

HHS Public Access

Psychoneuroendocrinology. Author manuscript; available in PMC 2019 December 01.

Published in final edited form as:

Author manuscript

Psychoneuroendocrinology. 2018 December; 98: 119–126. doi:10.1016/j.psyneuen.2018.08.012.

Multiple Forms of Discrimination, Social Status, and Telomere Length: Interactions within Race

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Abstract

Previous research has demonstrated inverse associations between experiences of interpersonal discrimination and telomere length, a marker of cellular aging. Here, we investigate within-race interactions between multiple indices of interpersonal discrimination and sociodemographic characteristics in relation to telomere length in African American and White adults. Participants were from the Healthy Aging in Neighborhoods of Diversity across the Life Span study (Baltimore, Maryland). Ages ranged from 30–64 years old and all self-identified as either African American (n = 176) or White (n = 165). Using linear regression, three patterns were observed within African Americans and one within Whites. Among African Americans, (1) women reporting greater lifetime burden (p = .02), racial (p = .03) and gender (p = .03) and racial discrimination (p = .02); and (3) younger adults reporting greater exposure to multiple sources of discrimination (p = .03) had shorter telomere length. Among Whites, younger and older men

Declarations of interest: none

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Keywords

discrimination; sociodemographic factors; telomere; race; gender

which may contribute to racial health disparities.

1. Introduction

For African Americans and Whites in the United States (U.S.), discrimination may be a qualitatively different phenomenon portending different health profiles. Indeed, research shows that interpersonal discrimination is more common among African Americans (Krieger et al., 2005), and the disproportionate burden of discrimination carried by African Americans is one determinant of their overall poorer health compared to Whites (Williams and Sternthal, 2010). However, a growing body of literature demonstrates that discrimination may have a negative impact on health in Whites as well (Hunte and Williams, 2009; Peterson et al., 2016). The current report examines within-race associations of interpersonal discrimination with telomere length, a marker of cellular aging, in African Americans and Whites.

Studies of epigenetic aging and inflammation show the deleterious influences of discrimination on health (Brody et al., 2016; Stepanikova et al., 2016). Discrimination is also linked with telomere length. Telomeres, nucleoprotein complexes located at the ends of chromosomes, safeguard genomic stability of cells but shorten with each cell replication (Epel, 2009). The rate of attrition of leukocyte telomeres accelerates in response to oxidative stressors, and in populations under chronic stress (Epel, 2009; Oliveira et al., 2015; Von Zglinicki, 2002). Shortened leukocyte telomeres are also associated with, and in some cases, predict chronic disease (Mathura et al., 2016). Thus, telomere length may be a psychobiomarker of both chronic stress and future illness (Epel, 2009; Mathura et al., 2016).

Several studies have reported inverse links between self-reported interpersonal discrimination, a common source of psychosocial stress, and telomere length (Chae et al., 2014, 2016; Lee et al., 2017; Liu and Kawachi, 2017; Ruiz et al., 2017), though one study failed to find an association (Geronminus et al., 2015). Most of these studies focused on racial minorities, especially African Americans, or included participants from a variety of racial/ethnic groups. A recent analysis of adults in the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study examined interactions between discrimination and sociodemographic variables for their association with telomere length across both African Americans and Whites (D. L. Beatty Moody, D. K. Leibel, T. M. Darden, et. al., unpublished observations). In that study, multiple forms of discrimination, including racial and gender discrimination, were linked to shorter telomeres, but these relationships were moderated by a number of sociodemographic factors. Whether the associations between discrimination and telomere length were similar in African Americans and Whites was not examined.

Although a between-groups framework can identify disparities between races, a withingroups approach is an important supplemental strategy when studying health correlates of discrimination (Everson-Rose et al., 2015; Whitfield et al., 2008). First, reports of discrimination are significantly higher in African Americans compared to Whites in the U.S (Krieger et al., 2005), and, accordingly, most studies showing negative health correlates of discrimination have focused on African Americans. However, there are similarly detrimental effects for health outcomes in Whites (Hunte and Williams, 2009; Peterson et al., 2016), and certain studies fail to find significant disparities between race groups (Everson-Rose et al., 2015; Pavalko et al., 2003; Smart et al., 2010). Second, the cognitive and social mechanisms involved in *perceiving* discrimination (Banks, 2014), particularly race-based discrimination, may be qualitatively different for majority versus minority groups (Major et al., 2002). Third, the impact of discrimination on health may be moderated by sociodemographic factors, (D. L. Beatty Moody, D. K. Leibel, T. M. Darden, et. al., unpublished observations) some of which also vary by race. For example, socioeconomic status (SES) is differentially distributed in African Americans and Whites (Williams et al., 2010a, 2010b). Even at similarly labeled SES strata, Whites have more wealth and resources than African Americans (Williams et al., 2010). Further, the potential benefits of higher SES on health may differ by race, with some studies reporting protective effects in Whites and mixed or null findings in African Americans (Fuller-Rowell et al., 2015; Waldstein et al., 2016; Williams et al., 2010; Williams and Sternthal, 2010). Finally, some data suggest that withinrace heterogeneity in health may exceed differences between races. Focusing solely on the latter may prevent a deeper understanding of the processes that influence health within an ethnoracial group (Lewis and Van Dyke, 2018; Monk, 2015; Williams and Sternthal, 2010).

Because discrimination, and its key potential moderators, are unequal when comparing Whites and African Americans, it is informative to investigate the health correlates of discrimination separately within race groups. Indeed, the one study to date that used a racestratified approach to examine discrimination and telomere length reported an inverse relationship in Blacks but not in Whites (Liu and Kawachi, 2017). All participants were 50 years or older; thus, it is important to determine if there are similar effects in a sample with a wider age distribution, while also accounting for the potential moderating roles of SES, age, and sex. Considering these factors for their interactions with discrimination is consistent with theoretical models of racism-related stress, as well as with intersectional theory's emphasis on understanding individuals that fall into multiply disadvantaged (or privileged) categories (Cole, 2009; Harrell, 2000). Such an approach may provide a more nuanced picture of how diversity within race groups correlates with cellular aging, which can be useful in understanding between-group differences in morbidity.

