

Study Design and Procedures

Data were from a cross-sectional study of African-American men. A total of 95 African-American men were recruited between February 2010 and May 2010 from the San Francisco Bay Area. Eligibility criteria for participation were (1) self-identification as an African-American man; (2) aged between 30 and 50 years; (3) U.S. nativity and parental U.S. nativity; (4) absence of serious or unstable disease (e.g., cancer, HIV/AIDS, tuberculosis, hepatitis); and (5) ability to read, write, and understand English.

Participants were recruited from socioeconomically diverse neighborhoods and at outlets where the population was most accessible, including churches, barbershops, and community events; through self-referral from posted advertisements; and via word of mouth. Eligible participants were provided with an appointment date and time to meet study staff in a private location in a nonclinical setting (e.g., university room, church). Study procedures were (1) a brief face-to-face interview assessing basic demographic characteristics; (2) a minimally invasive physical exam; (3) administration of the IAT; and (4) a computer-assisted self-interview including more sensitive measures of racial discrimination, psychological factors, and socioeconomic measures. The physical exam included the collection of anthropometric data and dried blood spots (DBS). Collection of DBS samples entailed pricking the finger with a micro-lancet, wiping away the first drop of blood, and applying four subsequent drops, each approximately 50 µL to filter paper. 65 Blood samples were allowed to dry and stored at -80° C.

Participants provided informed consent and were compensated with a \$70.00 gift card. All study procedures were approved by the University of California San Francisco Committee on Human Research.

Leukocyte Telomere Length Assay

The Blackburn laboratory at the University of California, San Francisco, conducted LTL assays. The protocol was adapted from previously published methods. 66-69 Genomic DNA was purified from DBS using QIAamp DNA Investigator Kit. This kit is used to obtain high-quality DNA that can be used in downstream analysis such as quantitative PCR (qPCR), which is the most commonly used method to obtain average telomere length. An average of 56 ng (range: 12 ng-340 ng) of total DNA was obtained from six of 3-mm punched spots. Telomere length was measured twice, each time using half of the obtained DNA. The average coefficient of variation between the two runs was 6.3%. A recent study reported very high correlations between telomere length from DBS and telomere length from both whole blood (R^2 =0.741) and peripheral mononuclear blood cells $(R^2=0.789)$. Technical information on the LTL assay procedure is presented in Appendix A (available at www.ajpmonline.org). The mean LTL in this study is consistent with expected values based on a prior study of midlife men that used the same line of control cells.⁷

Implicit Association Test

Implicit racial bias was assessed using the black–white IAT administered to participants via computer using Inquisit software. The IAT is a continuous measure that ranges from -1 to +1, with a score of zero representing neutral. Increasing scores less than zero indicate a stronger pro-black bias, whereas those greater than zero reflect a stronger anti-black bias. Test–retest reliability of the IAT has been shown to be high. 64

Supporting the validity of the IAT as a measure of unconscious racial bias, a study found that those with a stronger anti-black bias

reported greater explicit prejudice against blacks and also had greater negative interactions with a black experimenter as rated by an independent judge.⁷² Research on the IAT in health domains have focused on provider bias and its potential role in the treatment and outcomes of patients^{73,74}; however, studies on implicit racial bias in relation to an individual's own health status are in their infancy. One study reported that lower racial bias is associated with lessened stress response, and greater anti-black bias is associated with decrements in executive function among white participants when interacting with blacks. 75,76 There is a paucity of research examining the IAT specifically in African-American samples. A study of African Americans found that in-group racial bias predicted greater attentional impairment when interacting with whites.⁷⁷ A recent study found a positive association between racial discrimination and hypertension risk among African Americans with an implicit antiblack bias.⁷⁸

Racial Discrimination

Racial discrimination was measured using the situation version of the Experiences of Discrimination (EOD) questionnaire. Participants were asked whether they had experienced discrimination because of their race in nine situations: getting a job; at work; getting housing; getting medical care; getting service at a store or restaurant; getting credit, bank loans, or a mortgage; on the streets or in other public settings; or from the police or in the courts. The total number of situations in which racial discrimination was reported ranged from 0 to 9. The EOD is a commonly used and validated measure of racial discrimination. Page 10

Sociodemographic Characteristics

Sociodemographic variables that were examined included selfreported age and poverty ratio calculated as the ratio of household income to the poverty threshold based on family size, with higher ratios reflecting lower levels of poverty.⁸¹ Categorical variables were educational attainment (high school or less versus some college or more) and employment status (employed versus unemployed). These variables were dichotomized based on the frequency of responses and because using more detailed categories did not change results. Relationship status (currently married, never married, or formerly married); occupation (nonmanual, manual, or unemployed) coded from self-reported primary job; and insurance status (insured or uninsured) based on current coverage were also examined but excluded because they were not significantly related to LTL and did not show evidence of confounding the relationship between primary predictors and LTL.

