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Discrimination, Mental Health, and Leukocyte Telomere Length Among African American Men

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Abstract

African American men in the US experience disparities across multiple health outcomes. A common mechanism underlying premature declines in health may be accelerated biological aging, as reflected by leukocyte telomere length (LTL). Racial discrimination, a qualitatively unique source of social stress reported by African American men, in tandem with poor mental health, may negatively impact LTL in this population. The current study examined cross-sectional associations

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Contributors

David H. Chae conceptualized the study, conducted data analyses, and held primary responsibility for data interpretation and writing. David H. Chae and Amani M. Nuru-Jeter collected the data. Jue Lin and Elizabeth H. Blackburn conducted the telomere length assays. All authors contributed to conceptualizing the study, interpreting the findings, and developing the manuscript, and have approved of the final version of this manuscript.

Conflict of Interest

JL is a consultant for Telomere Diagnostics, a company related to telomere biology; ESE and EHB were past consultants. No other financial disclosures were reported by the authors of this paper.

between LTL, self-reported racial discrimination, and symptoms of depression and anxiety among 92 African American men 30–50 years of age. LTL was measured in kilobase pairs using quantitative polymerase chain reaction assay. Controlling for sociodemographic factors, greater anxiety symptoms were associated with shorter LTL (b=−0.029, standard error [SE]=0.014; p<0.05). There were no main effects of racial discrimination or depressive symptoms on LTL, but we found evidence for a significant interaction between the two (b=0.011, SE=0.005; p<0.05). Racial discrimination was associated with shorter LTL among those with lower levels of depressive symptoms. Findings from this study highlight the role of social stressors and individual-level psychological factors for physiologic deterioration among African American men. Consistent with research on other populations, greater anxiety may reflect elevated stress associated with shorter LTL. Racial discrimination may represent an additional source of social stress among African American men that has detrimental consequences for cellular aging among those with lower levels of depression.

Keywords

African American men; leukocyte telomere length; racial discrimination; depression; anxiety

1. Introduction

Racial disparities in health are well-documented and represent a serious public health concern in the US (National Center for Health Statistics, 2007). African Americans have an overall life expectancy of 73.6 years compared to 78.4 years for Whites. At age 65, African Americans can expect to die 1.2 years sooner compared to their White counterparts. Among those between 45–64 years of age, death rates from cardiovascular diseases are approximately twice as high for African Americans than for Whites. African Americans have more than twice the prevalence of diabetes (19.9% vs. 9.2%), suffer higher rates of diabetes-related complications and mortality, and experience earlier onset compared to Whites (Peek et al., 2007). African Americans also experience aging-related disability, functional impairment, and cognitive declines at earlier ages (National Center for Health Statistics, 2007). These data indicate that African Americans are at greater risk for leading causes of death and disability, experience aging-related diseases earlier in life and accelerated disease progression, accompanied by greater severity and worse consequences of disease.

A common thread underlying racial disparities across multiple health outcomes may be accelerated aging at the biological level (Calado and Young, 2009). There has been emerging interest in telomeres, repetitive sequences of DNA capping the ends of chromosomes that generally shorten with increasing chronological age. Telomere length may provide insight on aging at the cellular level, which has been associated with physiologic deterioration resulting from heightened inflammation and oxidative stress (Monaghan, 2010). Shorter telomere length has been associated with several diseases, including cardiovascular diseases such as atherosclerosis, metabolic syndrome, osteoporosis, osteoarthritis, cognitive declines and dementias including Alzheimer’s disease, in addition to mortality. Leukocyte telomere length (LTL) in particular has been posited as an indicator of general systemic aging of the

organism; specifically, short LTL may be a risk factor for a number of aging- and stress-related diseases (Andrews et al., 2010; Zhu et al., 2011).