Finally, most studies on discrimination and telomere length have focused on either racial discrimination or unfair treatment, broadly defined. However, discrimination is a multidimensional construct, and the links between discrimination and risk markers may depend on how discrimination is conceptualized and assessed (Lewis and Van Dyke, 2018; Shariff-Marco, 2011). For instance, a distinction can be made between the sheer frequency of exposure to experiences of discrimination versus the subjective impact or distress associated with such experiences. Further, differences in attributions for discrimination – i.e., ascribing unfair treatment to one's race versus gender versus sexual orientation - may

influence how such experiences are perceived, and, in turn, affect correlations with other variables. Thus, measuring multiple forms of discrimination may be beneficial in determining which forms are most important for health. Here, we focus on assessing the *frequency* of experiences of discrimination (assessing both experiences that are attributed to a specific status as well as more general, non-status specific experiences), status-specific instances of discrimination due to *gender* and *race*, and the *subjective burden* or hardships perceived because of discrimination.

The primary goal of the current study is to investigate within-race interactions between interpersonal discrimination and sociodemographic variables in relation to telomere length in a sample of working aged, urban-dwelling African Americans and Whites. Specifically, we investigate SES, age, and gender as potential moderators of the relationship between discrimination and telomere length. We evaluate several forms of interpersonal discrimination in an attempt to identify the specific experiences most closely linked to cellular aging.

2. Methods

2.1 Participants and Parent Study Procedure

As previously described (Evans et al., 2010), HANDLS is an ongoing longitudinal study of health disparities attributable to race and SES. Participants are a fixed cohort of urbandwelling adults who were recruited from one of 13 groups of contiguous census segments in Baltimore, MD. All HANDLS participants self-identified their race as either African American or White, and were 30-64 years old. The first wave of HANDLS occurred between 2004–2009. After initial selection, potential participants were excluded from HANDLS if they met any of the following criteria at baseline: (1) outside of the age range of 30–64 years, (2) currently pregnant, (3) within six months of active cancer treatment (i.e., chemotherapy, radiation, or biological treatments), (4) diagnosed with AIDS, (5) unable to provide informed consent, (6) unable to provide data for at least five measures, (7) unable to provide valid government-issued identification or were currently without a verifiable address. Data collection occurred of two phases: (1) recruitment, written informed consent, and an interview and survey in participants' homes; and (2) medical history assessment, physical examination, and assessments on mobile medical research vehicles within participants' neighborhoods. Altogether, 3,720 participants met criteria for the HANDLS study, of whom 2,707 (57.7% AA, 42.3% White) completed both phases of data collection. All but 39 of these participants consented to genetic analyses. Age, sex, and poverty status were not associated with consenting for genetic assays. African Americans were more likely to consent to genetic analyses (OR=2.6, p < .05).

Subsequently, 360 participants with DNA in the biorepository from waves 1 and 3 of HANDLS were selected at random for telomere assays from a cross of race, sex, and baseline age (median-split). After additional exclusions for missing data (n = 19), there final sample consisted of 341 participants (176 African Americans and 165 Whites).

2.2 Sociodemographic Information

Participants reported their age, sex, self-identified race, annual household income (adjusted for household size), and years of education. SES was determined based on income and education. Participants were classified as "higher SES" if they reported an adjusted annual household income above or equal to 125% of the 2004 federal poverty level, *and* greater than or equal to 12 years of education. Participants were classified as "lower SES" if they reported (1) an adjusted annual household income below 125% of the 2004 federal poverty level, *or* (2) fewer than 12 years of education.

2.3 Interpersonal Discrimination Measures

2.3.1. Frequency of sources of discrimination—Ten items derived from LaVeist et al. (2003) assessed the frequency with which various sources of discrimination were experienced ("Overall how much have you experienced prejudice or discrimination due to…" gender, race, ethnicity, income, age, religion, physical appearance, sexual orientation, health status, or disability, with a 4-point response scale: (1) "not at all," (2) "a little," (3) "some," or (4) "a lot."). Total scores ranged from 10 to 40, with higher scores indicating greater frequency of sources of discrimination.

2.3.2 Lifetime burden of discrimination—Two items assessed lifetime discrimination burden: (1) "Overall, how much has discrimination interfered with you having a full and productive life?" and (2) "Overall, how much harder has your life been because of discrimination?" Participants responded on a 4-point scale: (1) "not at all," (2) "a little," (3) "some," or (4) "a lot." These two items were drawn from the MacArthur Midlife Survey, (The John D. and Catherine T. MacArthur Foundation, 2008) which measures interpersonal discrimination across lifespan.

2.3.3 Gender discrimination and racial discrimination—Five items measured experiences of gender discrimination in various settings: at school, when getting a job, at work, at home, and when getting medical care. Six items measured discrimination due to race in four of the same settings (at school, when getting a job, at work, when getting medical care), as well as when getting housing and from police or in judicial courts. Participants responded Yes (1) or No (0) to each item. Scores on the gender and racial discrimination scales ranged from 0–5 and 0–6, respectively, with greater scores indicating greater discrimination. These two measures have been used in previous epidemiological research (Krieger, 1990).

2.3.4 Everyday discrimination—The nine-item Everyday Discrimination scale (Williams et al., 1997) measures the frequency of routine experiences of discrimination without requiring the participant to make an explicit attribution (e.g., race) for the experience. Responses included: (1) "almost every day," (2) "at least once a week," (3) "few times a month," (4) "few times a year," (5) "less than once per year" and (6) "never". Responses were reversed scored, with a score of 6 indicating "almost every day." Scores range from 9–54, with higher scores indicating greater everyday discrimination.

