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Racial Discrimination and Telomere Shortening Among African Americans: The Coronary Artery Risk Development in Young Adults (CARDIA) Study

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Abstract

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Objective—Telomeres are protective sequences of DNA capping the ends of chromosomes that shorten over time. Leukocyte telomere length (LTL) is posited to reflect the replicative history of cells and general systemic aging of the organism. Chronic stress exposure leads to accelerated LTL shortening, which has been linked to increased susceptibility to and faster progression of aging-related diseases. This study examined longitudinal associations between LTL and experiences of racial discrimination, a qualitatively unique source of minority psychosocial stress, among African Americans.

Method—Data are from 391 African Americans in the Coronary Artery Risk Development in Young Adults (CARDIA) Telomere Ancillary Study. We examined the number of domains in which racial discrimination was experienced in relation to LTL collected in Years 15 and 25 (Y15: 2000/2001; Y25: 2010/2011). Multivariable linear regression examined if racial discrimination was associated with LTL. Latent change score analysis (LCS) examined changes in racial discrimination and LTL in relation to one another.

Results—Controlling for racial discrimination at Y15, multivariable linear regression analyses indicated that racial discrimination at Y25 was significantly associated with LTL at Y25. This relationship remained robust after adjusting for LTL at Y15 (b = −.019, p = .015). Consistent with this finding, LCS revealed that increases in experiences of racial discrimination were associated with faster 10-year LTL shortening (b = −.019, p = .015).

Conclusions—This study adds to evidence that racial discrimination contributes to accelerated physiologic weathering and health declines among African Americans through its impact on biological systems, including via its effects on telomere attrition.

Keywords

African Americans; racial discrimination; leukocyte telomere length

Chronic psychosocial stress contributes to physiologic weathering and premature declines in health due to the burden placed on biological systems, as indexed by health indicators including several biomarkers that reflect elevated disease risk (Geronimus, Hicken, Keene, & Bound, 2006; Harris & Schorpp, 2018; McEwen & Stellar, 1993; McEwen, 1998). One such measure that has been posited to reflect this process is telomere length. Telomeres are repetitive sequences of DNA that cap the ends of chromosomes, and have an important role in supporting chromosomal stability (Buxton et al., 2014; Mason, Schuller, & Skordalakes, 2011; Riethman, 2008). Telomere attrition occurs during cell replication, resulting in shorter length on average over time and an inverse association with chronological age (Aviv, Valdes, & Spector, 2006; Müezzinler, Zaineddin, & Brenner, 2013). Studies suggest that both psychosocial and physiologic stressors can accelerate telomere shortening (Cherkas et al., 2006; Epel et al., 2004, 2006; Simon et al., 2006). Accordingly, telomere length has been posited to be a biological marker of replicative history and a cumulative indicator of “wear and tear” at the cellular level (Geronimus et al., 2010; Monaghan, 2010). Leukocyte telomere length (LTL) in particular has emerged as a marker of immune system aging as well as general systemic aging of the organism, and has been associated with several aging-related health outcomes, including cardiovascular diseases, metabolic syndrome,

osteoporosis and osteoarthritis, cognitive decline, and mortality (Blackburn, Epel, & Lin, 2015).

Among African Americans, racism is a psychosocial stressor that contributes to well-documented disparities in health, including the incidence and severity of multiple adverse disease outcomes, which may be driven by accelerated biological aging (Chae et al., 2014; Chae, Nuru-Jeter, Lincoln, & Francis, 2011). Compared to other facets of racism, racial discrimination has been most commonly studied in relation to risk factors for poor health. Although there is some variation in how scholars define discrimination, it is often conceptualized as the direct interpersonal experience of unfair treatment because of membership in a particular social group (Krieger, 2000). Racial discrimination specifically has been studied in relation to biological precursors of clinical disease outcomes (Berger & Sarnyai, 2015; Paradies et al., 2015). Studies have found associations between racial discrimination and glucocorticoids, proinflammatory cytokines, and other markers of inflammation (Williams, 2018). One study found that everyday discrimination was associated with C-reactive protein prospectively over a 7-year period in a racially diverse sample of women, particularly among those who were nonobese (Beatty Moody, Matthews, Bromberger, & Brown, 2014). Additionally, African American adolescents reporting consistently high levels of racial discrimination over time were found to have greater allostatic load, a composite measure of multisystem dysregulation (Brody et al., 2014). Another study found significant effects of discrimination on trajectories of allostatic load among women; higher allostatic load among African American women was partially explained by greater discrimination (Upchurch et al., 2015). Recent research has found that racial discrimination may also be associated with patterns of DNA methylation indicative of epigenetic aging (Brody, Miller, Yu, Beach, & Chen, 2016). The presence of biological stress mediators, particularly those related to inflammation and oxidative stress, may lead to telomere shortening. Cellular senescence and apoptosis associated with critically short telomere length also induces an inflammatory response (Monaghan, 2010; O'Donovan et al., 2011). Accordingly, a compelling pathway through which racial discrimination may become biologically embedded is through its effects on the telomere maintenance system (Chae et al., 2014, 2011; Geronimus et al., 2010; Liu & Kawachi, 2017).