Importantly, studies have found that LTL may exhibit accelerated shortening due not only to physiological but also psychosocial stress, and therefore may be one pathway to generating racial disparities in health. One study reported that African Americans have shorter LTL compared to Whites (Fitzpatrick et al., 2011). Others have found that whereas African Americans may initially have longer LTL, their rates of LTL shortening may be faster compared to Whites. For example, a cross-sectional study found a significant interaction between race and chronological age in predicting LTL, with a steeper inverse association between chronological age and LTL for African Americans compared to Whites (Hunt et al., 2008). Similarly, longitudinal studies have found that African Americans had a faster rate of LTL shortening compared to Whites (Diez Roux et al., 2009; Rewak et al., 2014). Racial differences in telomere trajectories may result from environmentally-induced and stress-related factors impacting LTL.

Along these lines, qualitatively unique psychosocial stressors that are particularly salient among African Americans may lead to accelerated LTL shortening in this population. Among these are experiences of racially-motivated discrimination. African American men in particular are susceptible to racial discrimination and prejudice in multiple domains. As highlighted in recent media reports and documented in research studies, African American men are disproportionately impacted by legally sanctioned forms of criminal profiling, and also receive harsher punishments in judicial contexts compared to their White counterparts after controlling for criminal history (Gelman et al., 2007; Stolzenberg et al., 2013). Companies have been found to improperly use background checks to exclude racial minority job applicants, and experimental studies have shown that prospective employers are less responsive to applications with “African American” compared to “White” names (Bertrand and Mullainathan, 2003; Dovidio and Gaertner, 2000). In addition to these more acute experiences of racial discrimination, African American men report high levels of everyday forms of unfair treatment, including instances of being treated with less courtesy and respect, or being perceived as less intelligent or being feared (Williams et al., 1997).

As a source of psychosocial stress, racial discrimination may exact physiologic tolls (Clark et al., 1999). However, findings on racial discrimination and physical health outcomes have been equivocal. For example, one study reported an inverse association between reports of racial discrimination and coronary artery calcification (Everage et al., 2012); another found an inverse association with hypertension among African American men, albeit non-significant (Roberts et al., 2008). An earlier study reported a U-shaped association, with working-class African Americans reporting no racial discrimination having the highest blood pressure (Krieger and Sidney, 1996). Other studies have reported null associations. A systematic review found that while there are stronger associations with mental and behavioral outcomes, almost two-thirds of studies examining racial discrimination in relation to physical health outcomes found no significant association (Paradies, 2006).

To explain these counterintuitive findings, other studies have suggested that the relationship between racial discrimination and health outcomes may be more complex, evincing

interactive rather than direct relationships. The health implications of racial discrimination may be contingent on individual-level appraisals and psychological responses to the event. When perceived to be a social evaluative threat, racial discrimination may result in anxiety, engaging biochemical mechanisms associated with greater physiologic reactivity (Bosch et al., 2009; Dickerson et al., 2009). Indeed, studies have found evidence for associations with inflammation and other biomarkers of health (Friedman et al., 2009; Lewis et al., 2010; Szanton et al., 2012). Racial discrimination may also result in poorer self-concept and negative affective responses (Williams and Williams-Morris, 2000). These psychological factors have been shown to have biological consequences resulting in greater disease risk (Hansel et al., 2010; Steptoe et al., 2013). Accordingly, the association between self-reported racial discrimination and LTL may vary according to whether individuals exhibit negative mental health symptoms. For example, one study found that African Americans with a history of mood disorder and who reported high levels of racial discrimination were at greatest risk for cardiovascular disease (Chae et al., 2012). Studies have also found that higher levels of anxiety are associated with shorter LTL (Hoen et al., 2013; Shalev et al., 2014). Findings on depression and LTL have been mixed, however, with some studies finding that they are inversely related, and others finding no association (Hartmann et al., 2010; Hoen et al., 2011; Needham et al., 2014; Verhoeven et al., 2014).

