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Interpersonal-level discrimination indices, sociodemographic factors, and telomere length in African-Americans and Whites

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ABSTRACT

Objective: Studies have linked self-reported discrimination to telomere attrition, a biological marker of accelerated cellular aging. However, it is unknown whether intersections between social categories—race, socioeconomic status (SES), sex, and age—influence the association of varying forms of discrimination with telomere length. We examined these associations in a socioeconomically and racially/ethnically diverse urban sample.

Methods: Cross-sectional data were from 341 middle-aged (30–64 years) African American and White, community participants in the Healthy Aging in Neighborhoods of Diversity across the Life Span Study (HANDLS). Multiple regression models examined up to 3-way interactions between a discrimination measure (i.e., everyday, racial, gender, lifetime burden, and frequency of discrimination across sources) and two social categories.

Results: After adjusting for depressive symptoms, waist circumference, and lifetime substance use, two themes emerged: 1) among women with higher SES, a) greater lifetime discrimination burden (b = -0.23, p = .011), gender discrimination (b = -0.29, p = .040), and racial discrimination (b = -0.24, p = 0.023) and 2) among younger adults, irrespective of race and sex, greater frequency of discrimination across sources (b = 0.002, p = .008) was associated with shorter telomeres.

Conclusions: Irrespective of race, women with higher SES and younger adults reporting greater discrimination may be at particular risk for accelerated aging. Telomere attrition promotes and accelerates chronic health conditions for which there are health disparities. Future research explicating intersections among specific discrimination indices and social categories is warranted.

1. Introduction

In the United States (U.S.), social stress is a pervasive aspect of daily life for many individuals. Indeed, social stressors are particularly persistent along the lines of marginalized statuses associated with race, age, sex, and socioeconomic status (SES) and are linked to a myriad of aging-related poorer health (e.g., Bosworth, 2018; Cunningham et al., 2017; Meyer, 2003; Schnittker & McLeod, 2005; Williams & Jackson, 2005). Importantly, the cellular mechanisms underlying these linkages remain largely understudied (Epel, 2009). Telomeres represent one such plausible pathway.

Within human somatic cells, telomeres consist of tandem repeats of the TTAGGG DNA sequence as well as specific associated proteins (Chan & Blackburn, 2004). Located at the ends of each chromosome, telomeres confer protection to the underlying genetic material, and thus help safeguard genetic stability within the cell. However, recurring cellular replication, the absence of telomerase activity within human somatic cells, and chronic stress exposure together contribute to a reduction in telomere length (Epel, 2009). Consequently, critically shortened telomeres not only compromise genetic stability within the cell, but also promotes cellular senescence, and ultimately, apoptosis (Calado & Young, 2009; Chan & Blackburn, 2004). Telomere attrition

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has been prospectively associated with all-cause mortality and morbidity across several disease endpoints, including cancer and cardiovascular disease (Epel et al., 2009; Haycock et al., 2014).

With respect to the contribution of chronic stress exposure to cellular apoptosis, telomeres have been conceptualized as "psychobiomarkers," or biological indices of psychosocial stress (Epel, 2009). A body of work has demonstrated the adverse linkage of psychosocial stress to telomere length across various forms of adversity. Meta-analyses and systematic reviews highlight that stress arising from psychiatric illness, early life adversity, violence exposure, caregiver strain, life events (e.g., divorce or death of a loved one), and poverty contribute to shortened telomeres (Darrow et al., 2016; Mathur et al., 2016; Oliveira et al., 2016; Ridout, Ridout, Price, Sen, & Tyrka, 2016, 2018; Schutte & Malouff, 2016). Importantly, these findings demonstrate that chronic sources of stress may have long-lasting consequences for health as reflected in accelerated biological aging.

Discrimination, is a specific type of chronic stressor reflecting unfair treatment unfolding in interpersonal interactions. Discrimination has been established as a potent and deleterious factor in mental and physical health disparities (Paradies, 2006; Paradies et al., 2015; Pascoe & Smart Richman, 2009). Discriminatory experiences typically vary along the lines of race and ethnicity (hereafter race), age, sex, and SES, paralleling sociohistorical demarcations of social categories in the U.S. (Kessler, Mickelson, & Williams, 1999). Similarly, disparities in health also vary along these established lines, with social categories functioning as robust predictors of disease endpoints (U.S. Department of Health & Human Services, 2014). Recent evidence suggests that telomere attrition is inversely associated with self-reported discrimination and also varies by sociodemographic category.