Factors tied to racism contribute to racial disparities in the onset and progression of aging-related diseases via chronic activation of the physiologic stress response over time (Chae et al., 2011; Williams & Mohammed, 2013). LTL among African Americans may in part reflect the accumulation of racism-related stress across the life course. Data from the Health and Retirement Study indicate that reporting high levels of discrimination is associated with shorter salivary telomere length specifically among African Americans but not Whites (Lee, Kim, & Neblett, 2017; Liu & Kawachi, 2017). Another study of African American midlife men reported an interactive effect between racial discrimination and implicit in-group racial bias, with those reporting high levels of racial discrimination and holding an anti-Black bias having the shortest LTL, although there was no main effect of racial discrimination (Chae et al., 2014). These and other cross-sectional studies provide suggestive evidence that racial discrimination may have detrimental consequences for telomere shortening (Beatty Moody et al., 2019; Pantesco et al., 2018).

Some studies on race and telomere length have found no racial differences in telomere length or in fact longer telomeres among African Americans compared to Whites, likely due to pressures stemming from selection bias over time (Hamad, Tuljapurkar, & Rehkopf, 2016). However, other studies have suggested that African Americans undergo a faster rate of telomere shortening compared to Whites (Brown, Needham, & Ailshire, 2017; Lynch et al., 2016). 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 among African Americans (Hunt et al., 2008). Other prospective cohort research has supported the observation that African Americans may have longer telomere length at birth but experience more rapid telomere shortening compared to Whites (Rewak et al., 2014). Collectively, this previous research suggests that identifying factors associated with telomere attrition may be particularly important for understanding biological processes underlying increasing disease vulnerability among African Americans. Longitudinal research examining changes in LTL may be particularly meaningful in elucidating the causes of worsening physiologic deterioration among African Americans and mechanisms involved in the production of racial disparities in health. The rate of LTL attrition over time may in part be informed by the embodiment of psychosocial stress. The current study is the first to our knowledge to explicitly examine changing experiences of racial discrimination in relation to LTL shortening among African Americans over time.

Method

Study Design and Sample

Data from this study are from the Coronary Artery Risk Development in Young Adults (CARDIA) Study, a prospective cohort study of African American and White adults. The study design and procedures of the CARDIA Study have been previously described in detail (Friedman et al., 1988; Hughes et al., 1987). Baseline data were first collected in 1985/1986. Briefly, participants were recruited from four metropolitan areas (Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA) using stratified random sampling procedures to obtain equal numbers of participants by race, gender, age subgroup (18–24 and 25–30 years of age), and education (less than and more than 12 years of education). The CARDIA Telomere Ancillary Study assayed banked blood specimens in a subsample who participated in Year 15 (Y15; 2000/2001) to Year 25 (Y25; 2010/2011). The current study is restricted to African American participants (n = 410).

Measures

Leukocyte telomere length—We examined LTL measured in Y15 and Y25. At each wave, the T/S ratio (number of repeats of a specific telomere sequence to a reference single-copy gene as compared with a reference DNA sample) was obtained using a quantitative polymerase chain reaction assay (Cawthon, 2002; Lin et al., 2010). In the current study, T/S ratios were converted to kilobase pairs (kbp) using the formula (3274 + (2413 × T/S)/1000; Cawthon, 2002).

Racial discrimination—Racial discrimination was assessed in Y15 and Y25 using an earlier seven-item situation version of the Experiences of Discrimination (EOD) measure

(Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005). The EOD is a widely used and validated measure of racial discrimination (Krieger et al., 2005; Paradies et al., 2015). The seven-item version used in CARDIA included an item assessing racial discrimination "at home," which is not part of the current EOD and was not included in the current analyses. The remaining six-item set asked participants whether they had "ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior" due to their "race or color" in the following contexts: (a) at school; (b) getting a job; (c) getting housing; (d) at work; (e) getting medical care; and (f) on the street or in a public setting. Three items in the EOD that were not included in the CARDIA version assessed racial discrimination in "getting service in a store or restaurant," "getting credit, bank loans, or a mortgage," and "from the police or in the courts." Consistent with other studies that have examined the EOD, in CARDIA as well as in other data sets, we examined the sum of the number of situations in which racial discrimination was experienced (Borrell et al., 2007; Chae et al., 2014; Krieger et al., 2005), which ranged from 0 to 6.