The purpose of the current investigation was to examine whether symptoms of depression and anxiety are associated with LTL, and if these psychological factors moderate the association between racial discrimination and LTL in a sample of African American men. We hypothesized that poorer mental health as indexed by symptoms of depression and anxiety would be associated with shorter LTL; and that racial discrimination would be associated with shorter LTL among participants reporting higher levels of depression and anxiety.

2. Method

2.1. Sample and Procedures

Data were from this study were from the Bay Area Heart Health Study, a cross-sectional study of African American men predominantly in midlife residing in the San Francisco Bay area. A total of 95 participants were recruited between February 2010 and May 2010 from community outlets, through individual outreach, self-referral from posted advertisements, and referral from other participants. Venues included barbershops, churches, community events, and coffee shops. Eligibility criteria were: self-identification as an African American man; age between 30–50 years; US and parental US nativity; ability to read, write, and understand English; and absence of serious or unstable disease (e.g., cancer, HIV, hepatitis, tuberculosis).

All assessments were conducted by a trained research assistant in a private room located at a nonclinical setting (e.g., church or university room). Basic demographic questions were asked through a brief face-to-face interview, followed by a minimally invasive physical examination, which included the collection of dried blood spots (DBS). Four blood drops from finger prick, each approximately 50 μL, were placed on filter paper, allowed to dry, and stored at −80°C. Computer-assisted self-interview was used to administer more sensitive

questions, including measures of socioeconomic position, mental health, and experiences of racial discrimination.

All participants provided informed consent and were compensated with a $70.00 gift card. The University of California, San Francisco Committee on Human Research approved all study procedures and protocols.

2.2 Measures

Leukocyte telomere length—Genomic DNA was extracted from DBS using a QIAamp DNA Investigator Kit (QIAGEN Cat# 56504). Average yield was 56 ng (range 12 ng–340 ng). LTL was measured by quantitative PCR adapted from published methods (Cawthon, 2002; Lin et al., 2010) and expressed as the ratio of telomere-to-a single copy gene (T/S) (Lin et al., 2010). To convert the T/S ratios to base pairs, the T/S ratios of a set of genomic DNA samples from the human fibroblast primary cell line IMR90 at different population doublings, as well as with the telomerase protein subunit gene (hTERT) transfected into a lentiviral construct were determined. The mean telomere restriction fragment (TRF) length from these DNA samples was determined using Southern blot analysis, and the slope of the plot of mean TRF length versus T/S for these samples served as the conversion factor for calculation of telomere length in base pairs from the T/S ratio. The resulting equation for conversion from T/S ratio to base pairs is: base pairs= 3274 + 2413*(T/S).

Telomere length was assayed twice for each sample, each time using half of the obtained DNA (average coefficient of variation 6.3%). We examined LTL in kilobase pairs (kb). Previous studies have found high correlations between telomere length obtained from DBS and both whole blood and peripheral mononuclear blood cells (Zanet et al., 2013).

Racial Discrimination—Racial discrimination was assessed using the 9-item Experiences of Discrimination (EOD) measure, which includes an index of racial discrimination experiences ever experienced: at school; getting a job; at work; getting housing; getting medical care; at a store or restaurant; getting credit or loans; on the street or public settings; and from the police or in courts (Krieger et al., 2005). The total number of situations in which racial discrimination was experienced ranges from 0 to 9. The EOD is a widely used and validated measure of racial discrimination.

Mental health—Depressive symptomology was measured using the 10-item Centers for Epidemiology Studies Depression Scale (CES-D), which assesses affective, cognitive, behavioral, and somatic symptoms of depression during the past week (Kohout et al., 1993). Each item was measured on a four point Likert scale (0 = rarely or none of the time, 3 = all of the time). Positively valenced items were reverse coded and scores were summed such that higher scores reflect greater depressive symptomatology ($\alpha = 0.75$).