Health-Related Variables

The number of physical health conditions was assessed using a checklist of 22 common diseases (e.g., cardiovascular diseases, diabetes, cancer, renal disease). Scores in this sample ranged from 0 to 9, with 0 being the most commonly reported (33.7%). The most frequently reported conditions were seasonal allergies, chronic back or neck problems, and other chronic pain. Doctor-prescribed medication use in the past week was assessed using a single item (yes versus no). Current smokers were defined using CDC-recommended criteria as those who reported smoking at

least 100 cigarettes in their lifetime and currently smoking at least "some days." Waist-hip ratio was calculated by dividing waist circumference by hip circumference in inches. 83

Data Analysis

Two outlying LTL values of 4.00 kilobase pairs (kb) and 6.91 kb (3.56 times the SD below and 3.18 times the SD above the mean) were excluded from analyses.

Ordinary least squares (OLS) regressions were used to examine continuous measures of racial discrimination, implicit racial bias, and their interaction in relation to LTL. Sociodemographic factors were added to the model in a single block group, followed by health-related covariates. Regression diagnostics consistently revealed one extremely influential observation that had the highest Cook's D (0.11; cut-off: 0.04) and DFITS (1.18; cut-off: 0.69), which indicate observations with the greatest residual and leverage. Accordingly, this observation was deleted from the analyses, resulting in a total analytic sample size of 92 participants. Including this observation in analyses lessened the magnitude of associations but did not result in substantively different conclusions. Analyses were completed in July 2013 using SAS, version 9.3, statistical software. Data analysis was completed in July 2013.

Results

Leukocyte telomere length values ranged from 4.80 to 6.44 kb (M=5.54; SD=0.38). Participants reported experiencing racial discrimination in several different situations. Only six participants (6.5%) reported not experiencing discrimination in any of the nine situations. Sixteen participants (17.4%) reported racial discrimination in 1-3 situations; 31 participants (33.7%) in 3-6 situations; and 39 participants (42.4%) in 7–9 situations. Most commonly reported was racial discrimination from the police or in the courts, which was endorsed by 79 (85.9%) of the African-American men in this study. This was followed by racial discrimination in getting a job (n=67, 72.7%) and at work (n=65, 70.7%). In this sample, 58 participants (63%) had IAT scores less than zero, reflecting an implicit problack bias; 34 participants (37%) had IAT scores greater than zero, reflecting an implicit anti-black bias.

Descriptive analyses revealed high levels of unemployment (54.3%). Furthermore, 29.2% participants were below the poverty threshold and 28.1% had poverty ratios between 1.00 and 1.99. Less than half of participants (42.7%) had poverty ratios of 2.00 or above. Additional sociodemographic characteristics of the sample are presented in Table 1.

In bivariate analyses, there was no association between racial discrimination or implicit racial bias on LTL. Significant bivariate relationships with demographic variables were the following: increasing age was associated with shorter LTL; greater household income to poverty threshold was associated with longer LTL; and prescription drug use was associated with shorter LTL.

Table 1. Distribution of characteristics in the sample of African-American men

Characteristic	M (SD) or n (%)	
Leukocyte telomere length (kb)	5.54 (0.38)	
Racial discrimination	5.55 (2.73)	
Implicit racial bias	-0.14 (0.35)	
Age***	43.86 (5.72)	
Poverty ratio**	1.95 (2.24)	
Education, n (%)		
High school or less	38 (41.3)	
Some college or more	54 (58.7)	
Employment status, n (%)		
Employed	42 (45.7)	
Unemployed	50 (54.3)	
Smoking, n (%)		
Noncurrent	41 (44.6)	
Current	51 (55.4)	
Health conditions	1.73 (1.89)	
Prescription medications,** n (%)		
No	63 (68.5)	
Yes	29 (31.5)	
Waist-hip ratio	0.92 (0.07)	

*p<0.05, **p<0.01, ***p<0.001, indicating significant bivariate relationships with leukocyte telomere length

Supplementary analyses revealed no significant relationships between demographic variables and implicit racial bias.