Sociodemographic variables—Demographic characteristics included gender reported at baseline (*women* = 1 vs. *men* = 0); and measured at Y15, age in years and relationship status as a categorical variable: married (referent), never married, and divorced, separated, or widowed. Socioeconomic variables measured at Y15 were education (*high school or less* = 0 vs. *more than high school* = 1), and total combined family income (1 = *less than $5,000*, 9 = *$100,000 and greater*). Health variables at Y15 were smoking status, coded as never (referent), former, and current; and body mass index (kg/m^2) calculated continuously based on height and weight measured by trained study staff.

Statistical Analysis

We examined cross-sectional relationships between racial discrimination and LTL at both Y15 and Y25 using multivariable linear regression. Cross-wave models examining LTL at Y25 included racial discrimination at both Y15 and Y25, and LTL at Y15 as predictors.

We utilized latent change score (LCS) analysis to explicitly model the change in LTL by change in racial discrimination. This approach confers several advantages: LCS allows us to index within-person changes in exposure to racial discrimination and to examine this in relation to their change in LTL; it does not conflate change scores with main effects; and also takes into account characteristics associated with change scores (de Haan, Prinzie, Sentse, & Jongerling, 2018). We specifically examined the relationship between the change in LTL and change in racial discrimination between Y15 ($t = 0$) and Y25 ($t = 1$).

The observed racial discrimination score of a participant, n, at each time point, t, is expressed by $X_{[t,\,n]}$, which is determined by the true score (latent score), $x_{[t,\,n]}$ and measurement error, $e_{[t,\,n]}$. The latent change in racial discrimination, Δx_n, is the difference in the latent racial discrimination scores between the two time points, $x_{[1,\,n]} - x_{[0,\,n]}$. Based on this equation, the latent racial discrimination score of a participant at Y25 is:

$$x_{[1,\,n]} = x_{[0,\,n]} + \Delta x_n .$$

Similarly, the latent LTL of a participant at Y25 is:

$$y_{[1,n]} = y_{[0,n]} + \Delta y_n .$$

The latent change in racial discrimination was indexed by racial discrimination at Y15, demographic, socioeconomic, and health characteristics, and the number of years elapsed between waves. The latent change in LTL was indexed by the latent change in racial discrimination, adjusting for LTL and racial discrimination at Y15, and demographic, socioeconomic, and health characteristics, as well as the number of years elapsed between waves.

Ten participants were excluded because they did not have valid LTL in either Y15 or Y25; nine additional participants with outlying LTL values (greater than three standard deviations from the mean) were not included in analyses, resulting in a total analytic sample size of n = 391. This sample size has been shown to be sufficient to detect meaningful effect sizes associated with racial discrimination in multivariable linear regression analyses of LTL (Chae et al., 2014, 2016). The current sample size also meets the minimum recommendation of 200 for structural equation modeling (Kline, 2011).

All analyses were conducted using Mplus 7.4 (Muthén & Muthén, 1998–2015). Continuous covariates were centered (age, income, BMI). All covariates were declared as endogenous variables by estimating their variances in the MODEL command (Muthén, 2009). Missing data was handled using full information maximum likelihood (FIML), a preferred method that uses all available information in the data to generate estimations (Graham, 2009).

Ethical Approval

All study protocols and procedures for the CARDIA Study were approved by the institutional review boards of each field center (Birmingham, AL: University of Alabama, Birmingham, School of Medicine; Chicago, IL: Northwestern University, Feinberg School of Medicine; Minneapolis, MN: University of Minnesota, School of Public Health; and Oakland, CA: Kaiser Permanente, Division of Research). All participants provided signed informed consent. The CARDIA Telomere Ancillary Study was approved by the University of California, San Francisco Committee on Human Research.

Results

Participant characteristics are shown in Table 1. The mean age of participants at Y15 was 39.74 years (SD = 3.86). The sample was predominantly women (74.4%) and working (86.7%); had approximately equal numbers of those with a high school degree or less (47.8%) versus more than a high school degree (44.2%); and those with annual household income less than $50,000 (54.4%) versus $50,000 or more (45.6%). LTL shortened on average from 5.62 kbp (SD = 0.45) to 4.98 kbp (SD = 0.27; approximately 64 bp/year) over the 10-year period between Y15 and Y25. The change in LTL ranged from −1.92 kbp to 0.21 kbp. There were eight participants (2%) for whom LTL values increased, and one participant whose value did not change.