The 7-item anxiety subscale of the Hospital Anxiety and Depression Scale (HADS) was used to measure symptoms of anxiety (Zigmond and Snaith, 1983). Items assess feelings of being worried, tense, and fearful, as well as restlessness and panic in the past. Scores for each item ranged from 0 (not at all) to 3 (most of the time), with higher scores reflecting greater levels of anxiety ($\alpha = 0.81$).

Covariates—Covariates included age in years; ratio of household income to the poverty threshold based on household composition; educational attainment (high school graduate or less vs. some college or more); employment status (currently working or unemployed); current smoking defined as smoking at least 100 lifetime cigarettes and self-reported current smoking at least "some days" (Centers for Disease Control and Prevention, 2004); number of health conditions assessed using a checklist of common diseases (e.g., cardiovascular diseases, diabetes, cancer, renal disease); and current doctor-prescribed medication use. We also assessed social desirability bias using the short-version Marlowe-Crowne Social Desirability Scale, which includes 10 true-false items assessing personality factors that have been shown to influence responses to sensitive items (Reynolds, 1982). Responses reflecting a tendency to over-report positive or under-report negative attributes are scored with a value of 1, with greater total scores reflecting high levels of social desirability bias.

2.3. Analyses

Two outlying LTL values of 4.00 kb and 6.91 kb (3.56 time below and 3.18 times above the standard deviation [SD], respectively) were excluded from analyses. Regression diagnostics (studentized residuals, leverage, Cook's D, and DFITS) consistently revealed one influential observation, which was also excluded from analyses. Including this observation reduced the magnitude of associations but resulted in substantively similar conclusions. As a result, the analytic sample consisted of a total of 92 participants.

There were three participants with missing data on either one or two items of the CES-D. The within-subject mean of items with complete data was used to substitute missing values for these participants. Mean substitution in cases where at least 80% of items are completed is considered to be a robust technique for handling missing data on multi-item scales (Roth et al., 1999).

Bivariate correlations for continuous independent variables and T-tests for dichotomous categorical independent variables were used to examine associations with LTL. Ordinary least squares regression models examining racial discrimination, depression, and anxiety in relation to LTL were specified. Interactions between racial discrimination and both depression and anxiety were examined to test whether the association between racial discrimination and LTL varied by mental health factors. Component variables of interactions terms were centered in multivariable analyses (Cohen et al., 2003). We tested for multicollinearity in all models, particularly between depression and anxiety, which showed the strongest correlation. We tested these variables individually in multivariable models and found no evidence of suppression effects. Variance inflation factors (VIF) for all variables, which ranged from 1.092 to 2.626, were all in the acceptable range. All analyses were conducted using SAS version 9.3 (Cary, NC).

3. Results

The mean LTL in our sample was 5.54 kb (SD = 0.38), consistent with what was reported in a recent study on a racially diverse sample (Geronimus et al., 2015). Only six participants (6.5%) reported no experiences of racial discrimination. Nine participants (9.8%) reported discrimination in one or two situations, and 26 (28.3%) reported discrimination in three to

five situations. Most participants reported experiencing racial discrimination in six or more situations (n = 51, 55.4%). Most commonly reported was racial discrimination by police or in the courts (n = 79, 85.9%), followed by discrimination in getting a job (n = 67, 72.8%). Mean levels of depression and anxiety were 7.5 (SD = 4.9) and 5.0 (SD = 3.9) respectively. Additional descriptive characteristics of our sample are shown in Table 1.

Inter-correlations between study variables are shown in Table 2. There was no significant bivariate relationship between racial discrimination and LTL ($r = -0.098$). Neither depression ($r = 0.004$) nor anxiety ($r = -0.138$) had significant bivariate correlations with LTL. Increasing age was associated with shorter LTL ($r = -0.357$, $p<0.001$) and greater ratio of household income to poverty was associated with longer LTL ($r = 0.332$, $p< 0.01$). Greater levels of social desirability bias were associated with shorter LTL ($r = -0.213$, $p< 0.05$). Those taking medications also had shorter LTL (M = 5.39) compared to those who were not ($r = -0.279$,$p< 0.01$). We did not find any significant relationships between racial discrimination and either depression ($r = -0.012$, $p = 0.913$) or anxiety ($r = 0.149$, $p = 0.157$).