1.1. Linkages across types of discrimination and telomere length

Two recent reports from the Health and Retirement Study show that different forms of discrimination are linked to telomere length in older (> 50 years) African Americans. First, in analyses exclusive to African Americans, major lifetime discrimination (e.g., not being hired for a job) – but not everyday discrimination (e.g., being treated with less courtesy in day-to-day life) – was inversely linked to telomere length (Lee, Kim, & Neblett, 2017). However, in race-stratified analyses, everyday discrimination – but not major life discrimination – was inversely linked to telomere length in African Americans, but not Whites (Liu & Kawachi, 2017). Some studies of discrimination and telomere length have reported null effects (Geronimus et al., 2015). Others examining specific forms of discrimination, principally racial discrimination, have documented inverse associations with telomere length conditional upon psychological factors, including greater depressive symptoms and perceptions of Anti-Black bias, in middle-aged African American men (Chae et al., 2014, 2016), or as part of a broader stress construct in pregnant Mexican-American women (Ruiz, Trzeciakowski, Moore, Ayers, & Pickler, 2017). Altogether, these findings provide initial evidence that self-reported discrimination may be implicated in the acceleration of telomere attrition.

1.2. Variations in telomere length as a function of social categories

Research has demonstrated sociodemographic variations in telomere length, which do not consistently reflect established variations in U.S. health disparities. For instance, some studies report that African Americans have longer telomeres than Whites from birth into adulthood (e.g., Rewak et al., 2014) but show a greater accelerated decline in older age (Hunt et al., 2008). Yet, there is also evidence that in middle to older age, African Americans have longer telomeres than Whites (Needham et al., 2013). With regard to sex, a meta-analysis demonstrated that men typically have shorter telomeres (Gardner et al., 2014); however, a study in middle-aged to older adults indicated that compared to Whites and men, African American women had the greatest attrition over time (Diez Roux et al., 2009). Similarly, middle-aged African American women were biologically 7.5 years older than White women of the same chronological age assessed by telomere length (Geronimus et al., 2010). While the overall evidence regarding SES and telomere length shows weak or null effects (Robertson et al., 2013), there is evidence that African Americans with higher SES have longer telomeres compared to Whites across all SES levels and African Americans with lower SES (Adler, 2013). For instance, data from the National Health and Nutrition Examination Survey (NHANES) demonstrated that less education was associated with shorter telomeres in African American and Whites, but no associations were observed with income. However, less income has been associated with shorter telomeres in midlife African American men (Schrock et al., 2018). Taken together, these data demonstrate variations in telomere length by social category, some of which are inconsistent with established health disparities. It is unknown whether the associations of these social statuses with telomeres are influenced by social factors such as discrimination.

1.3. Rationale for present study

The present study examines self-reported discrimination and social categories to understand if their interaction yields differential patterning in relation to telomere length. We seek to extend existing research in two ways. First, drawing upon an intersectionality framework, we examine the linkage of discrimination to telomere attrition as conditional upon multiple social categories, specifically, race, SES, age, and age. Health disparities research has begun to use this framework to highlight how interdependent social categories simultaneously converge to inform *lived* experiences, and in turn, shape health (Williams et al., 2012). Further, the Healthy People 2020 U.S. objectives set forth by the U.S. Department of Health and Human Services (2014), highlight the need for research on social statuses and discrimination to further elucidate health disparities. A prior report on discrimination and telomere length (Lee et al., 2017) observed that neither age nor sex moderated the associations between major life discrimination and telomere length in older African Americans. Perhaps concurrently considering age and sex alongside other social statuses may reveal different effects. Indeed, our group recently published a report using the present study's sample examining interactive relations between discrimination and sociodemographic variables with telomere length in race-stratified (i.e., within-race) analyses (Pantesco et al., 2018). Findings revealed within-race associations between discrimination and telomere length in African Americans and Whites that varied by age, sex, and/or SES. In light of previous research stressing the importance of examining both within-race and between-race effects in health disparities research (Whitfield, Allaire, Belue, & Edwards, 2008), the present study will expand on our previous work by examining these trends across both African Americans and Whites, including potential moderating effects of race.

Second, we extend the prior research by using a comprehensive examination of discrimination. Prior telomere reports have either focused explicitly on discrimination (e.g., major and/or everyday discrimination; Lee et al., 2017) or attributions for that discrimination (e.g., race, ancestry, or national origin; Liu & Kawachi, 2017 or racial discrimination (Chae et al., 2014, 2016;). Discrimination, however, is a multidimensional construct composed of various forms, experiences, and magnitudes, which in turn, may yield different links with telomere attrition. Health disparities scholars have strongly recommended examining a fuller spectrum of interpersonal discrimination. Although various forms of interpersonal discrimination would be expected to be moderately interrelated, they may also capture unique aspects of the experience of discrimination when concurrently assessed (Krieger, 2014; Lewis, Cogburn, & Williams, 2015). To this end, we assess three categories of interpersonal discrimination; 1) day-to-day, social status non-specific unfair treatment (everyday discrimination) 2) lifetime, social status specific (frequency of discrimination across sources, racial

and gender discrimination), and 3) lifetime burden, social status non-specific (lifetime discrimination burden). Whereas everyday discrimination assesses unfair treatment irrespective of the reason or attribution for the experience, and the lifetime burden measure captures the weight of an individual's full experience with discrimination, the assessments of social status specific forms of discrimination – e.g., racial and gender discrimination – reflect discrimination rooted in power differentials related to the sociohistorical marginalization of the targeted individual as a function of their low status group membership. To our knowledge, health disparities research has yet to concurrently examine multiple forms of discrimination within the context of social categories. We propose that there are interactive relations of each form of self-reported discrimination with race, SES, age, and sex in relation to telomere attrition. Directional hypotheses were not proposed a priori given the exploratory nature of the research.