Racial discrimination was most frequently reported "on the street or in a public setting," followed by "at work" and "getting a job" in both Y15 (59.1, 50.8, and 44.0%, respectively) and in Y25 (46.5, 40.5, and 37.3%, respectively). Reports of racial discrimination decreased overall, from a mean of 2.28 (*SD* = 1.88) in Y15 to 1.79 (*SD* = 1.87) in Y25. However, 22.0% of participants reported greater experiences of racial discrimination in Y25 than in Y15, compared to 35.8% who reported no change, and 42.2% who reported less racial discrimination. Additional sample characteristics are shown in Table 1.

Correlations between study variables are shown in Table 2, which were derived in Mplus using FIML to generate estimates using all available information in the entire sample. No significant bivariate relationships were found between racial discrimination measured at Y15/Y25 and LTL at either time point.

We did not find evidence of a significant cross-sectional relationship between Y15 racial discrimination and Y15 LTL (b = .009, 95% confidence interval (CI) [−.017, .034], p = .504). Results from multivariable linear regression analyses examining Y25 LTL are presented in Table 3. When examined in separate models, neither racial discrimination in Y15 (Model 1: b = .006, 95% CI [−.017, .034], p = .392) nor in Y25 (Model 2: b = −.009, 95% CI [−.024, .005], p = .208) were significantly associated with LTL in Y25. However, when including both in the model, Y25 racial discrimination was significantly associated with Y25 LTL (Model 3: b = −.019, 95% CI [−.038, −.001], p = .040), with greater reports of racial discrimination at Y25 being associated with shorter LTL. Further adjusting for LTL at Y15, the magnitude of this association remained unchanged (Model 4: b = −.019, 95% CI [−.035, −.004], p = .015).

Latent Change Score Analyses

Results from LCS analyses predicting the latent change in LTL are illustrated in Figure 1. Model fit was acceptable: CFI = .915, RMSEA = .053, SRMR = .038 (cut-off values for satisfactory model fit are: CFI > .90, RMSEA < .08, SRMR < .08; Kline, 2011). Results indicated that greater racial discrimination at Y15 was associated with less increases in reports of discrimination from Y15 to Y25 (b = −.431, 95% CI [−.515, −.347], p < .001). Those with longer LTL at Y15 had more telomere shortening (b = −.674, 95% CI [−.721, −.627], p < .001). Racial discrimination at Y15 was not significantly associated with the change in telomere length (b = −.005, 95% CI [−.019, .009], p = .482). However, consistent with linear regression findings, there was a significant association between the latent change in racial discrimination and the latent change in LTL (b = −.019, 95% CI [−.035, −.004], p = .015). Experiencing racial discrimination in more domains was associated with more LTL shortening.

Sensitivity Analyses

Several post hoc sensitivity analyses were conducted. First, we examined the full sample of 410 participants without restriction to those with valid LTL data at both Y15 and Y25. The association between the latent change in racial discrimination and latent change in LTL remained the same (b = −.019). Supplementary analyses were conducted excluding 26 participants who reported cancer or HIV, two serious health conditions that can impact the

interpretation of LTL values. Additional models were specified controlling for the presence of chronic disease at Y15 and Y25. Across these analyses, results remained unchanged. Additional analyses were conducted taking into account time-varying covariates, further adjusting for Y25 variables that showed a low correlation with Y15 values ($r < .70$): work status, being divorced, and quitting smoking. Including both Y15 and Y25 measures of these covariates did not result in substantively different conclusions; the association between the latent change in racial discrimination and the latent change in LTL remained the same.

Additional analyses using alternative coding for Y25 racial discrimination were also explored. At both Y15 and Y25, participants were asked if they had "ever" experienced racial discrimination in any of six domains; however, the overall mean reports of racial discrimination decreased between the two time periods, suggesting recall bias in retrospective reports. To address this concern, Y25 racial discrimination scores were recoded to reflect whether racial discrimination was assessed in a new domain that was not previously reported in Y15. Analyses examining incident racial discrimination reported in Y25 yielded substantively similar results. In LCS analyses, the relationship between the latent change in racial discrimination and the latent change in LTL in fact increased from b = −.019 (95% CI [−.035, −.004], $p = .015$) to b = −.027 (95% CI [−.054, −.001], $p = .042$).

We also examined the functional form of the association between racial discrimination change scores and changes in LTL by recoding racial discrimination into quartiles; we found that increasing discrimination categories showed successively greater LTL shortening. Furthermore, testing a quadratic effect of change in racial discrimination on the latent change in LTL showed nonsignificant results. These post hoc analyses suggested that modeling a linear association between changes in racial discrimination and LTL was appropriate.