Results from multivariable linear regression models are presented in Table 3. Controlling for covariates, there was no significant association between racial discrimination and LTL (Model 1: b = − 0.010, standard error [SE] = 0.014, $p = 0.46$). In a model including only depression and covariates, depression still showed no significant relationship with LTL (b = −0.002, SE = 0.008, $p = 0.763$. Examining only anxiety and covariates revealed that greater levels of anxiety were associated with shorter LTL at the trend level (b = −0.017, SE = 0.010, $p = 0.092$). When examining racial discrimination, depression, and anxiety concurrently (Model 2), the association between anxiety and LTL became significant (b = −0.029, SE = 0.014, $p< 0.05$).

Testing interactions between racial discrimination and mental health indicators (Model 3) revealed no significant interaction between racial discrimination and anxiety (b = −0.008, SE = 0.005, $p = 0.12$). However, there was a significant interaction between racial discrimination and depression predicting LTL (b = 0.011, SE = 0.005, $p = 0.020$). In this final model, significant covariates were age and medication use. Ratio of household income to poverty threshold had a marginally significant association with LTL (b = 0.037, SE = 0.019, $p = 0.05$). Concordant with this model, when examining interaction terms separately, the interaction between depression and racial discrimination approached significance at the trend level (b=0.006, SE=0.003, p=0.086), while the interaction between anxiety and racial discrimination remained non-significant (b=0.000, SE=0.004, p=0.998).

To illustrate the interaction between racial discrimination and depression, we constructed predicted values of LTL. We plotted the relationship between racial discrimination and LTL values for those with high vs. low levels of depression. We chose a value of 8 to represent participants with high levels of depression, the cut-off for probable depression based on the conventional cut-off of 16 for the full 20-item CES-D (Radloff, 1977); we used a value of 4 to represent those non-depressed, which corresponds to the median for those with scores less than 8. For all other independent variables, mean values were used for continuous variables and the proportion of total participants belonging in a group was used for categorical

variables in order to illustrate relationships for the average participant. Using alternative values did not substantively change the shape of the plot, shown in Figure 1. The relationship between racial discrimination and LTL was stronger among those non-depressed. In this group, there was an inverse association, with greater racial discrimination being associated with shorter LTL; in contrast, among those meeting the cut-off for probable depression, there was a modest positive association.

4. Discussion

Findings from this study contribute to existing research on the association between mental health and LTL. We extend this area of research by exploring these relationships in a sample of African American men. We found that greater levels of anxiety were associated with shorter LTL. However, we found no evidence for a main effect of depressive symptoms on LTL. These findings are concordant with some other studies that have found more consistent relationships with anxiety and LTL, but not depression. For example, a large population-based longitudinal study found that anxiety disorder was a significant predictor of LTL; however, depressive disorders had no relationship (Hoen et al., 2013). Large population-based studies have also reported non-significant associations between LTL and depression, particularly for less severe depressive symptoms and among older adults (Needham et al., 2014; Phillips et al., 2013; Schaakxs et al., 2014; Shaffer et al., 2012). Though both anxiety and depression have been linked with indicators of inflammation and oxidative stress – factors that have been shown to impact telomere shortening – differences in their relationship with LTL may be due to phenomenological distinctions between the two. Among the characteristics of anxiety are hyper vigilance, arousal, and fear of threat, whereas depression often manifests in negative affect and anhedonia. The psychological sequelae associated with anxiety may trigger physiologic responses that are more proximal to LTL shortening.