2. Methods

2.1. Participants and procedure

Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) is an ongoing longitudinal study of disparities in health and disease attributable to race and SES. Evans et al. (2010) have previously detailed the design of the HANDLS study. Briefly, HANDLS participants are a fixed cohort of urban-dwelling adults, recruited via household screenings from an area probability sample of 13 census segments in the city of Baltimore, Maryland. The census segments were pre-determined for their likelihood of yielding representative samples of individuals who were African American and White, men and women, and with adjusted household incomes above and below 125% of the 2004 U.S. Department of Health and Human Services poverty guidelines. HANDLS participants self-identified as African American or White and were between 30–64 years of age at baseline. The Institutional Review Board at the National Institute of Environmental Health Sciences approved the HANDLS study protocol. 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, (8) had uncontrolled high blood pressure ($>$ 160/100).

The first wave of HANDLS occurred between 2004–2009 and consisted of two phases: (1) recruitment, written informed consent, and an interview in participants' homes, and (2) medical history, physical examination, and other assessments on mobile medical research vehicles parked within participants' neighborhoods (Evans et al., 2010). Of the 3720 participants selected for the original HANDLS cohort, 2802 completed both phases. A subset of these participants consented to DNA collection, of whom 360 with DNA in the biorepository from Waves 1 and 3 were randomly selected from a cross of race, sex, and baseline age (median-split) for telomere assays. The present study included 341 participants with valid data for relevant variables. Analysis-specific sample sizes varied slightly due to missing data on the different discrimination measures, ranging from 338 to 341 participants. A power analysis using the G*Power software (version 3.1; for more information, see Faul, Erdfelder, Lang, & Buchner, 2007) revealed that all analysis-specific sample sizes were adequately powered ($1 - \beta = 0.80$) to detect a small–medium Cohen's effect size of $f^2 = 0.06$ for the present analyses, which included a maximum of 12 predictor variables (see Statistical Analyses for a description of the regression models).

2.2. Measures

2.2.1. Discrimination

2.2.1.1. Everyday Discrimination. The Everyday Discrimination scale (Williams, Yu, Jackson, & Anderson, 1997) is a nine-item measure assessing the frequency of routine experiences of unfair treatment which does not require an explicit attribution (e.g., race) for the experience. Some examples of items are "being treated with less courtesy," "getting worse service at stores," or "people acting like you are not smart." Participants rated the frequency of their experiences on the following scale: (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". All responses were reversed scored, such that a score of 6 corresponded to "almost every day." Possible scores on this measure ranged from 9 to 54, with higher scores indicating greater everyday discrimination. This scale has previously been shown to have good internal consistency (e.g., $\alpha = 0.88$ in Williams et al., 1997), and internal consistency was similar in our study ($\alpha = 0.81$).

2.2.1.2. Gender and racial discrimination. Gender and racial discrimination were assessed with two measures used previously in epidemiologic research (e.g., Krieger, 1990). Gender discrimination was assessed by five items that asked whether participants have ever experienced gender discrimination at school, when getting a job, at work, at home, and when getting medical care. Racial discrimination was measured using a six-item inventory that assessed whether participants have ever experienced racial discrimination at school, when getting a job, at work, when getting housing, when getting medical care, and from police or in courts. For each item in both measures, participants could reply *Yes* (1) or *No* (0). Possible scores on the gender and racial discrimination scales ranged from 0 to 5 and 0–6, respectively, with greater summed scores indicating greater levels of discrimination. In our study, internal consistencies for the gender discrimination and racial discrimination scales were $\alpha = 0.74$ and $\alpha = 0.84$, respectively.

2.2.1.3. Sources of discrimination. Sources of discrimination were assessed with a ten-item measure adapted from a previous measure of discrimination in healthcare settings (LaVeist, Rolley, & Diala, 2003). Items asked, "Overall how much have you experienced prejudice or discrimination due to…" gender, race, ethnicity, income, age, religion, physical appearance, sexual orientation, health status, and disability. Participants rated their experiences on a 4-point scale ranging from 1 (*not at all*) to 4 (*a lot*). Scores ranged from 10 to 40, with higher scores indicating a higher number of sources of discrimination experienced more frequently. In our study, this scale had good internal consistency ($\alpha = 0.83$).

2.2.1.4. Lifetime discrimination burden. Lifetime discrimination burden was assessed with a two-item measure. Specifically, these items asked (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 ranging from 1 (*not at all*) to 4 (*a lot*). Possible scores ranged from 2 to 8, with higher scores indicating greater lifetime discrimination burden. These items have been used previously in epidemiological research (i.e., Jackson Heart Study, Friedman, Williams, Singer, & Ryff, 2009; & Survey of Midlife Development in the United Status, Sims, Wyatt, Gutierrez, Taylor, & Williams, 2009). The two items comprising this scale were strongly correlated in our study, $r = .80$, $p < 0.001$.