Discussion

Previous cross-sectional research examining associations between discrimination and telomere length have been equivocal. Studies using data from the Health and Retirement Study have reported significant inverse associations between salivary telomere length and both major discrimination events and everyday discrimination (Lee et al., 2017; Liu & Kawachi, 2017). However, this research was conducted in an older sample of African Americans (Mean age = 70 years) using a measure of discrimination that did not distinguish between experiences that were racially motivated versus attributed to another factor. In general, among African Americans in midlife there is greater evidence for interactive rather than main effects of racial discrimination on telomere length. Similar to the CARDIA Study, the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) Study recruited a midlife sample and administered the race version of the EOD. Studies using the HANDLS data have not reported main associations of racial discrimination, but rather significant interactions with gender and socioeconomic status in relation to telomere length (Beatty Moody et al., 2019; Pantesco et al., 2018). Other research using cross-sectional data from a sample of African American men in midlife detected significant interactions with psychosocial variables, but did not find evidence for significant main effects of racial discrimination in examining telomere length (Chae et al., 2014, 2016).

In accordance with these prior studies, we did not find evidence for cross-sectional associations between racial discrimination and LTL; however, we did find a significant relationship when examining a cross-wave model. Specifically, reporting experiences of racial discrimination in more domains was associated with shorter LTL when adjusting for earlier reports of racial discrimination; this relationship persisted after controlling for baseline LTL. These results were supported by LCS analyses that modeled the change in racial discrimination in relation to changes in LTL. Results indicated that each additional domain in which racial discrimination was experienced was associated with approximately 19 bp greater LTL shortening. This finding may be contextualized in light of a systematic review that found estimates of annual loss of approximately 20–30 bp in most large cross-sectional studies (Müezzinler et al., 2013).

This study is the first to our knowledge to examine the association between changes in LTL and racial discrimination longitudinally among African Americans. Our study advances scientific directions by explicitly modeling LTL shortening, which may be specifically relevant to changing experiences of racial discrimination in midlife. Findings from this study suggest that experiencing racial discrimination in greater domains has detrimental effects on the telomere maintenance system, which may contribute to racial disparities in health. Several studies have found that African Americans experience faster telomere shortening compared to Whites, and that any initial biological advantage of longer telomeres among African Americans are outweighed by social adversities across the life course (Hamad et al., 2016; Rewak et al., 2014). Our study suggests that experiencing racial discrimination in more settings is a source of social adversity that may result in biological tolls. Such experiences may be particularly pertinent to midlife. In Y15 of CARDIA, the mean age of participants was approximately 40 years old. Some types of experiences of racial discrimination may emerge over the subsequent 10-year period, such as in employment, housing, and medical care contexts, and may be more applicable to this age cohort compared to earlier developmental periods when racial discrimination in other domains (e.g., school) might be more salient. Accumulating experiences of racial discrimination in this midlife age range may result in stress-related cell aging.

Results from this study are concordant with other findings on the embodiment of minority stress and experiences of racial discrimination (Krieger, 2012). Racial discrimination reinforces unjust patterns in residential segregation and hampers socioeconomic mobility, subsequently increasing exposure to health-damaging neighborhood conditions and diminishing access to protective resources that can impact LTL (Massey et al., 2018; Needham et al., 2014; Park, Verhoeven, Cuijpers, Reynolds, & Penninx, 2015). Studies have consistently linked racial discrimination to mental health outcomes, such as depression and psychological distress, which have been linked to shortening telomeres (Berger & Sarnyai, 2015; Blackburn et al., 2015). Recent studies indicate that racial discrimination is also associated with a range of other health concerns, including sleep problems and cardiometabolic risk, which have also been associated with shorter LTL (Cribbet et al., 2014; Fuller-Rowell et al., 2017; Goosby, Straley, & Cheadle, 2017; Mazidi, Kengne, Sahebkar, & Banach, 2018). Racial discrimination may more directly compromise health by eliciting a cascade of biochemical responses associated with physiologic arousal (Chae et al., 2011; Clark, Anderson, Clark, & Williams, 1999; Korous, Causadias, & Casper, 2017). Repeated

psychosocial insults have potential to cause dysregulation of biological systems involved in the stress response, resulting in a chronic heightened proinflammatory state associated with LTL shortening (Geronimus et al., 2010; Monaghan, 2010). Findings from this study contribute to increasing documentation of negative biological sequelae associated with psychosocial stress for African Americans.