The current study also extends this area of research by examining racial discrimination, a qualitatively unique source of social stress disproportionately impacting African American men. While we found no evidence for a main effect of racial discrimination on LTL, we found evidence for a moderated association by levels of depression. However, contrary to what was hypothesized, racial discrimination was associated with shorter LTL only among respondents with lower levels of depressive symptomatology. On the other hand, racial discrimination was not strongly related to LTL among those meeting the cut-off for probable depression, though there was a slight positive association. As expected, we found that participants reporting low levels of racial discrimination and who had lower levels of depression had the longest LTL. Further, greater racial discrimination was associated with shorter LTL among those with lower but not higher levels of depression. This finding resonates with another study that found that while adverse childhood experiences were associated with shorter LTL among healthy controls, there was no association among those with major depressive disorder (Chen et al., 2014). One plausible explanation for this finding is that those with high levels of depressive symptoms may be less reactive to adverse social stressors such as racial discrimination. Studies have found evidence for blunted cortisol responses to stress among those with depression (Burke et al., 2005). One study found that individuals with higher levels of depressive symptoms showed lower cardiovascular

reactivity to induced psychological stress compared to those with lower levels of depression (York et al., 2007). Physiologic arousal accompanying the experience of racial discrimination may not be apparent among those with high levels of depression, and hence, may not have an association with LTL.

It should be noted that in contrast to other studies, we did not find evidence for a main effect of racial discrimination on depression in our sample (e.g., Hudson et al., 2013). However, our findings are consistent with other frameworks that posit that the harmful psychological effects of psychosocial stressors may be lessened among African Americans who respond with maladaptive health behaviors, such as smoking, alcohol use, or poor diet, which have subsequently been linked to worse physical health outcomes. There is evidence that such coping behaviors could buffer against negative psychological responses to social stressors, such as socioeconomic disadvantage (Mezuk et al., 2010). There may be a trade off between mental and physical health in this population, such that the lower prevalence of clinically significant psychiatric problems is compensated by relatively worse physiologic consequences (Jackson et al., 2010). Along these lines, shorter LTL resulting from racial discrimination may be more apparent among individuals with lower levels of depression.

Concordant with previous research on this sample, we found that greater ratio of household income to the poverty threshold was associated with longer LTL, albeit at the trend level in multivariable analyses (Chae et al., 2014). We also found, as expected, that participants currently taking prescription medications had significantly shorter LTL than those not taking any medications. These relationships were in the expected direction. In bivariate analyses, greater social desirability bias was related to shorter LTL, suggesting that personality factors reflecting the tendency to over-report positive characteristics may be associated with shorter LTL; however, this was not significant in multivariable analyses.

There are several caveats to our findings. The cross-sectional design of this study and the correlational nature of the data present limitations in making causal inferences and deducing the direction of the associations observed. For example, we could not ascertain whether shorter LTL leads to higher levels of anxiety, i.e., those with worse physical health associated with shorter LTL may exhibit greater anxiety around their conditions. It is also possible that those with worse health and who are not depressed are more likely to perceive being the victims of racial discrimination. In order to help address these alternative explanations, in our multivariable models we controlled for the number of self-reported chronic health conditions as well as medication use. However, it is possible that there are other unmeasured confounders that were not included in our analytic models, such as duration of depression or anxiety. Our study also recruited a self-selected sample of African American men primarily in midlife who are not representative of the underlying population. Accordingly, our results are not generalizable to all groups, including African American men who are younger or older, women, or those living in other geographic areas. The limited sample size may have also restricted our ability to detect meaningful associations; or alternatively, it is possible that some associations may have been driven by a small subset of participants. However, we performed a number of regression diagnostics to identify potentially influential observations and outliers. Future research may consider additional

factors that likely have an impact on LTL in this population, such as those related to neighborhood context (Geronimus et al., 2015).