2.2.2. Sociodemographic information

Sociodemographic information was collected in the household interview component of Phase 1. Participants reported their age, sex, and self-identified race. Participants' SES was calculated from a composite

score that included self-reported annual household income and educational attainment. Participants were classified as *higher SES* if they reported (1) an annual household income (adjusted for household size) above or equal to 125% of the 2004 U.S. Department of Health and Human Services poverty guidelines, *and* (2) educational attainment greater than or equal to a high school diploma or GED. Participants were classified as *lower SES* if they reported (1) an annual household income (adjusted for household size) below 125% of the 2004 Health and Human Services poverty guidelines, or (2) educational attainment less than high school diploma or GED.

2.3. Telomere assay

Telomere length was measured by the quantitative polymerase chain reaction (qPCR)-based method described previously by Cawthon (2002). Briefly, 10 ng of DNA isolated from peripheral blood mononuclear cells (PBMC), was used in each PCR reaction in triplicates for each participant. Both telomere (T) and a single copy gene (*36B4*) (S) were included in the same 384-well plate using SYBR master mix on an Applied Biosystem 7900 H T system (ThermoFisher). The average cycle threshold (Ct) values of T and S were calculated from the triplicates to generate the average T/S ratio of each sample. To convert T/S ratio into actual telomere length in kilobases (kb), we measured one hundred thirty samples by both qPCR and the Southern method (Lin et al., 2015) and used the resulting conversion equation to calculate telomere length in kb from the T/S ratio.

2.4. Adjustment variables

Depression symptoms, lifetime substance use burden, and waist circumference were selected as adjustment variables based on the inclusion of similar variables in past studies of telomere length (Beach, Lei, Brody, Yu, & Philibert, 2014; Puterman et al., 2016; Wolkowitz et al., 2011). The Center for Epidemiologic Studies Depression (CES-D) scale (Radloff, 1977) was administered to participants during Phase 2. The CES-D is a 20-item inventory used to assess depressive symptoms over the past week. Participants responded to each item on a 4-point scale ranging from 0 (Rarely) to 3 (*Mostly*). Possible scores ranged from 0 to 60, with higher scores indicating greater depressive symptomatology.

Waist circumference in centimeters (cm) and substance use history were collected during Phase 2. Participants reported their substance use history during the broader medical history assessment. For any specified substance of abuse, participants could reply with one of four response options: *Never tried*, *Tried, never used regularly*, *Former user (Used > 6 months ago)*, or *Current user (Used in past 6 months)*. In the present study, responses for cigarette, marijuana, cocaine/crack, and opiate use were collapsed into dichotomous variables that were coded as 0 (*Never used*; i.e., never tried or tried, never used regularly) and 1 (*Ever used*; i.e., former or current user). Dichotomous scores for each of the four substances were summed to compute a *lifetime substance use burden* variable. Scores ranged from 0 to 4, with higher scores indicating greater lifetime substance use burden.

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 (i.e., using age, sex, race, and poverty status as predictors) was used for imputation for the purpose of replicability.

2.5. Statistical analyses

Statistical analyses were conducted with the Statistical Package for the Social Sciences (SPSS) version 24. Multiple linear regression modeling was used to examine interactive relations of discrimination, SES, and other sociodemographic factors with telomere length. Specifically, we were interested in whether the interaction of discrimination and SES would vary as a function of age, sex, or race to predict telomere length. Therefore, we examined interaction effects up to the three-way interaction level, which included (a) self-reported discrimination scores, (b) SES, and (c) age, sex, or race. All analyses began with fully adjusted models, which contained a three-way interaction effect, two-way interaction effects nested beneath it, as well as all main effects and adjustment variables. If the three-way interaction effect was significant, the fully adjusted model was retained. Conversely, if the three-way interaction effect was nonsignificant, data analysis proceeded through the backward elimination procedure, which guides removal of nonsignificant, higher-level interaction terms from regression analyses (Morrell, Pearson, & Brant, 1997). Consistent with the procedure, the three-way interaction was removed from the regression model if found to be to nonsignificant, and analyses were then rerun. Subsequently, significant two-way interactions were identified and retained in the next step, while nonsignificant two-way interactions were removed from analysis. If no significant three- or two-way interactions were identified in the previous steps, then regression analysis proceeded with only main effects and covariates. Finally, the PROCESS macro for SPSS, Version 2.16 (Hayes, 2013) was used to probe and visualize significant two- and three-way interaction effects.