2.4 Adjustment Variables

All analyses included depressive symptoms, lifetime substance use burden, and waist circumference as adjustment variables based on results of past studies and their potential associations with discrimination and telomere length (Chae et al., 2014, 2016; Geronimus et al., 2015; Lee et al., 2017; Liu and Kawachi, 2017; Ruiz et al., 2017). The Center for Epidemiologic Studies Depression (CES-D) scale (Radloff, 1977) is a 20-item inventory measuring depressive symptoms during the past week. Response options range from 0 (*Rarely*) to 3 (*Mostly*). Total scores may range from 0–60, with higher scores indicating greater depression. Waist circumference in centimeters was measured during the physical examination following a standard protocol. Participants self-reported their substance use history. For each substance, response options included: "Never tried," "Tried, never used regularly," "Former user (Used >6 months ago)," or "Current user (Used in past 6 months)." Participants' responses for cigarette, marijuana, cocaine/crack, and opiate use were collapsed into two levels: (1) Ever used (i.e., former or current user), or (0) Never used (i.e., never tried, or tried, but never used regularly). Scores were summed to produce a Lifetime substance use burden variable. Totals range from 0-4, with higher scores indicating a greater lifetime burden of substance use.

Data imputation was performed for all adjustment variables with < 10% missing within each race, poverty status, and sex subgroup (i.e., CES-D and waist circumference). Multiple linear regression (using age, sex, race, and poverty status as predictors) was used for imputation for the purpose of replicability.

2.5 Telomere Assays

Telomere length was determined via quantitative polymerase chain reaction (qPCR) (Cawthon, 2002). Briefly, 10 ng of DNA isolated from peripheral blood mononuclear cells was used in each PCR reaction, and triplicate reactions were performed per sample. From each triplicate set, the average cycle threshold (Ct) values of T and S were calculated to generate the average T/S ratio value. Telomere length from 130 samples were measured by both qPCR and the Southern method (Lin et al., 2015), and the resulting conversion equation was used to calculate telomere length in kb from the T/S ratio value.

2.6 Statistical Approach

All statistical analyses were run with the Statistical Package for the Social Sciences (SPSS) version 24 or R version 3.5. Multiple linear regression with hierarchical entry was used for all analyses. We examined interactive relations of each of the different forms of discrimination with age, sex, or SES up to the three-way interaction level. All analyses were stratified by race and separate, concurrent models were run to examine the different indices of discrimination described above. Analyses began with minimally adjusted models that included only main effects and adjustment variables. Subsequently, three two-way interaction effects were added to the model in the second step, followed by the addition of one three-way interaction effect in the third step. If the three-way interaction (i.e., highest-order) effect was significant, the third model was retained. Conversely, if the three-way interaction effect, the second model was retained. Finally, if no significant interactions were significant, then

the first (i.e., minimally adjusted) model was retained. Across all analyses, regression model fit improved slightly as interactions were added to the models.

To assist with interpretation, significant interactions were probed using the PROCESS macro for SPSS (version 2.16, models 1 and 3; Hayes, 2013). For significant interactions with continuous moderators, the Johnson-Neyman technique was used within PROCESS to identify the range of moderator values at which the association between discrimination and telomere length was significant, allowing for a more precise inspection of the moderator's effect (Hayes, 2013; Johnson, 1950).

3. Results

There were no race differences in the distribution of sex, SES indicators, age, depressive symptoms, or telomere length. There were race differences in waist circumference, lifetime substance use burden, and all discrimination measures except everyday discrimination (Table 1). Correlations among all study variables in the full sample are shown in Table A.1.

Among African Americans analyses revealed six significant two-way interaction effects(Table 2; for complete data from the regression models, see Tables A.2, A.3, A.4, and A.5). Three significant two-way interactions demonstrated that, among African American women, shorter telomere length was associated with greater (a) belief that discrimination made life harder, b = 0.25, p = .02 (Figure 1); simple effect, b = -0.34, p = .002; (b) racial discrimination, b = 0.14, p = .02; simple effect, b = -0.22, p = .001 (Figure 2); and (c) gender discrimination, b = 0.20, p = .01; simple effect, b = -0.21, p = .01 (Figure A.1). Next, two significant two-way interactions demonstrated that, among African Americans with higher SES, shorter telomere length was associated with greater (a) belief that discrimination made life harder, b = 0.27, p = .02; simple effect, b = -0.34, p = .002 (Figure 1); and (b) racial discrimination, b = 0.18, p = .01; simple effect, b = -0.21, p = .001 (Figure A.2). In addition, there was a significant two-way interaction of Frequency of Sources of Discrimination \times Age, b = .002, p = .03. Subsequent analysis with the Johnson-Neyman technique revealed that greater frequency of sources of discrimination was associated with shorter telomere length in African Americans 40.42 years old and younger (all p's < .05; Table A.6). Analyses revealed no further significant interactions in African Americans. Everyday discrimination did not predict telomere length among African Americans, neither as a main effect nor in an interaction term (p's > .05). However, results revealed a significant main effect of sex when adjusting for everyday discrimination (and in all other analyses), such that African American women had shorter telomeres than African American men, b =0.54, p < .001. In African Americans, there was no main effect of age with telomere length when adjusting for the effects of sex, SES, discrimination, and covariates (p's > .05).

One of the African American participants had an average telomere length of 2.60 kb, which was 4.09 standard deviations below the mean of 5.64 kb for all African Americans in the study. We retained this participant in the above analyses after confirming that their score was not physiologically aberrant and passed quality control measures. However, because previous telomere studies have used a cutoff of ± 4 standard deviations to identify outliers (Carlson et al., 2015; Epel et al., 2010; Lin et al., 2016), we ran subsequent sensitivity

analyses with this participant excluded to determine if their score influenced the significance of interactions or main effects. After removing this participant, all previously significant interaction effects and main effects in the African American subsample remained significant, except a pair of two-way interactions involving racial discrimination. Specifically, Racial Discrimination \times SES reduced to b = .12, p = .06, and Racial Discrimination \times Sex reduced to b = .09, p = .14.