Our findings advance the literature on racial discrimination and health outcomes in several ways. Research on the association between racial discrimination and LTL has heretofore been cross-sectional (Beatty Moody et al., 2019; Chae et al., 2014, 2016; Lee et al., 2017; Liu & Kawachi, 2017; Pantesco et al., 2018). The present study extends this line of research to a relatively large sample of African Americans in midlife, a developmental period characterized by diverging health trajectories, and when stressful life experiences may manifest in signs of biological dysregulation (House, Lantz, & Herd, 2005; Lachman, Teshale, & Agrigoroaei, 2015). Accordingly, identifying risk factors for LTL shortening during this time may yield insight into causes of racial disparities in health. We tested this association over a 10-year period in order to help deduce a temporal relationship between racial discrimination and LTL. The LCS approach allowed us to study the dynamic relationship between changes in these measures, which more robustly supports causal inferences in observational research.

Despite strengths related to the longitudinal design of this study, some caveats related to the unique characteristics of the CARDIA cohort should be noted. Because this was a largely urban sample from four specific metropolitan areas, caution should be taken in generalizing findings to other geographic regions. It is also important to note the uneven gender distribution, especially in light of prior research suggesting that African American women and men may differ in their appraisals of racial discrimination and physiologic responses to such experiences (Kershaw et al., 2016; Lewis & Van Dyke, 2018). Furthermore, findings from this study may be particular to African Americans in midlife. We assessed how experiencing racial discrimination in additional societal domains specifically during this developmental period is related to telomere shortening; however, future research that takes into account earlier experiences during childhood, adolescence, and young adulthood will be important to advancing a more comprehensive developmental perspective on biological consequences of racial discrimination during the life course (Brody et al., 2014, 2016).

Another limitation is related to the study design and analytic method we employed. Using two waves of data, we were able to correlate change in our primary exposure and outcome of interest, but were limited in examining trajectories or sequential changes in racial discrimination and LTL. As a result, inferences regarding the causal direction of the associations we found are more susceptible to alternative explanations. Although examining 10-year change in LTL in our study represents a significant advancement in research on this topic, using more data points will be an important forthcoming step. In addition, because of disagreement on methods to quantify biological aging, in the future it will be important to examine changes in racial discrimination in relation to changes in other genomic and physiologic processes (Belsky et al., 2018).

The CARDIA measure of racial discrimination is largely limited to specific domains, and does not assess more routine experiences, such as instances of being treated with less courtesy or respect in everyday interactions. It is calculated as the sum of the number of domains in which racial discrimination is experienced, and not necessarily an indicator of frequency, chronicity, intensity, or appraisals. However, the situation version of the EOD has been widely used in epidemiologic studies documenting the prevalence of racial discrimination, and has also been used to examine associations across a wide range of health-related outcomes (e.g., Borrell, Kiefe, Diez-Roux, Williams, & Gordon-Larsen, 2013; Cunningham et al., 2012, 2013; Mustillo et al., 2004; Sims et al., 2012; Subramanyam et al., 2012). Other studies have also used measures of racial discrimination, which like the EOD, assess whether participants had ever experienced discrimination in specific contexts using a yes–no response format (Borrell et al., 2010; Dominguez, Dunkel-Schetter, Glynn, Hobel, & Sandman, 2008; Harris et al., 2012; Kessler, Mickelson, & Williams, 1999; Williams et al., 2008), including a recent study that examined racial discrimination in relation to telomere length (Pantesco et al., 2018). In their validation survey, Krieger and colleagues (2005) also reported that the situation version of the EOD was highly correlated with the frequency version ($r = .90$), as well as strong associations with other commonly used measures of discrimination in a diverse cohort of working class adults.

Additionally, although the explicit focus on racial discrimination allows for more direct inferences about this specific form of unfair treatment, it may not capture those experiences that are not clearly attributable to race or which are motivationally ambiguous. Conceptually important moderators, including dimensions of racial identity that have been shown to influence self-reports of discrimination, were also not examined (Chae et al., 2017). Future studies may integrate a broader panel of measures related to racial discrimination in addition to assessments of other racism-related constructs. In addition, examining potential mediators, such as other forms of psychosocial stress (e.g., financial stress, general stress) and mental health channels, may help to further elucidate mechanisms linking racial discrimination and LTL.

Despite these limitations, results of this study contribute to evidence of the deleterious consequences of racial discrimination at the cellular level, and also point to racism as an important contributor to enduring racial disparities in health. Findings from our study also have relevance to understanding the impact that the current sociopolitical climate may have on the health of racial minority groups in the United States and the embodiment of racism at the population level (Krieger, Huynh, Li, Waterman, & Van Wye, 2018; Pew Research Center, 2019). Our study supports previous findings suggesting that LTL is sensitive to racial discrimination, and that it may be one pathway through which racism-related factors contribute to increased disease risk and accelerated declines in health among African Americans. The negative effect of racial discrimination on the telomere maintenance system may be a mechanism underlying racial disparities across multiple aging-related diseases. Results from this study suggest that addressing health inequities will require efforts to curtail endemic racial discrimination.