There were no main effects of racial discrimination (b=-0.02, SE=0.02, p=0.28) and implicit racial bias (b=-0.09, SE=0.11, p=0.44) in an OLS regression model examining LTL that included these two variables, but as predicted there was a significant interaction between them (b=-0.10, SE=0.04, p=0.02; Table 2, Model 1). This interaction remained significant after controlling for sociodemographic factors (Model 2) as well as health-related variables (Model 3). Age, poverty ratio, and prescription drug use remained significant covariates in the final model.

Predicted LTL values were calculated for varying levels of racial discrimination and implicit racial bias. Values of 4, 6, and 8 were chosen to represent low, moderate, and high levels of racial discrimination, which correspond to quartile values. The median value on the IAT among participants with values less than zero (-0.31)

Table 2. Linear regressions predicting leukocyte telomere length in kilobase pairs among African-American men, b (SE)

	Model 1	Model 2	Model 3
Intercept	5.68 (0.09)***	6.20 (0.30)***	6.57 (0.53)***
Racial discrimination	-0.03 (0.02)	-0.01 (0.01)	-0.02 (0.01)
Implicit racial bias	0.51 (0.27)	0.53 (0.26)*	0.54 (0.26)*
Discrimination × bias	-0.10 (0.04)*	-0.10 (0.04)*	-0.10 (0.04)*
Age		-0.01 (0.01)*	-0.02 (0.01)*
Poverty ratio		0.06 (0.02)**	0.05 (0.02)**
Some college versus high school or less		-0.08 (0.08)	-0.08 (0.07)
Unemployed versus employed		-0.09 (0.08)	-0.08 (0.08)
Current versus nonsmoker			0.00 (0.08)
Health conditions			0.01 (0.02)
Medication: yes versus no			-0.22 (0.08)**
Waist-hip ratio			-0.23 (0.53)
R^2	0.08	0.27	0.33

^{*}p<0.05, **p<0.01, ***p<0.001

and greater than zero (value=0.13) were chosen to represent those with an implicit pro- versus anti-black bias, respectively. Mean values were used for continuous covariates, and the overall proportion of participants belonging to a group for categorical covariates was used in order to calculate LTL values for the average participant. Figure 1 illustrates that among participants with a pro-black bias, there was a slight positive relationship between racial discrimination and LTL. In contrast, among those with an anti-black bias, there was an inverse relationship between racial discrimination and LTL.

Discussion

African-American men face unique psychosocial stressors that contribute to worsening health. Among these challenges are legally sanctioned forms of discrimination, such as "stop-and-frisk" policies, and pervasive racial profiling across multiple domains, ranging from the judicial system to healthcare contexts. Studies also indicate that African-American men are susceptible to racial discrimination in obtaining jobs and in mortgage markets, although expressly prohibited by law law standard aggressions through interpersonal interactions. Results from this study suggest that such experiences result in physiologic tolls among those who have internalized negative racial group attitudes.

Specifically, racial discrimination was associated with shorter LTL among African-American men with an implicit anti-black bias. African-American men with an implicit bias against their own group may be compromised in their ability to psychologically manage or cope with stress resulting from racial discrimination. 94-96 Holding an anti-black bias in tandem with the experience of externally perpetrated racial discrimination may represent threats to both self- and group identity and together have especially detrimental consequences for telomeric aging.^{27,31} In contrast, holding a pro-black bias may serve as a buffer against racial stressors.^{27,97}

Results are consistent with prior studies that have found that those with a bias against their own racial group are more vulnerable to the impact of racial stigma, and that greater in-group identification and positive racial

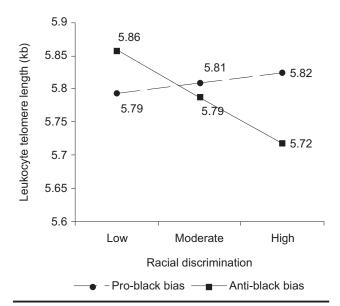


Figure 1. Predicted leukocyte telomere length by racial discrimination and implicit racial bias among African-American men

Note: Quartile values were chosen to represent low (4); moderate (6); and high (8) levels of racial discrimination. Median values for participants with values less than zero and greater than zero on the Implicit Association Test were chosen to represent those with an implicit problack and anti-black bias, respectively.

evaluation may lessen the negative impact of racial discrimination. ^{27,31,96–98} One possible interpretation is that those who internalize negative racial group attitudes may be more likely to perceive that experiences of discrimination against the target group are deserved. Conversely, among those with a pro-black bias, interpreting adverse experiences as being racially motivated may have self-protective properties by deflecting from personal deficiencies and through attribution of blame to external factors. ^{99,100}