Among Whites, findings revealed three significant three-way interaction effects. First, there was a significant three-way interaction of Racial Discrimination \times Age \times Sex with telomere length, b = -0.04, p = .02 (see Table 2; for complete data from the regression models, see Table A.7). As depicted in Figure 2, among younger White men (39 years), greater racial discrimination was associated with significantly shorter telomere length, b = -0.27, p = .047; conversely, among older White men (56 years), greater racial discrimination was associated with significantly longer telomere length, b = 0.39, p = .02. Racial discrimination was not associated with telomere length in White women. Next, findings revealed two significant three-way interactions of (1) Belief that Discrimination Interfered with Life \times Sex \times SES, b = -0.81, p = .004; and (2) Belief that Discrimination Made Life Harder \times Sex \times SES, b = -0.58, p = .04. However, further analysis failed to demonstrate significant simple effects (all p's > .05). Analyses revealed no other significant interactions in Whites. Everyday discrimination did not predict telomere length among Whites, neither as a main effect nor within an interaction term (p's > .05). However, results revealed a significant main effect of age when adjusting for everyday discrimination (as well as in all other analyses), such that greater age was associated with shorter telomere length, b = -0.01, p = .03.

Post-hoc analyses were run to compare parameter estimate differences for the significant main effects and interaction effects (i.e., only those that yielded significant simple effects) across parallel race-stratified models (see Table A.8). Briefly, analyses revealed significant racial differences in parameter estimates for the three-way interaction of Racial Discrimination \times Age \times Sex and main effect of sex (p's .05), which were previously found to be significant in the White and the African American samples, respectively.

4. Discussion

We found that various forms of discrimination were related to telomere length and that these relationships may occur in different patterns for African Americans and Whites. Among African Americans, greater gender discrimination and the belief that discrimination had made life harder were related to shorter telomeres in women; the belief that discrimination had made life harder were related to shorter telomeres in those of higher SES. There was also evidence that racial discrimination may be related to shorter telomeres in African Americans, experiencing more frequent discrimination from a variety of sources was associated with shorter telomeres. Among Whites, racial discrimination was related to telomere length in men only, but the direction of this relationship varied by age. Greater racial discrimination was related to shorter telomeres in older men. Finally, main effects differed by race. In African Americans, women had shorter telomeres

than men, while in Whites, telomere length decreased with older age. Relationships were not influenced by waist circumference, history of substance use, or depressive symptoms.

Results from this study are generally consistent with the larger literature showing inverse associations between discrimination and telomere length (Chae et al., 2014, 2016; Lee et al., 2017; Liu and Kawachi, 2017; Ruiz et al., 2017). In the aggregate, these data support models of discrimination as a psychosocial stressor (Harrell, 2000). When encountered repeatedly, experiences of discrimination may result in chronic physiological arousal, which in turn may affect telomere maintenance (Mathura et al., 2016). Our findings add to the literature by focusing on the correlates of discrimination separately within African Americans and Whites, thus providing an opportunity to examine the effects of overlapping demographic categories in more depth. Gender discrimination was associated with shorter telomeres in African American women in the current sample, which is consistent with the "double jeopardy" hypothesis (Beale, 1970), as well as studies demonstrating different, and in some cases, stronger, associations between discrimination and health outcomes in African American women compared to African American men (Beydoun, Poggi-Burke, Zonderman, et al., 2017; Cunningham, Berkman, Kawachi, et al., 2013; Cunningham, Seeman, Kawachi, et al., 2012). Specifically, African American women are likely to encounter multiple sources of discrimination as a function of occupying doubly disadvantaged statuses. Indeed, the cooccurrence of sexism and racism in African American women is common and together more deleterious with regard to a myriad of health outcomes (Thomas et al., 2008). When encountered repeatedly, experiences of discrimination may contribute to the accelerated biological aging observed in African American women here and in other studies (Geronimus et al., 2010).

Studies of the interaction of race and social class consistently show that higher-SES African Americans report more discrimination than lower-SES African Americans (Lewis and Van Dyke, 2018). Studies examining the effect of SES on racial disparities in health, however, are less consistent (Fuller-Rowell et al., 2015; Waldstein et al., 2016; Williams et al., 2010, 2016). Our finding that an increased subjective burden of discrimination was linked to shorter telomeres in African Americans of higher SES supports the "diminishing returns" hypothesis, which proposes that attainment of higher education or income does not offer the same health benefits in African Americans as it does in Whites, with the greatest health disparities between races observed at higher levels of SES (Farmer and Ferraro, 2004). Some have theorized that attributions to discrimination may be more detrimental to African Americans of higher SES, who likely have more interactions with Whites and other racial groups in work and other day to-day settings than African Americans of lower SES and/or may be more likely to recognize social injustices (Farmer and Ferraro, 2004; Fuller-Rowell et al., 2012). The lack of equivalence between African Americans and Whites in similar SES strata, and data showing that reports of discrimination increase with SES in African Americans (Borrell et al., 2007; Colen et al., 2017; Williams et al., 2010, 2016), should also be considered when interpreting these findings.