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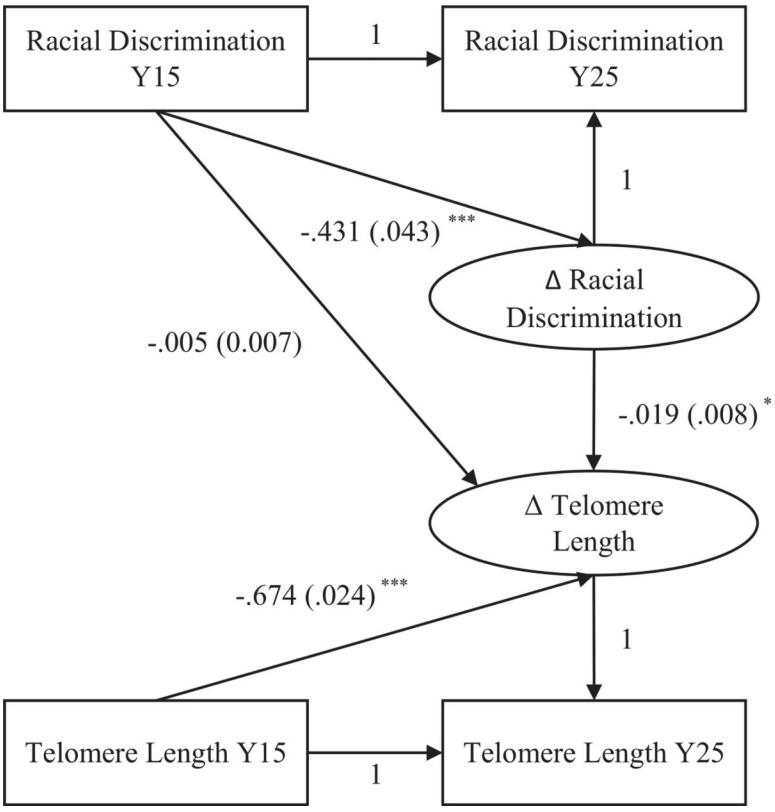

Figure 1.

Structural equation model of latent change in discrimination and telomere length among African Americans in the Coronary Artery Risk Development in Yong Adults (CARDIA) Study ($n = 391$). * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 1

Characteristics of African Americans in the Coronary Artery Risk Development in Young Adults (CARDIA) Telomere Ancillary Study, Year 15 (2000/2001) and Year 25 (2010/2011)

Characteristic	*n* (%) or *M* (*SD*)	*N*	% missing
Telomere length, kbp, Y15, *M* (*SD*)	5.62 (.45)	391	0%
Telomere length, kbp, Y25, *M* (*SD*)	4.98 (.27)	391	0%
Racial discrimination, Y15, *M* (*SD*)	2.28 (1.88)	391	0%
Racial discrimination, Y25, *M* (*SD*)	1.79 (1.87)	391	0%
Age, *M* (*SD*)	39.74 (3.86)	391	0%
Years elapsed Y15 to Y25, *M* (*SD*)	9.93 (.32)	391	0%
Gender, *n* (%)		391	0%
Women	291 (74.4)		
Men	100 (25.6)		
Education, *n* (%)		360	8%
High school or less	187 (47.8)		
More than high school	173 (44.2)		
Household income, *n* (%)		384	2%
<$25,000	91 (23.7)		
$25,000–$49,999	118 (30.7)		
$50,000+	92 (24.0)		
$75,000+	83 (21.6)		
Work status, *n* (%)		389	1%
Working	339 (86.7)		
Not working	50 (12.8)		
Marital status, *n* (%)		387	1%
Married	176 (45.5)		
Divorced, separated, widowed	102 (26.4)		
Never married	109 (28.2)		
Smoking, *n* (%)		390	0%
Never	250 (64.1)		
Former	45 (11.5)		
Current	95 (24.4)		
Body Mass Index,[a] *M* (*SD*)	30.49 (6.59)	390	0%

Note. *N* = 391. Education, household income, employment, marital status, smoking, and body mass index measured at Y15. Sum of categories may not total 391 due to missing data. kbp = kilobase pairs; *M* = mean; *SD* = standard deviation; Y15 = Year 15; Y25 = Year 25.

[a]Body mass index calculated as (weight in kilograms)/(height in meters)^2.