The finding of a significant interaction between racial discrimination and implicit racial bias is supportive of theories of minority stress and discrimination, which highlight both personally mediated and internalized forms of racism as risk factors for poor health. 7,9,10 Results are also in accordance with frameworks that integrate the role of racial identity as a moderator of the effect of racial discrimination.²⁸ Dimensions of racial identity have been found to play a key role in whether individuals interpret negative experiences as being racially motivated, and subsequently also influence the extent to which selfreports of racial discrimination are appraised as being stressful. 101,102 Among those with an anti-black bias, racial discrimination may represent an additional source of threat; whereas among those with a pro-black bias, reports of racial discrimination may reflect a greater awareness of issues of systemic social inequality and may not necessarily be perceived as sources of stress.

In addition to these main findings, this study contributes methodologic and technical knowledge, and evidence for other substantive associations. This study assayed LTL from DBS, which future research using community samples may employ as a minimally invasive alternative to venipuncture. DBS can be readily collected in non-clinical settings and can facilitate studies including biomarkers of health. Results also contribute to evidence indicating that socioeconomic factors affect biological aging, sextending research in this area by showing that poverty is associated with LTL in a sample exclusively of African-American men. Endemic socioeconomic disparities experienced by African Americans may also contribute to LTL shortening in this population.

The cross-sectional design of this study limits inferences regarding the causal direction of associations. For example, it is possible that worse health associated with shorter LTL could result in greater perceptions of racial discrimination. Although a number of socioeconomic and health-related covariates were controlled for in analytic models, there are additional unmeasured constructs (e.g., neighborhood factors) such that alternative explanations cannot be discounted. Possible analytic concerns include those related to sample size and the selection and operationalization of covariates, which may

have resulted in spurious findings. The purposive sampling of African-American men from the San Francisco Bay Area also limits the generalizability of findings to the broader community in this area, as the characteristics of participants differed from those of the underlying population. Generalizability to other groups is also limited, including to women, the elderly, and those residing in other geographic regions. Accordingly, replication of these findings in other samples is warranted.

Despite these caveats, results are suggestive of potential pathways that have not been previously examined, and offer new directions for measurement and research on the health of African-American men. This study merges perspectives from different disciplines and demonstrates the importance of integrating research on in-group racial bias in studies of racial discrimination and health. Incorporating this typically understudied aspect of racial minority stress can further elucidate reasons for pervasive racial disparities in aging and the disproportionately greater disease burden faced by African-American men. Findings suggest that racial discrimination in concert with the internalization of racial bias has pernicious effects on biological aging, and that this is one pathway through which social inequities generate greater disease vulnerability in this population.

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References

- Hayward MD, Heron M. Racial inequality in active life among adult Americans. Demography 1999;36:77–91.
- Rogers RG, Hummer RA, Nam CB. Living and dying in the USA: behavioral, health, and social differential in adult mortality. San Diego CA: Academic Press, 2000.
- Sorlie PD, Backlund E, Keller JB. U.S. mortality by economic, demographic, and social characteristics: the National Longitudinal Mortality Study. Am J Public Health 1995;85:949–56.
- National Center for Health Statistics. Health, U.S., 2007 with chartbook on trends in the health of Americans. Hyattsville MD, 2007.
- Palloni A, Ewbank DC. Selection processes in the study of racial and ethnic differentials in adult health and mortality. In: Anderson NB, Bulatao RA, Cohen B, eds. Critical perspectives on racial and ethnic differences in health in late life. Washington DC: National Academies Press, 2004:171–226.