Finally, experiencing more frequent discrimination attributed to a variety of sources was associated with shorter telomeres in younger African Americans (i.e., 40 years old and younger). This is interesting considering some data showing that younger African

Americans report more *overall* discrimination (i.e., not necessarily race-specific) than their older counterparts (Lewis and Van Dyke, 2018). Lewis and Van Dyke (2018) also raise the possibility that younger African Americans may be exposed to more serious or traumatizing forms of discrimination, which could partially explain the stronger link with shorter telomeres in this group. Further, the fact that more frequent discrimination from a variety of sources was specifically linked to telomere length may suggest that younger individuals who perceive persistent and/or multi-faceted forms of discrimination linked to various parts of their identity sustain the most severe impact on their well-being and health. Further work disentangling which of the various social statuses, and subsequent unfair treatment, are most closely associated with telomere length in young, African American adults would be beneficial.

For Whites, the relationship between racial discrimination and telomere length was moderated by gender and age. In younger White men, discrimination was inversely associated with telomere length, consistent with other studies reporting links between poor health and discrimination, irrespective of race (Everson-Rose et al., 2015; Smart et al., 2010). However, it was somewhat surprising that unfair treatment attributed to one's race was the only form of discrimination that emerged in relation to telomere length within White individuals. This finding is especially interesting the context of emerging evidence that: 1) growing mortality rates among young to middle-aged White Americans are due to their mounting "cumulative disadvantage" (Case et al., 2017); and 2) a growing number of Whites perceive themselves as more heavily targeted for race-related discrimination than in the past (Gonyea, 2017). Thus, the finding in younger White males may reflect a complex interaction of race, age, and health during a time in which the appearance of diversity yields heightened perceptions of unfairness, which may be particularly threatening for younger individuals working to establish themselves. On the other hand, the positive association between racial discrimination and telomere length in older White men was unexpected. One possible explanation is that increased perceptions of discrimination in this subgroup may be protective or beneficial. Wilkins et al. (2016) demonstrated that priming White participants with the concept of racial progress was experienced as threatening to their self-worth; however, providing individuals with a chance to attribute negative events to discrimination, rather than to themselves, resulted in a rebound of self-worth. For older White men, their relation to the concept of race-related discrimination across their lifetimes may have differentially oriented them to such experiences. Overall, these data suggest that both gender and age, or perhaps cohort effects, may influence appraisals, as well as the potential consequences, of racial discrimination.

When considered in the aggregate, these findings may offer insight into the salience of dimensions of discrimination for subcategories of African Americans and Whites with regard to telomere length, and, potentially, healthy versus accelerated aging. For instance, experiences of discrimination that were social status-specific (i.e., gender, race), as well as those reported as making one's life more difficult, both emerged in relation to telomere length in African American women and African Americans of higher SES, while in younger, White men, only racial discrimination was related to shorter telomeres. As African American women and higher SES African Americans have faced unique challenges to attain and maintain gains in educational settings and the workforce (Hardaway & Mcloyd, 2009;

Kwate & Goodman, 2015), blocked opportunities and obstacles due to their social statuses may be particularly meaningful with regard to health. The social status-specific (i.e., racial discrimination) finding in younger, White men, on the other hand, could potentially be related to the emerging literature demonstrating the growing salience of race for Whites due to shifting demographics in the U.S.(Gonyea, 2017). Altogether, these findings underscore the need to consider the health implications of discrimination as a multidimensional construct influenced by intersecting social statuses, allowing for recognition of intersectional invisibility (e.g., African American women; Purdie-Vaughns & Eibach, 2008) as well as intersectional paradoxes (e.g., African Americans with higher SES; Bowleg, 2012).

Finally, post-hoc analyses compared racial differences in parameter estimates for the significant effects described (see Table A.8). Across the race-stratified parallel models only two effects were significant: the parameter estimates for the main effect of sex (significant in African Americans only) and the three-way interaction of Racial Discrimination \times Age \times Sex (significant in Whites only). In light of the varying patterns of results within racial groups, the lack of specific racial differences in parameter estimates may reflect inadequate statistical power to detect racial differences for the majority of the effects. Future studies should re-examine the present findings with a larger sample, and researchers should continue to examine both within- and between-race effects in discrimination studies.

There are limitations to consider. Our analyses were unable to determine temporal relationships or causal links between discrimination and telomere length. We did not adjust for some health behaviors that could potentially mediate the relationship between discrimination and shortened telomeres, such as smoking and physical activity (Liu et al., 2017; Mathura et al., 2016). We focused only on interpersonal forms of discrimination without assessing other types of unfair experiences, such as institutional racism, and we did not fully explore attributions due to personal characteristics other than sex and race (e.g., sexual orientation). Additionally, we examined each form of interpersonal discrimination as a separate entity. It may be interesting to consider the cumulative burden of, as well as the interactions among, multiple types of discrimination on health. For example, future work may want to focus on the health correlates of gendered racism, or the simultaneous overlap of the two forms of discrimination, in women (Thomas et al., 2008). Also, we focused on only two racial groups, and it is important to investigate the health correlates of discrimination in under-studied racial and ethnic groups. Finally, although telomere length is considered a psychobiomarker of aging, we were unable to quantify the clinical significance of telomere shortening in this sample.

4.1 Conclusions

In sum, we report associations between multiple indices of interpersonal discrimination and telomere length, a psychobiomarker of cellular functioning, in African American and White adults. The types of discrimination that are associated with telomere length may differ by race, as well as by sociodemographic moderators. Specifically, we observed that several forms of discrimination were associated with shorter telomeres in female, younger, or higher-SES African Americans, while only racial discrimination was linked to telomere length in White men. These findings underscore: 1) the value of examining the health

correlates of multiple forms of discrimination, 2) that there may be critical variations in the patterning of discrimination and health between racial groups, and 3) the importance of considering contextual moderators such as SES, age, and sex when examining the health implications of discrimination. As telomere length may be an indicator of current and future health status, our findings may ultimately contribute to an improved understanding of the physiological mechanisms linking discrimination to morbidity and mortality, as well as biopsychosocial models of racial health disparities.