Despite these limitations, our study provides a more nuanced approach to investigating salient social and psychological stressors that may be associated with LTL among African American men, and presents future avenues for research on racial disparities in health. Our study suggests that social hazards such as the experience of racial discrimination, in addition to more traditionally studied mental health factors should be examined when identifying the determinants of aging- and stress-related racial disparities in this population. Findings from our study highlight the need to consider how social stressors disproportionately impacting racial minorities in conjunction with psychological risk factors might have a deleterious impact on accelerated cellular aging among African American men.

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Highlights

- This study examined leukocyte telomere length (LTL) among African American men.
- We examined LTL in relation to racial discrimination and mental health factors.
- Anxiety is a risk factor for LTL shortening among African American men.
- Racial discrimination is related to shorter LTL among those with low depression.
- Psychosocial stressors experienced by African American men negatively impact LTL.

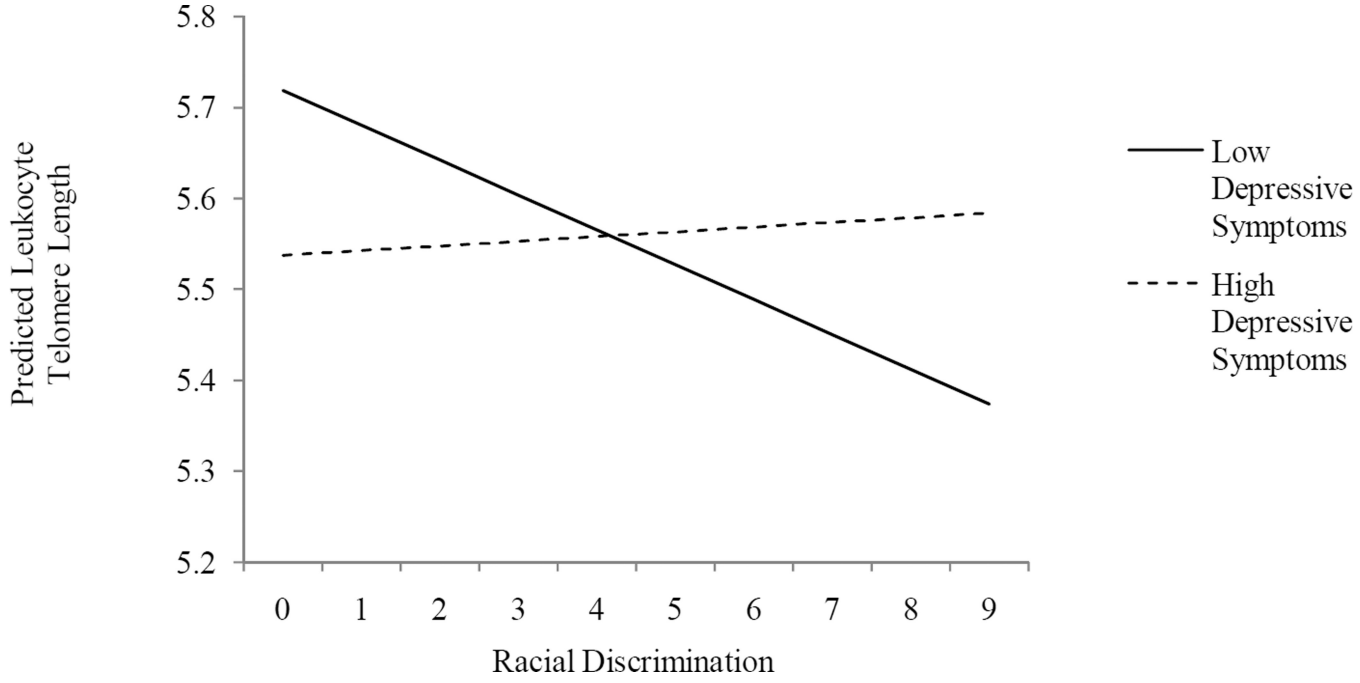

Figure 1.
Predicted leukocyte telomere length in kilobase pairs by racial discrimination and depression among African American men (n = 92).

Table 1

Descriptive characteristics of African American men (n = 92).