- Liang J, Xu X, Bennett JM, Ye W, Quinones AR. Ethnicity and changing functional health in middle and late life: a person-centered approach. J Gerontol B Psychol Sci Soc Sci 2010;65:470–81.
- Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans. A biopsychosocial model. Am Psychol 1999;54:805–16.
- 8. Clark VR. The perilous effects of racism on blacks. Ethn Dis 2001;11: 769–72.
- 9. Jones CP. Levels of racism: a theoretic framework and a gardener's tale. Am J Public Health 2000;90:1212-5.
- Chae DH, Nuru-Jeter AM, Lincoln KD, Francis DD. Conceptualizing racial disparities in health. Du Bois Rev 2011;8:63–77.
- Broman CL. The health consequences of racial discrimination: a study of African Americans. Ethn Dis 1996;6:148–53.
- Williams DR, Neighbors HW, Jackson JS. Racial/ethnic discrimination and health: findings from community studies. Am J Public Health 2008;98:S29–S37.
- Williams DR, John DA, Oyserman D, Sonnega J, Mohammed SA, Jackson JS. Research on discrimination and health: an exploratory study of unresolved conceptual and measurement issues. Am J Public Health 2012;102:975–8.
- Ong AD, Fuller-Rowell T, Burrow AL. Racial discrimination and the stress process. J Pers Soc Psychol 2009;96:1259–71.
- Mays VM, Cochran SD, Race Barnes NW. race-based discrimination, and health outcomes among African Americans. Annu Rev Psychol 2007;58:201–25.
- Krieger N, Kosheleva A, Waterman PD, Chen JT, Koenen K. Racial discrimination, psychological distress, and self-rated health among U.S.-born and foreign-born black Americans. Am J Public Health 2011;101:1704–13.
- Williams DR, Williams-Morris R. Racism and mental health: the African American experience. Ethn Health 2000;5:243–68.
- Borrell LN, Jacobs DR Jr, Williams DR, Pletcher MJ, Houston TK, Kiefe CI. Self-reported racial discrimination and substance use in the Coronary Artery Risk Development in Adults Study. Am J Epidemiol 2007;166:1068–79.
- Minior T, Galea S, Stuber J, Ahern J, Ompad D. Racial differences in discrimination experiences and responses among minority substance users. Ethn Dis 2003;13:521–7.
- Martin JK, Tuch SA, Roman PM. Problem drinking patterns among African Americans: the impacts of reports of discrimination, perceptions of prejudice, and "risky" coping strategies. J Health Soc Behav 2003;44:408–25.
- Harrell JP, Hall S, Taliaferro J. Physiological responses to racism and discrimination: an assessment of the evidence. Am J Public Health 2003;93:243–8.
- Cooper DC, Mills PJ, Bardwell WA, Ziegler MG, Dimsdale JE. The
 effects of ethnic discrimination and socioeconomic status on endothelin1 among blacks and whites. Am J Hypertens 2009;22:698–704.
- 23. Green TL, Darity WA Jr. Under the skin: using theories from biology and the social sciences to explore the mechanisms behind the blackwhite health gap. Am J Public Health 2010;100(S1):S36–S40.
- Lewis TT, Aiello AE, Leurgans S, Kelly J, Barnes LL. Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African-American adults. Brain Behav Immun 2010;24:438–43.
- Chae DH, Nuru-Jeter AM, Lincoln KD, Jacob Arriola KR. Racial discrimination, mood disorders, and cardiovascular disease among black Americans. Ann Epidemiol 2012;22:104–11.
- 26. Chae DH, Lincoln KD, Adler NE, Syme SL. Do experiences of racial discrimination predict cardiovascular disease among African American men? The moderating role of internalized negative racial group attitudes. Soc Sci Med 2010;71:1182–8.
- Sellers RM, Shelton JN. The role of racial identity in perceived racial discrimination. J Pers Soc Psychol 2003;84:1079–92.