Acknowledgements

This work was supported by: K01AG043581 (Beatty Moody), R01AG034161 (Waldstein), the National Institute on Aging's Intramural Research Program ZIAG000513 (Evans), and the Claude D. Pepper Older Americans Independence Center (P30AG028747; Katzel).

Funding

We wish to thank the HANDLS participants for their continued commitment to the study, as well the HANDLS research team. The National Institute on Aging Intramural Research Program of the National Institutes of Health (NIH) directed this research.

Appendix

Table A.1

Pearson Correlations among Study Variables in the Full Sample (N = 341)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Age	1	.02	.07	.05	.07	13	12	.07	09	02	.19*	06	15*
2. Sex ^{<i>a</i>}		1	01	03	09	04	27 **	10	04	21 **	.10	.03	.02
3. Sociaeconomic status (SES)			1	02	12	02	.05	<.01	.12	.36**	.14	.19*	08
4. Discrimination interfered with life				1	.80**	.43**	.54 **	.53 **	.28 **	.21**	.22**	13	01
5. Discrimination made life harder					1	.44 **	.50**	.58**	.30**	.16*	.10	14	.04
6. Racial discrimination						1	.49 **	.46**	.20**	.04	.04	.03	003
7. Gender discrimination							1	.44 **	.22 **	.18*	.01	14	10
8. Sources of discrimination								1	.29 **	.28 **	.15	.01	08
9. Everyday discrimination									1	.39 **	.21 **	.12	08
10. Depressive symptoms										1	.13	.12	07
11. Waist circumference											1	06	17*
12. Lifetime substance use burden												1	.13
13. Telomere length (kb)													1

Note.

p < .05

p < .01

^{*a*}Sex: 0 = women, 1 = men

^bSES: 0 = higher SES, 1 = lower SES

 C Lifetime substance use burden = number of substances (cigarettes, marijuana, cocaine/crack, heroin) that participants ever used regularly

Table A.2

Regression Models Estimating Interactive Relations of Belief that Discrimination Made Life Harder, Sex, and Socioeconomic Status with Telomere Length among African Americans

	Model 1	Model 2	Model 3
Age	-0.01	-0.002	-0.002
Depressive symptoms	0.003	0.002	0.002
Waist circumference	<.001	<.001	<.001
Lifetime substance use burden	-0.91	-0.09	-0.09
Discrimination made life harder	-0.42	-0.34 **	-0.45 **
Sex	0.55 ***	0.18	-0.22
SES	-0.05	-0.48	-0.79 *
Discrimination Made Life Harder × Sex		0.25 *	0.44
Discrimination Made Life Harder × SES		0.27 *	0.43 *
$Sex \times SES$		-0.23	0.39 **
Discrimination Made Life Harder \times Sex \times SES			-0.30

Note. Model 2 (shown in **bold** above) was retained as the final regression model, and significant effects were interpreted $p^* < .05$

p < .01*** p < .01*** p < .001Model 1 fit: R = .36, $R^2 = .13$, $R^2_{adj} = .09$ Model 2 fit: R = .42, $R^2 = .18$, $R^2_{adj} = .13$ Model 3 fit: R = .43, $R^2 = .19$, $R^2_{adj} = .13$

Table A.3

Regression Models Estimating Interactive Relations of Racial Discrimination, Sex, and Socioeconomic Status with Telomere Length among African Americans

	Model 1	Model 2	Model 3
Age	-0.01	-0.01	-0.01
Depressive symptoms	0.003	0.001	0.001
Waist circumference	< 0.001	<0.001	<.001
Lifetime substance use burden	-0.09	-0.08	-0.08
Racial discrimination	-0.02	-0.21 **	-0.31 **
Sex	0.56 ***	0.60 **	-0.39
SES	-0.05	-0.13	-0.28
Racial Discrimination \times Sex		0.14 *	0.28 **
Racial Discrimination \times SES		0.18 **	0.31 **
$\mathbf{Sex} \times \mathbf{SES}$		-0.40	0.09
Racial Discrimination \times Sex \times SES			-0.21

Note. Model 2 (shown in **bold** above) was retained as the final regression model, and significant effects were interpreted.

p < .05



p < .001

Model 1 fit: R = .36, $R^2 = .13$, $R^2_{adj} = .09$ Model 2 fit: R = .43, $R^2 = .19$, $R^2_{adj} = .14$ Model 3 fit: R = .45, $R^2 = .21$, $R^2_{adj} = .15$

Table A.4

Regression Models Estimating Interactive Relations of Gender Discrimination, Sex, and Socioeconomic Status with Telomere Length among African Americans

	Model 1	Model 2	Model 3
Age	-0.01	-0.01	-0.01
Depressive symptoms	0.003	0.003	0.003
Waist circumference	< 0.001	<0.001	0.001
Lifetime substance use burden	-0.09	-0.10 *	-0.09
Gender discrimination	-0.03	-0.21 *	-0.31 **
Sex	0.53 ***	0.48 *	-0.32
SES	-0.05	-0.03	-0.15
Gender Discrimination \times Sex		0.20 *	0.38 **
Gender Discrimination \times SES		0.11	0.26 *
$\text{Sex} \times \text{SES}$		-0.21	0.03
Gender Discrimination \times Sex \times SES			-0.26

Note. Model 2 (shown in **bold** above) was retained as the final regression model, and significant effects were interpreted.

p < .05*** p < .01*** p < .001Model 1 fit: R = .36, $R^2 = .13$, $R^2_{adj} = .09$ Model 2 fit: R = .41, $R^2 = .17$, $R^2_{adj} = .12$ Model 3 fit: R = .43, $R^2 = .18$, $R^2_{adj} = .13$

Table A.5

Regression Models Estimating Interactive Relations of Sources of Discrimination and Age with Telomere Length among African Americans