	Mean or n	SD or %
Leukocyte Telomere Length, Mean (SD)	5.54	(0.38)
Racial Discrimination, Mean (SD)	5.55	(2.73)
Depression, Mean (SD)	7.51	(4.86)
Anxiety, Mean (SD)	5.02	(3.86)
Age, Mean (SD)	43.86	(5.73)
Income to Poverty Ratio, Mean (SD)	1.95	(2.24)
Education, n (%)		
High School or Less	38	(41.3)
Some College or More	54	(58.7)
Work Status, n (%)		
Working	42	(45.7)
Unemployed	50	(54.4)
Smoking Status, n (%)		
Noncurrent	41	(44.6)
Current	51	(55.4)
Health Conditions, Mean (SD)	1.73	(1.89)
Medication Use, n (%)		
No	63	(68.5)
Yes	29	(31.5)
Social Desirability, Mean (SD)	6.07	(1.95)

Table 2

Inter-correlations between study variables among African American men (n=92).

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Telomere Length	−0.098	0.004	−0.138	−0.357***	0.332**	−0.038	−0.007	−0.106	−0.029	−0.279**	−0.213*
2. Discrimination	1.000	−0.012	0.149	0.109	−0.208*	0.057	−0.030	0.006	0.044	−0.225*	0.045
3. Depression		1.000	0.743***	−0.110	−0.038	−0.203	−0.199	0.113	0.332**	0.166	−0.123
4. Anxiety			1.000	−0.029	−0.122	−0.116	−0.177	0.159	0.393***	0.063	−0.082
5. Age				1.000	−0.211*	0.010	0.035	0.143	−0.014	−0.032	0.056
6. Income/Poverty Ratio					1.000	0.254*	0.353***	−0.304**	−0.029	−0.172	−0.142
7. Education						1.000	0.295**	−0.175	0.055	−0.049	0.017
8. Work Status							1.000	−0.383***	−0.144	−0.036	0.177
9. Smoking Status								1.000	0.126	0.091	−0.004
10. Health Conditions									1.000	0.309**	−0.060
11. Medication Use										1.000	0.086
12. Social Desirability											1.000

* $p<0.05$

** $p<0.01$

*** $p<0.001$

Table 3

Multivariable linear regression analyses predicting leukocyte telomere length in kilobase pairs among African American men (n = 92).

	Model 1 b (SE)	Model 2 b (SE)	Model 3 b (SE)
Intercept	6.594 (0.310) ***	6.582 (0.308) ***	6.668 (0.304) ***
Racial Discrimination	−0.010 (0.014)	−0.006 (0.014)	0.000 (0.014)
Depression		0.013 (0.011)	0.015 (0.011)
Anxiety		−0.029 (0.014) *	−0.031 (0.014) *
Discrimination × Depression			0.011 (0.005) *
Discrimination × Anxiety			−0.008 (0.005)
Age	−0.020 (0.006) **	−0.020 (0.006) **	−0.021 (0.006) ***
Income to Poverty Ratio	0.037 (0.019) †	0.033 (0.019) †	0.037 (0.019) †
Some College vs. HS or Less (ref)	−0.071 (0.076)	−0.071 (0.076)	−0.069 (0.075)
Unemployed vs. Employed (ref)	−0.023 (0.084)	−0.019 (0.083)	−0.032 (0.083)
Current vs. Nonsmoker (ref)	−0.004 (0.078)	0.009 (0.077)	−0.018 (0.077)
Health Conditions	0.011 (0.020)	0.024 (0.021)	0.016 (0.021)
Medication Use vs. None (ref)	−0.225 (0.084) **	−0.247 (0.084) **	−0.238 (0.085) **
Social Desirability	−0.025 (0.019)	−0.025 (0.018)	−0.023 (0.018)
R^2	0.296	0.331	0.377

† $p<0.10$

* $p<0.05$

** $p<0.01$

*** $p<0.001$