3. Results

African Americans reported significantly greater gender discrimination, racial discrimination, frequency of discrimination across sources, and lifetime discrimination burden than their White counterparts (all p's < .001; see Table 1). African Americans also reported greater lifetime substance use burden than Whites ($t(339) = -2.13$, $p < 0.05$), whereas Whites had a higher waist circumference than African Americans ($t(339) = 4.29$, $p < 0.001$). There were no racial differences in sociodemographic factors (i.e., age, sex, and SES variables), everyday discrimination, or telomere length (all p's > .05). Unadjusted bivariate correlations between discrimination measures ranged from $r = 0.31$ to $r = 0.70$ (all p's < .01; see Supplementary Table 1 for correlations among all study variables). Overall, telomere length in the present sample ranged from 2.60 to 8.50 kb.

Findings revealed four significant three-way interactions: (a) Sex × SES × Lifetime Discrimination Burden, $b = -0.23$, $p = .011$; (b) Sex × SES × Gender Discrimination, $b = -0.29$, $p = .040$; (c) Sex × SES × Racial Discrimination, $b = -0.24$, $p = .023$ (see Supplementary Table 2 for full regression model results). As shown in Fig. 1, among women with higher SES, shorter telomeres were associated with greater (a) lifetime discrimination burden, $b = -0.17$, $p = .003$ (b) gender discrimination, $b = -0.30$, $p = .001$; and (c) racial discrimination, $b = -0.30$, $p < .001$. Across these three measures, every 1-point increase in discrimination was associated with a 0.17–0.30 kb decrease in telomere length among women with higher SES. Discrimination was not associated with telomere length among women with lower SES, or men of either SES group (all p's > .05). Other models with three-way interaction terms were nonsignificant, thus, three-way interactions were eliminated from all subsequent models.

Next, findings revealed a significant two-way interaction of Age × Frequency of Discrimination across Sources with telomere length, $b = .002$, $p = 0.008$ (see Supplementary Table 3 for full regression results). As depicted in Fig. 2, among younger participants (38.87 years), greater frequency of discrimination across sources was related to shorter telomeres, $b = -0.02$, $p = 0.020$. Frequency of discrimination across sources was not associated with telomere length among middle-aged (47.78 years), $b = -0.01$, $p = .458$, or older participants (56.69 years), $b = 0.01$, $p = .173$.

Everyday discrimination was not associated with telomere length, neither as a main effect nor within interactions. However, backward elimination of nonsignificant interaction terms in the everyday discrimination models revealed a significant two-way interaction of Sex × Race with telomere length, $b = 0.35$, $p = .025$ (Supplementary

Table 1
Participant Characteristics in the Overall Sample and Stratified by Race.

	African American (n = 176)	White (n = 165)	All (n = 341)
% African American	—	—	51.6%
% Women	48.3%	52.1%	50.1%
% Lower SES [a]	61.4%	63.6%	62.5%
% < High school diploma or GED	29.0%	37.6%	33.1%
% < 125% federal poverty level	50.0%	51.5%	50.7%
Age	47.57 (± 9.40)	47.89 (± 8.41)	47.72 (± 8.92)
Depressive symptoms	13.52 (± 9.86)	15.56 (± 11.54)	14.51 (± 10.74)
Waist circumference (cm) ***	95.48 (± 16.97)	103.71 (± 18.39)	99.46 (± 18.12)
Lifetime substance use burden [b] *	1.64 (± 1.24)	1.36 (± 1.17)	1.50 (± 1.21)
Everyday discrimination	21.15 (± 7.88)	19.73 (± 7.81)	20.46 (± 7.86)
Gender discrimination ***	0.96 (± 1.37)	0.32 (± 0.74)	0.65 (1.16)
Racial discrimination ***	1.78 (± 1.96)	0.34 (± 0.91)	1.09 (± 1.70)
Sources of discrimination [c] ***	17.84 (± 6.31)	14.93 (± 4.60)	16.43 (± 5.73)
Lifetime discrimination burden ***	3.94 (± 1.90)	2.93 (± 1.54)	3.45 (± 1.80)
Telomere length (kb)	5.62 (± 0.75)	5.69 (± 0.69)	5.66 (± 0.72)

Note. Racial differences in all study variables were examined with independent samples *t*-tests and chi-square tests of independence. * $p < .05$, ** $p < 0.01$, *** $p < .001$.

[a] Participants were considered to have *lower SES* if they reported having an educational attainment less than a high school diploma or GED and/or adjusted household incomes below 125% of the 2004 federal poverty level.

[b] Number of substances (cigarettes, marijuana, cocaine/crack, or opiates) participants ever used.

[c] Refers to frequency of discrimination across sources.

Table 4). As depicted in Supplementary Fig. 1, African American women had shorter telomeres than African American men, $b = 0.45$, $p < .001$. In contrast, there were no significant differences in telomere length between White women and White men, $b = .10$, $p = 0.353$. In addition, there was a significant main effect of age in this model (as well as all other models), such that greater age was related to shorter telomeres, $b = -0.01$, $p = 0.025$. Notably, findings revealed no racial differences in telomere length, neither as a main effect nor within interactions (all p's > .05).