Table 2

Correlations Between Measures of Participant Characteristics Among African Americans in the Coronary Artery Risk Development in Young Adults (CARDIA) Telomere Ancillary Study, Year 15 (2000/2001) and Year 25 (2010/2011)

Variable	2	3	4	5	6	7	8	9	10	11	12	13
1. Telomere Length, kbp, Y15	.552***	.054	.030	−.083	.042	.030	.135**	.016	−.021	.008	−.098*	−.010
2. Telomere Length, kbp, Y25		.048	−.063	−.024	−.007	.074	.030	−.026	−.049	.017	−.059	−.047
3. Racial Discrimination, Y15			.594***	.100*	.001	−.034	.185**	.159**	−.008	.080	−.011	−.026
4. Racial Discrimination, Y25				.099*	−.048	.002	.175**	.172**	.088	.033	−.037	.015
5. Age					.035	.068	.074	.010	−.052	−.024	.094	−.002
6. Years elapsed Y15 to Y25						−.149**	.031	.115*	−.032	.017	−.070	−.060
7. Women (1)/Men (0)							.139**	−.148**	−.029	−.068	−.036	.152**
8. >(1) vs. ≤High School (0)								.225***	.103*	.093	−.127*	−.064
9. Household Income									.251***	.470***	−.204***	−.094
10. Working (1)/Not Working (0)										.041	−.121*	.042
11. Married (1)/Not Married (0)[a]											−.071	.070
12. Smoker (1)/Non-Smoker (0)[b]												−.124*
13. Body Mass Index[c]												

Note. N = 391. Age, education, household income, work status, marital status, smoking, and body mass index measured at Year 15. kbp = kilobase pairs; Y15 = Year 15; Y25 = Year 25.

[a]Not married included those never married and divorced, separated, or widowed.

[b]Non-smokers included never smokers and former smokers.

[c]Body mass index calculated as (weight in kilograms)/(height in meters)^2.

*$p < .05$.

**$p < .01$.

***$p < .001$.

Table 3

Multivariable Linear Regression Examining Leukocyte Telomere Length at Year 25 (2010/2011) Among African Americans in the Coronary Artery Risk Development in Young Adults (CARDIA) Telomere Ancillary Study

	Model 1: LTL, Year 15		Model 2: LTL, Year 25		Model 3: LTL, Year 25		Model 4: LTL, Year 25	
Variable	***b***	**95% CI**	***b***	**95% CI**	***b***	**95% CI**	***b***	**95% CI**
Racial discrimination, Y15	.006	[−.017, .034]			.017	[−.001, .035]	.015	[>.000, .029]
Racial discrimination, Y25			−.009	[−.024, .005]	−.019	[−.038, −.001]*	−.019	[−.035, −.004]*
Telomere length, Y15							.326	[.280, .373]***
Age	−.003	[−.010, .005]	−.002	[−.009, .005]	−.002	[−.009, .005]	.001	[−.004, .007]
Years elapsed Y15 to Y25	.005	[−.077, .087]	.001	[−.082, .084]	−.001	[−.084, .082]	−.021	[−.091, .048]
Women vs. Men	.047	[−.018, .112]	.044	[−.021, .109]	.048	[−.016, .112]	.042	[−.010, .095]
> High school vs. ≤ high school	.008	[−.052, .068]	.018	[−.043, .078]	.011	[−.049, .071]	−.028	[−.079, .022]
Household income	−.006	[−.023, .011]	−.004	[−.021, .013]	−.005	[−.022, .012]	−.003	[−.018, .012]
Not working vs. working	−.031	[−.102, .039]	−.030	[−.101, .040]	−.024	[−.094, .047]	−.007	[−.068, .053]
Relationship status (ref: Married)								
Divorced, separated, widowed	−.016	[−.086, .055]	−.011	[−.081, .060]	−.012	[−.083, .058]	−.021	[−.084, .042]
Never married	−.031	[−.102, .041]	−.032	[−.103, .039]	−.026	[−.097, .045]	−.017	[−.077, .043]
Body Mass Index[a]	−.003	[−.007, .001]	−.003	[−.007, .001]	−.003	[−.007, .002]	−.002	[−.006, .001]
Former vs. never smoker	.060	[−.023, .143]	.067	[−.016, .151]	.061	[−.023, .145]	.054	[−.011, .120]
Current vs. never smoker	−.034	[−.098, .030]	−.032	[−.096, .032]	−.034	[−.098, .030]	−.008	[−.065, .048]

Note. N = 391. Age, education, household income, work status, marital status, smoking, and body mass index measured at Year 15. CI = confidence interval; Y15 = Year 15; Y25 = Year 25.

[a]Body mass index calculated as (weight in kilograms)/(height in meters)^2.

*$p < .05$.

***$p < .001$.