- Tull ES, Sheu YT, Butler C, Cornelious K. Relationships between perceived stress, coping behavior and cortisol secretion in women with high and low levels of internalized racism. J Natl Med Assoc 2005;97:206–12.
- Torres A, Bowens L. Correlation between the internalization theme of racial identity attitude survey-B and systolic blood pressure. Ethn Dis 2000;10:375–83.
- Scott LD. The relation of racial identity and racial socialization to coping with discrimination among African American adolescents. J Black Stud 2003;33:520–38.
- Sellers RM, Caldwell CH, Schmeelk-Cone KH, Zimmerman MA. Racial identity, racial discrimination, perceived stress, and psychological distress among African American young adults. J Health Soc Behav 2003;44:302–17.
- **32.** Carter RT, Reynolds AL. Race-related stress, racial identity status attitudes, and emotional reactions of black Americans. Cultur Divers Ethnic Minor Psychol 2011;17:156–62.
- Yip T, Seaton EK, Sellers RM. African American racial identity across the lifespan: identity status, identity content, and depressive symptoms. Child Dev 2006;77:1504–17.
- 34. Fuller-Rowell TE, Cogburn CD, Brodish AB, Peck SC, Malanchuk O, Eccles JS. Racial discrimination and substance use: longitudinal associations and identity moderators. J Behav Med 2011(epub ahead of print PMID: 22113318).
- Aubert G, Lansdorp PM. Telomeres and aging. Physiol Rev 2008;88: 557–79.
- Calado RT, Young NS. Telomere diseases. N Engl J Med 2009;361: 2353–65.
- Monaghan P. Telomeres and life histories: the long and the short of it. Ann N Y Acad Sci 2010;1206:130–42.
- Riethman H. Human telomere structure and biology. Annu Rev Genomics Hum Genet 2008;9:1–19.
- Mason M, Schuller A, Skordalakes E. Telomerase structure function. Curr Opin Struct Biol 2011;21:92–100.
- 40. Kaszubowska L. Telomere shortening and ageing of the immune system. J Physiol Pharmacol 2008;59(S9):169–86.
- Koppelstaetter C, Kern G, Mayer G. Biomarkers of aging with prognostic and predictive value in non-oncological diseases. Curr Med Chem 2009;16:3469–75.
- 42. Saliques S, Zeller M, Lorin J, et al. Telomere length and cardiovascular disease. Arch Cardiovasc Dis 2010;103:454–9.
- 43. Goronzy JJ, Shao L, Weyand CM. Immune aging and rheumatoid arthritis. Rheum Dis Clin North Am 2010;36:297–310.
- 44. Georgin-Lavialle S, Aouba A, Mouthon L, et al. The telomere/ telomerase system in autoimmune and systemic immune-mediated diseases. Autoimmun Rev 2010;9:646–51.
- 45. Valdes AM, Deary IJ, Gardner J, et al. Leukocyte telomere length is associated with cognitive performance in healthy women. Neurobiol Aging 2010;31:986–92.
- **46.** Huzen J, de Boer RA, van Veldhuisen DJ, van Gilst WH, van der Harst P. The emerging role of telomere biology in cardiovascular disease. Front Biosci (Landmark Ed) 2010;15:35–45.
- 47. Epel ES. Psychological and metabolic stress: a recipe for accelerated cellular aging? Hormones (Athens) 2009;8:7–22.
- Gilley D, Herbert BS, Huda N, Tanaka H, Reed T. Factors impacting human telomere homeostasis and age-related disease. Mech Ageing Dev 2008:129:27–34.
- Effros RB. Telomere/telomerase dynamics within the human immune system: effect of chronic infection and stress. Exp Gerontol 2011;46:135–40.
- Wolkowitz OM, Epel ES, Reus VI, Mellon SH. Depression gets old fast: do stress and depression accelerate cell aging? Depress Anxiety 2010;27:327–38.
- Wolkowitz OM, Mellon SH, Epel ES, et al. Leukocyte telomere length in major depression: correlations with chronicity, inflammation and oxidative stress—preliminary findings. PLoS One 2011;6:e17837.