4. Discussion

In a sample of middle-aged African American and White adults, women with higher SES and younger adults (38.87 years old) reporting greater exposure to discrimination had shorter telomeres. Specifically, greater lifetime discrimination burden and gender and racial discrimination were each associated with shorter telomere length among women with higher SES. Among younger participants, greater frequency of discrimination across sources was associated with shorter telomeres. Associations were independent of race, as well as depressive

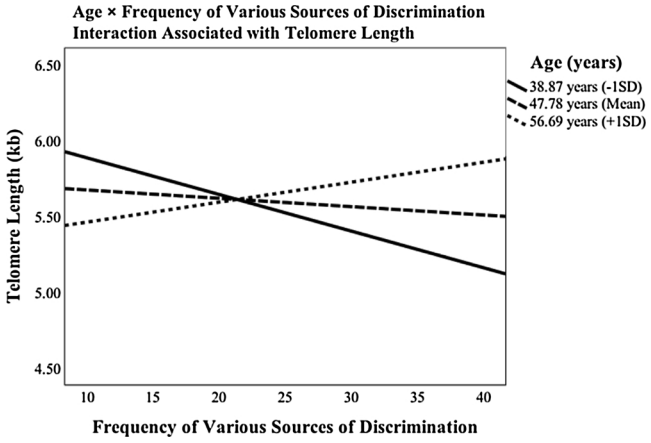

Fig. 2. Significant moderating effect of age on the association between frequency of discrimination across various sources and telomere length.

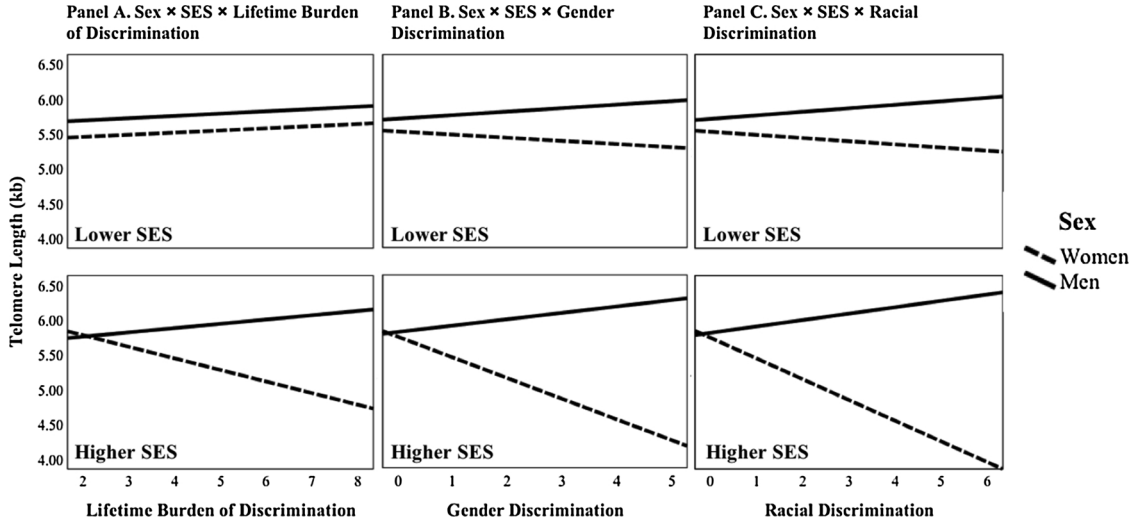

Fig. 1. Significant moderating effects of sex and SES on the association between telomere length and (A) lifetime discrimination burden, (B) gender discrimination, and (C) racial discrimination.

symptoms, waist circumference, and substance use. These findings highlight the importance of considering the interwoven nature of historically demarcated social categories with the social experience of discrimination, as well as how these linkages may bear upon health.

The current findings extend the applications of extant stress theories (Clark, Anderson, Clark, & Williams, 1999; Paradies, 2006) and complement previous work showing inverse links between discrimination and specifically, racial discrimination and telomere length (Chae et al., 2014, 2016; Lee et al., 2017; Liu & Kawachi, 2017). They also expand upon prior reports in several ways. Most studies of discrimination and telomere length do not consider additional contextual factors, such as race, SES, sex, and age, especially in combination with each other. In the current study, discrimination was associated with shorter telomeres in both African American and White women with higher SES. Elevated health risk among those falling at the intersection of a high status (e.g., high SES) and low status social category (e.g., female sex) may represent what Bowleg (2012) refers to as an intersectionality paradox. These data reflect a pattern similar to the work showing diminishing returns for African Americans ascending the ranks of SES who experience poorer, not better, health (Diez-Roux, Nieto, Tyroler, Crum, & Szklo, 1995; Farmer & Ferraro, 2005; Waldstein et al., 2016). Diminishing returns is posited to be influenced by the price of economic progress for a minority group. Specifically, achieving greater SES typically situates African Americans in predominately White settings, which may lead to heightened exposure to chronic, interpersonal discrimination and, in turn, compromised health. A similar pattern may be at play in women with higher SES. Greater access by way of higher SES may increase the likelihood that these women are met with discriminatory interactions steeped in traditional expectations of gender roles. This dynamic may pertain to both African American and White women due to the shared challenges arising from male privilege. For instance, as women continue to make gains in male-dominated settings (Pew Research Center, 2013), men increasingly see themselves as disadvantaged and see women as becoming more advantaged at their expense (Kehn & Ruthig, 2013). Such an orientation may be particularly aversive for women with higher SES. It is also plausible that some women are mistreating other women (Reuben, Sapienza, & Zingales, 2014), which could be a consequence of competition for resources in a patriarchal society.