	Model 1	Model 2	Model 3
Sex	0.55 ***	0.59 ***	0.58 ***
Depressive symptoms	0.003	0.01	0.01
Waist circumference	< 0.001	-0.002	-0.002
Lifetime substance use burden	-0.10	-0.10 *	-0.10 *
Sources of discrimination	-0.004	-0.12 *	-0.09
Age	-0.01	-0.05 *	-0.04
SES	-0.06	1.04	-0.06
Sources of Discrimination \times Age		0.002 *	0.001 **
Sources of Discrimination \times SES		0.02	-0.04 *
$Age \times SES$		0.01	-0.01
Sources of Discrimination \times Age \times SES			0.001

Note. Model 2 (shown in **bold** above) was retained as the final regression model, and significant effects were interpreted. * p < .05

p < .01**** p < .001Model 1 fit: $R = .36, R2 = .13, R^{2}adj = .09$ Model 2 fit: $R = .41, R2 = .17, R^{2}adj = .11$ Model 3 fit: $R = .41, R2 = .17, R^{2}adj = .12$

Table A.6

Johnson-Neyman Results Depicting the Association between Sources of Discrimination and Telomere Length at Various Ages among African Americans

Age	<u>b</u>	<u>se</u>	p
30.00	06	.02	.019
31.70	05	.02	.021
33.40	05	.02	.023
35.10	05	.02	.026
36.80	04	.02	.030
38.50	04	.02	.037
40.20	03	.02	.048
41.42	03	.02	.050
41.90	03	.02	.065
43.60	03	.02	.092
45.30	02	.02	.134
47.00	02	.02	.198
48.70	02	.02	.291
50.40	01	.02	.416
52.10	01	.02	.571
53.80	01	.02	.744
55.50	002	.02	.921
57.20	.002	.02	.912
58.90	.01	.02	.764
60.60	.01	.02	.638
62.30	.01	.02	.535
64.00	.02	.02	.451
64.00	.02	.02	.451

Note. Ages at which the association between frequency of sources of discrimination and telomere length are significant (p < .05) are shown in **bold**. The age (i.e., 41.42 years old) at which the association between frequency of sources of discrimination and telomere length transitions to statistical significance is shown in **bold and italics**.

Table A.7

Regression Models Estimating Interactive Relations of Racial Discrimination, Age, and Sex with Telomere Length among Whites

	Model 1	Model 2	Model 3
SES	-0.11	-0.13	-0.12
Depressive symptoms	-0.002	-0.001	-0.001
Waist circumference	-0.01	-0.01	-0.01
Lifetime substance use burden	0.08	0.08	0.08 *
Sources of discrimination	-0.02	-0.59	0.20

	Model 1	Model 2	Model 3
Age	-0.01 *	-0.02	-0.01
Sex	0.09	0.02	-0.54
Racial Discrimination × Age		0.01	-0.01 **
Racial Discrimination \times Sex		0.01	-2.00 *
$Age \times Sex$		0.001	-0.01
Racial Discrimination \times Age \times Sex			-0.04 *

Note. Model 3 (shown in **bold** above) was retained as the final regression model, and significant effects were interpreted. * p < .05

** p < .01Model 1 fit: R = .29, $R^2 = .09$, $R^2_{adj} = .04$ Model 2 fit: R = .31, $R^2 = .10$, $R^2_{adj} = .04$ Model 3 fit: R = .36, $R^2 = .13$, $R^2_{adj} = .06$

Table A.8

Racial Differences in Parameter Estimates for Significant Effects across Parallel Analyses

Significant effects in the African American sample							
Effect	z-difference	P					
Discrimination Made Life Harder $\times Sex$ interaction	-0.81	.42					
Discrimination Made Life Harder \times SES interaction	-0.84	.40					
Racial Discrimination × Sex interaction	-0.35	.73					
Racial Discrimination × SES interaction	0.03	.98					
Gender Discrimination \times Sex interaction	-1.62	.11					
Frequency of Sources of Discrimination \times Age interaction	0.00	1.00					
Sex main effect (significant in African Americans only)	-2.71	.01					
Significant effects in the White sample							
Racial Discrimination \times Age \times Sex interaction	1.92	.05					
Age main effect	-0.71	.48					

Note. Significant racial differences in parameter estimates across parallel, race-stratified regression models are shown in **bold**, p = .05.

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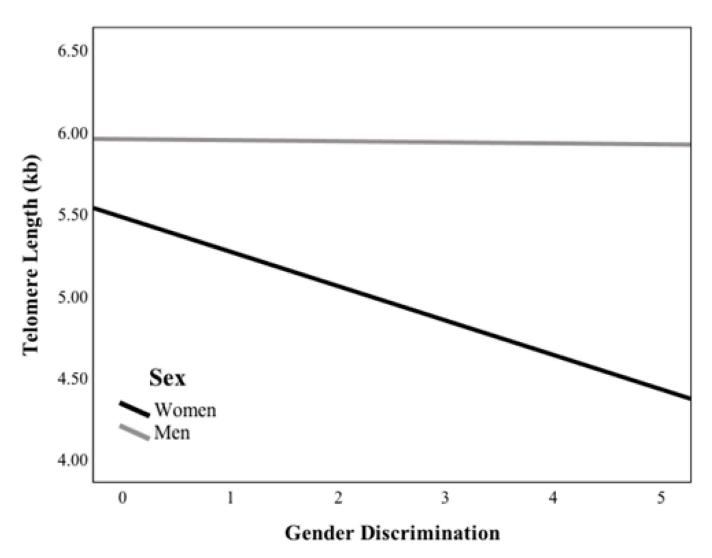


Figure A.1.

Significant moderating effect of sex on the association between gender discrimination and telomere length among African Americans.