- 52. Bauer ME, Jeckel CM, Luz C. The role of stress factors during aging of the immune system. Ann N Y Acad Sci 2009;1153:139–52.
- 53. Kiecolt-Glaser JK, Gouin JP, Weng NP, Malarkey WB, Beversdorf DQ, Glaser R. Childhood adversity heightens the impact of later-life caregiving stress on telomere length and inflammation. Psychosom Med 2011;73:16–22.
- 54. Epel ES, Blackburn EH, Lin J, et al. Accelerated telomere shortening in response to life stress. Proc Natl Acad Sci U S A 2004;101:17312–5.
- 55. Aviv A, Chen W, Gardner JP, et al. Leukocyte telomere dynamics: longitudinal findings among young adults in the Bogalusa Heart Study. Am J Epidemiol 2009;169:323–9.
- 56. Geronimus AT, Hicken MT, Pearson JA, Seashols SJ, Brown KL, Cruz TD. Do U.S. black women experience stress-related accelerated biological aging? A novel theory and first population-based test of black-white differences in telomere length. Hum Nat 2010;21:19–38.
- Zhu H, Wang X, Gutin B, et al. Leukocyte telomere length in healthy Caucasian and African-American adolescents: relationships with race, sex, adiposity, adipokines, and physical activity. J Pediatr 2011;158:215–20.
- 58. Hunt SC, Chen W, Gardner JP, et al. Leukocyte telomeres are longer in African Americans than in whites: the National Heart, Lung, and Blood Institute Family Heart Study and the Bogalusa Heart Study. Aging Cell 2008;7:451–8.
- Fitzpatrick AL, Kronmal RA, Kimura M, et al. Leukocyte telomere length and mortality in the Cardiovascular Health Study. J Gerontol A Biol Sci Med Sci 2011;66:421–9.
- Roux AVD, Ranjit N, Jenny NS, et al. Race/ethnicity and telomere length in the Multi-Ethnic Study of Atherosclerosis. Aging Cell 2009;8:251–7.
- Geronimus AT, Hicken M, Keene D, Bound J. "Weathering" and age patterns of allostatic load scores among blacks and whites in the U.S. Am J Public Health 2006;96:826–33.
- **62.** Greenwald AG, McGhee DE, Schwartz JL. Measuring individual differences in implicit cognition: the implicit association test. J Pers Soc Psychol 1998;74:1464–80.
- Greenwald AG, Poehlman TA, Uhlmann EL, Banaji MR. Understanding and using the Implicit Association Test: III. Meta-analysis of predictive validity. J Pers Soc Psychol 2009;97:17–41.
- Nosek BA, Greenwald AG, Banaji MR. Understanding and using the Implicit Association Test: II. Method variables and construct validity. Pers Soc Psychol Bull 2005;31:166–80.
- 65. McDade TW, Williams S, Snodgrass JJ. What a drop can do: dried blood spots as a minimally invasive method for integrating biomarkers into population-based research. Demography 2007;44: 899–925.
- **66.** Cawthon RM. Telomere measurement by quantitative PCR. Nucleic Acids Res 2002;30:e47.
- 67. Lin J, Epel E, Cheon J, et al. Analyses and comparisons of telomerase activity and telomere length in human T and B cells: insights for epidemiology of telomere maintenance. J Immunol Methods 2010;352:71–80.
- **68.** O'Donovan A, Epel E, Lin J, et al. Childhood trauma associated with short leukocyte telomere length in posttraumatic stress disorder. Biol Psychiatry 2011;70:465–71.
- Epel ES, Puterman E, Lin J, Blackburn E, Lazaro A, Mendes WB. Wandering minds and aging cells. Clin Psychol Sci 2013;1:75–83.
- Zanet DL, Saberi S, Oliveira L, Sattha B, Gadawski I, Cote HC. Blood and dried blood spot telomere length measurement by qPCR: assay considerations. PLoS One 2013;8:e57787.
- Kroenke CH, Pletcher MJ, Lin J, et al. Telomerase, telomere length, and coronary artery calcium in black and white men in the CARDIA study. Atherosclerosis 2012;220:506–12.
- McConnell AR, Leibold JM. Relations among the Implicit Association Test, discriminatory behavior, and explicit measures of racial attitudes. J Exp Soc Psychol 2001;37:435–42.