Reports of racial discrimination were associated with shorter telomeres among women with higher SES, irrespective of race. The evidence that African American women with higher SES reported higher levels of racial discrimination and had shorter telomeres is not surprising. Indeed, it is consistent with the literature showing the physical costs of racism among African Americans overall (e.g., Paradies, 2006), and particularly those with more socioeconomic resources (Farmer & Ferraro, 2005).

This association was also observed in White women with higher SES, which was an unexpected finding. Several explanations may exist for this finding (e.g., Apfelbaum, Norton, & Sommers, 2012; Craig & Richeson, 2017; DiAngelo, 2011; Norton & Sommers, 2011; Wilkins & Kaiser, 2013). Of late, a growing number of Whites in the U.S. have been reported as perceiving an increase in Anti-White bias and racial discrimination toward their group (see report byNNational Public Radio, Robert Wood Johnson Foundation, & Harvard T. H. Chan School of Public Health, 2017; Norton & Sommers, 2011). In addition, these perceptions have been linked with poor health outcomes in Whites (e.g., Peterson, Matthews, Derby, Bromberger, & Thurston, 2016. Thus, researchers have sought to elucidate the underlying psychological mechanisms for these attributions to racial discrimination among Whites. Individual construal's of explanations for mistreatment in interpersonal interactions are in part shaped by social and cultural ideologies and the broader societal milieu. In this regard, an emerging body of work highlights how the current and impending demographic shift in the U.S. – wherein by 2050 Whites will be the "majority-minority" – are contributing to concerns of a fundamental change in American culture (Craig & Richeson, 2017). Thus, perceptions of progress among racial minorities have been found to stoke concerns of destabilization of the traditional social hierarchy among non-racial minorities (Wilkins, Hirsch, Kaiser, & Inkles, 2016). Nevertheless, altogether, these reports and the emerging linkages to health outcomes are unfolding in a context in which racial minorities have long fared poorly across multiple domains (e.g., health, education, criminal justice, and wealth; Alexander, 2010; Hoffman, Trawalter, Axt, & Oliver, 2016; Pager & Shepherd, 2008; Washington, 2006), indicative of embedded multilevel racial discrimination, which are largely not observed in Whites.

The second novel finding was that greater frequency of discrimination across sources was associated with shorter telomere length among younger participants. One prior study (Lee et al., 2017) reported that age did not modify the linkage of major life discrimination to telomeres, but the sample consisted of older African Americans. While telomere length declines with age (Epel, 2009), the current findings suggest that social factors may be associated with telomere length earlier in the life course and could possibly point to a cascading stress-health effect emerging in young adulthood. Considering the shared meaning of youth across race could shed light on these findings. Urban enclaves around the U.S. report an increase in racial, political, economic, and cultural frustration (Dobuzinskis, 2015). Younger Americans of different races may be acutely aware of these tensions. This may raise vigilance for bias, whether the bias is actual or not (Sewell, Horsford, Coleman, & Watkins, 2016), possibly explaining why these associations emerged irrespective of race. Given data showing that telomere shortening may contribute to accelerated aging and that racial minorities experience an earlier onset of poorer health, as well as emerging research highlighting health disparities in middle-aged Whites (Case & Deaton, 2015), examination of the relationship between discrimination and health earlier in the life course is an important next step.

4.1. Limitations, strengths, and future directions

The study has some limitations. First, the data were cross-sectional, and conclusions regarding causation, as well as the temporal links among the factors, are not possible. Future work should examine discrimination and telomere length within the context of social categories across the life course to establish temporal patterns. Such data may also shed light on epidemiological inconsistencies, such as African Americans having longer telomeres than Whites throughout the lifespan. Second, this work focused explicitly on individual-level discrimination. While the measures employed share a moderate amount of variance because they represent dimensions of the same underlying latent construct, they also have substantial unique variance. Assessing other forms of discrimination, especially at the structural-level, may lead to a better understanding of health disparity trajectories. Third, the study sample size was small (n = 341), which raises potential power-related concerns. Given that underpowered studies can produce biased findings (Crutzen & Peters, 2017; Simonsohn, 2015), ensuring that studies are powered adequately for the analyses being conducted is an important consideration. In the present study, we ran a power analysis (see Methods) that revealed our sample was powered to detect a small–medium f^2 effect size, suggesting that our sample, although small, was acceptable for drawing conclusions from the present analyses.