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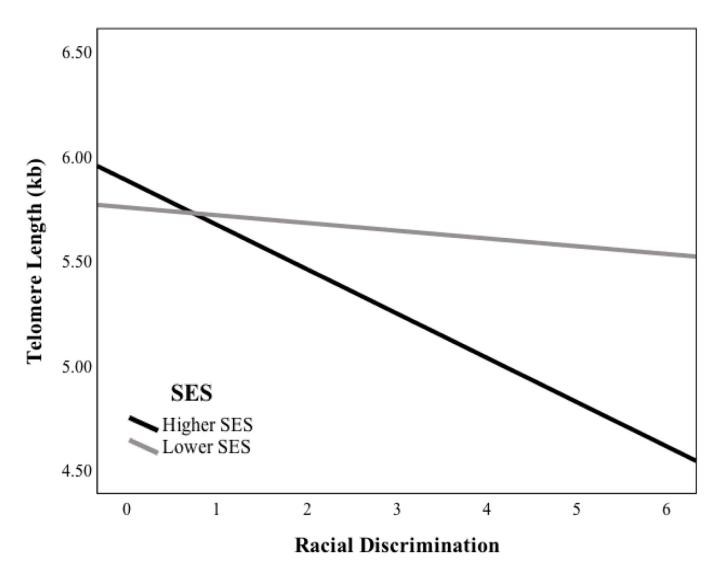


Figure A.2.

Significant moderating effect of SES on the association between racial discrimination and telomere length among African Americans.

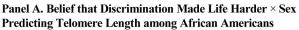
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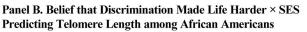
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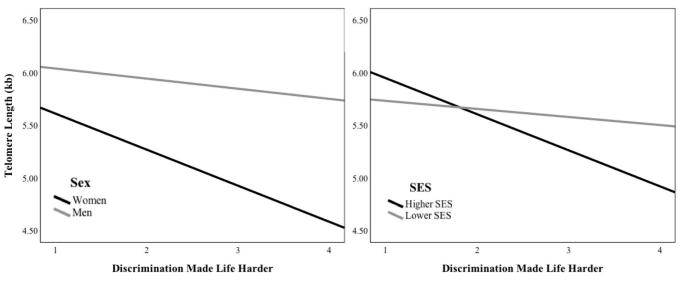


Figure 1.

Panels A and B: Significant moderating effects of (A) sex and (B) SES on the association between belief that discrimination made life harder and telomere length among African Americans.

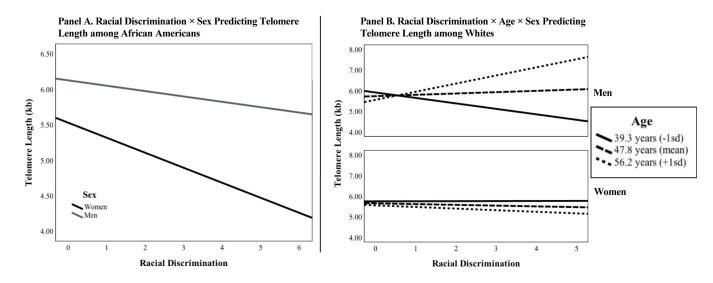


Figure 2.

Panel A: Significant moderating effect of sex on the association between racial discrimination and telomere length among African Americans. *Panel B*: Significant moderating effects of age and sex on the association between racial discrimination and telomere length among Whites.

Table 1

Participant Characteristics Stratified by Race

	African Americans (n = 176)	<u>Whites (<i>n</i> =165)</u>
% Women	48.3%	52.1%
% <12 years education	29.0%	37.6%
% <125% 2004 federal poverty level	50.0%	51.5%
Lower SES ^a	61.4%	63.6%
Age	47.57 (±9.40)	47.89 (±8.41)
CES-D scale score	13.52 (±9.86)	15.56 (±11.54)
Waist circumference	95.48 (±16.97) ***	103.71 (±18.39) ***
Lifetime substance use burden b	1.64 (±1.24) *	1.36 (±1.17) *
Sources of discrimination	17.84 (±6.31) ***	14.93 (±4.60) ***
Discrimination interfered with life	1.88 (±1.00) ***	1.48 (±0.82) ***
Discrimination made life harder	2.06 (±1.01) ***	1.45 (±0.80) ***
Gender discrimination	0.96 (±1.37) ***	0.32 (±0.74) ***
Racial discrimination	1.78 (±1.96) ***	0.34 (±0.91) ***
Everyday discrimination	21.15 (±7.88)	19.73 (±7.81)
Telomere length (kb)	5.62 (±0.75)	5.69(±0.69)

Note. Significant racial differences examined with independent samples t-tests and chi-square tests of independence

* p<.05

*** p<.001

^{*a*}Participants were considered to have *lower SES* if they reported (a) < 12 years of education and/or (b) adjusted household incomes < 125% of the 2004 federal poverty level.

 b Number of substances (cigarettes, marijuana, cocaine/crack, heroin) that participants ever used regularly

Table 2

Significant Interaction Effects across Regression Analyses

African American participants				
Significant interaction effects	<u>b</u>	<u>se</u>	р	$\underline{\eta}^2_{partial}$
Discrimination Made Life Harder \times Sex *	0.25	.11	.022	.03
Discrimination Made Life Harder \times SES $*$	0.27	.11	.020	.03
Racial Discrimination \times Sex *	0.14	.06	.020	.03
Racial Discrimination × SES **	0.18	.06	.005	.05
Gender Discrimination \times Sex *	0.20	.08	.010	.04
Sources of Discrimination \times Age *	0.002	.001	.032	.03
White participants				
Significant interaction effects a	<u>b</u>	<u>se</u>	P	$\underline{\eta}^2_{partial}$
Racial Discrimination \times Age \times Sex *	04	.02	.021	.04

Note.

* p<.05

** p < .01. Variables are the highest-order significant interaction effects from regression models. See Tables A.2, A.3, A.4, A.5, and A.7 for complete results from each regression model.