- Blair IV, Steiner JF, Fairclough DL, et al. Clinicians' implicit ethnic/ racial bias and perceptions of care among black and Latino patients. Ann Fam Med 2013;11:43–52.
- Sabin J, Nosek BA, Greenwald A, Rivara FP. Physicians' implicit and explicit attitudes about race by MD race, ethnicity, and gender. J Health Care Poor Underserved 2009;20:896–913.
- Mendes WB, Gray HM, Mendoza-Denton R, Major B, Epel ES. Why
 egalitarianism might be good for your health: physiological thriving
 during stressful intergroup encounters. Psychol Sci 2007;18:991–8.
- Richeson JA, Shelton JN. When prejudice does not pay: effects of interracial contact on executive function. Psychol Sci 2003;14:287–90.
- Richeson JA, Trawalter S, Shelton JN. African Americans' implicit racial attitudes and the depletion of executive function after interracial interactions. Soc Cognition 2005;23:336–52.
- Chae DH, Nuru-Jeter AM, Adler NE. Implicit racial bias as a moderator of the association between racial discrimination and hypertension: a study of Midlife African American men. Psychosom Med 2012;74:961–4.
- Krieger N, Smith K, Naishadham D, Hartman C, Barbeau EM. Experiences of discrimination: validity and reliability of a self-report measure for population health research on racism and health. Soc Sci Med 2005;61:1576–96.
- Krieger N, Waterman PD, Kosheleva A, et al. Exposing racial discrimination: implicit & explicit measures—the My Body, My Story study of 1005 U.S.-born black & white community health center members. PLoS One 2011;6:e27636.
- U.S. Census Bureau. Poverty thresholds by size of family and number of children. www.census.gov/hhes/www/poverty/data/threshld/index.html.
- **82.** CDC. Indicators for chronic disease surveillance. MMWR Recomm Rep 2004;53:19–32.
- 83. CDC. National Health and Nutrition Examination Survey (NHANES): Anthropometry Procedures Manual, 2007.
- 84. Gelman A, Fagan J, Kiss A. An analysis of the New York City Police Department's "Stop-and-Frisk" policy in the context of claims of racial bias. J Am Stat Assoc 2007;102:813–23.
- 85. Stolzenberg L, D'Alessio SJ, Eitle D. Race and cumulative discrimination in the prosecution of criminal defendants. Race Justice 2013;3:275–99.
- **86.** Malek SK, Keys BJ, Kumar S, Milford E, Tullius SG. Racial and ethnic disparities in kidney transplantation. Transpl Int 2011;24:419–24.
- 87. Smedley BD, Stith AY, Nelson AR. Unequal treatment: confronting racial and ethnic disparities in health care [with CD]. Washington DC: National Academies Press, 2009.
- 88. Pager D, Shepherd H. The sociology of discrimination: racial discrimination in employment, housing, credit, and consumer markets. Annu Rev Sociol 2008;34:181–209.
- 89. Mendez DD, Hogan VK, Culhane J. Institutional racism and pregnancy health: using Home Mortgage Disclosure act data to develop an index for mortgage discrimination at the community level. Public Health Rep 2011;126(S3):102–14.
- Kau J, Keenan D, Munneke H. Racial discrimination and mortgage lending. J Real Estate Finan Econ 2012;45:289–304.
- 91. Dovidio JF, Gaertner SL. Aversive racism and selection decisions: 1989 and 1999. Psychol Sci 2000;11:315–9.
- Bertrand M, Mullainathan S. Are Emily and Greg more employable than Lakisha and Jamal? A field experiment on labor market discrimination. Cambridge: National Bureau of Economic Research, 2003. NBER Working Paper No.: 9873.
- 93. Williams DR, Yan Y, Jackson JS, Anderson NB. Racial differences in physical and mental health: socio-economic status, stress and discrimination. J Health Psychol 1997;2:335–51.
- 94. Heard E, Whitfield KE, Edwards CL, Bruce MA, Beech BM. Mediating effects of social support on the relationship among perceived stress, depression, and hypertension in African Americans. J Natl Med Assoc 2011;103:116–22.

- Branscombe NR, Schmitt MT, Harvey RD. Perceiving pervasive discrimination among African Americans: implications for group identification and well-being. J Pers Soc Psychol 1999;77:135.
- Livingston RW. The role of perceived negativity in the moderation of African Americans' implicit and explicit racial attitudes. J Exp Soc Psychol 2002;38:405–13.
- 97. Chae DH, Walters KL. Racial discrimination and racial identity attitudes in relation to self-rated health and physical pain and impairment among two-spirit American Indians/Alaska Natives. Am J Public Health 2009;99(S1):S144–S151.
- 98. Chae DH, Lincoln KD, Jackson JS. Discrimination, attribution, and racial group identification: implications for psychological distress among black Americans in the National Survey of American Life (2001–2003). Am J Orthopsychiatry 2011;81:498–506.
- 99. Neighbors HW, Jackson JS, Broman C, Thompson E. Racism and the mental health of African Americans: the role of self and system blame. Ethn Dis 1996;6:167–75.
- Crocker J, Major B. Social stigma and self-esteem: the self-protective properties of stigma. Psychol Rev 1989;96:608.
- 101. Bennett GG, Merritt MM, Edwards CL, Sollers JJ. Perceived racism and affective responses to ambiguous interpersonal interactions among African American men. Am Behav Sci 2004;47:963–76.

- 102. Fischer AR, Shaw CM. African Americans' mental health and perceptions of racist discrimination: the moderating effects of racial socialization experiences and self-esteem. J Couns Psychol 1999;46: 395.
- 103. Shiels PG, McGlynn LM, MacIntyre A, et al. Accelerated telomere attrition is associated with relative household income, diet and inflammation in the pSoBid cohort. PLoS One 2011;6: e22521.
- 104. Cherkas LF, Aviv A, Valdes AM, et al. The effects of social status on biological aging as measured by white-blood-cell telomere length. Aging Cell 2006;5:361–5.
- 105. Adler N, Pantell MS, O'Donovan A, et al. Educational attainment and late life telomere length in the Health, Aging and Body Composition Study. Brain Behav Immun 2013;27:15–21.

Appendix

Supplementary data

Supplementary data associated with this article can be found at http://dx.doi.org/10.1016/j.amepre.2013.10.020.

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