However, given the sample size, we opted to not test beyond 3-way interaction models. Future studies with larger samples should examine interactions that are more complex and investigate additional social categories, such as sexual orientation, in more depth. Fourth, there is no established analytical strategy for examining intersectionality. While interaction modeling allows some insight into how social categories may influence each other, it may not fully capture subtle nuances in these linkages (Cole, 2009). Fifth, findings may not be generalizable to African Americans and Whites living in non-urban settings. Different

communities facilitate different types of social interactions and, in turn, may yield different linkages between discrimination and health. Finally, exploring the factors underlying perceptions of unfair treatment in different social groups will be important in future work.

Our study has several strengths. Participants were recruited from an area probability sample representing an economically diverse group of working aged African American and **white** adults. Participants included in our analyses were sampled randomly from the parent study. In addition, we assessed several specific forms of discrimination. In line with a conceptualization of discrimination as a multidimensional construct, we examined various forms of discrimination such as lifetime burden and gender. Our analyses used an intersectional approach to examine complex interactions among race, SES, sex, and age.

Our findings from midlife adults in an urban setting suggest a need for more research on the potential effects of discrimination and social statuses on telomere length. We investigated various forms of discrimination and showed that less commonly studied types matter. Our results also point to the value of considering an intersectional approach when examining discrimination and health endpoints which influence the perceptions and management of unfair treatment. Speculatively, the difference in telomere length across women with higher SES and younger adults, suggests a physiological age deterioration for these individuals when reporting greater exposure to particular types of discrimination. Thus, these subgroups may assume health trajectories that are paradoxical to what is expected as a function of social statuses they occupy (Rehkopf et al., 2016). In the absence of telomerase activity that would allow estimation of metric of years lost physiologically, the current findings are underscored by prior studies demonstrating that the difference in telomere length intimates impending health outcomes (Cherkas et al., 2006; Geronimus et al., 2010).

5. Conclusions

The current findings show that various forms of interpersonal discrimination are associated with accelerated biological aging, as indexed by telomere length, among African American and White adults in the U.S. In concurrently demonstrating the relevance of multiple forms of interpersonal discrimination this work may promote the conceptualization of discrimination as a multidimensional construct, which has unique effects in groups falling at the intersections of multiple statuses. Here, such an approach uncovered a subgroup (specifically, African American and White women with higher SES) with the strongest evidence of biological aging in relation to discrimination, which may have relevance for understanding future patterns of health risk as women continue to ascend the SES ladder. The observation that race may not always contribute to differential associations between discrimination and health endpoints demonstrates a need to more comprehensively assess ideological values that underlie expectations of treatment, opportunity, and fairness. In sum, if telomeres function as "psychobiomarkers," reflecting exposure to discrimination-related stress at the cellular level, the current findings hold promise in revealing linkages to later life health disparities in understudied subgroups.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.biopsycho.2018.12.004.

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Supplementary Table 1.

Correlations among All Study Variables

	1	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Everyday discrimination	1	.31**	.33**	.40**	.34**	-.03	-.002	.09	.05	.31**	.06	.08	-.06
2. Gender discrimination		1	.70**	.56**	.61**	.04	-.08	.28**	.03	.10	-.05	.02	-.09
3. Racial discrimination			1	.54**	.64**	.06	.16**	.42**	-.01	-.01	-.12	.12*	-.01
4. Sources of discrimination				1	.57**	.12*	-.02	.26**	-.003	.15**	-.01	.08	-.07
5. Lifetime discrimination burden					1	.15**	.06	.28**	-.04	.08	-.01	.01	-.01
6. Age						1	.02	-.02	.02	-.07	.15**	.01	-.15**
7. Sex							1	.04	-.01	-.21**	-.02	.19**	.19**
8. Race								1	-.02	-.10	-.23**	.12	-.05
9. SES									1	.26**	-.08	.13*	-.06
10. Depressive symptoms										1	.11	.11	-.06
11. Waist circumference											1	-.15**	-.07
12. Lifetime substance use burden												1	.03
13. Telomere length													1

Note. * $p < .05$, ** $p < .01$

Supplementary Table 2

Multiple Regression Models Estimating 3-way Interactive Relations of Sex, SES, and Varying Forms of Discrimination with Telomere Length.

(a) Sex × SES × Lifetime Discrimination Burden

Model predictors	*b*	*se*	*p*	$\eta^2_{partial}$
Age*	-0.01	.004	.017	.017
Race	-0.13	.08	.123	.007
Depressive symptoms	-0.001	.004	.792	<.001
Waist circumference	-0.002	.002	.288	.004
Lifetime substance use burden	0.01	.03	.703	<.001
Sex	-0.48	.28	.086	.009
SES*	-0.72	.25	.004	.024
Lifetime discrimination burden **	-0.17	.06	.003	.028
Sex × SES *	0.71	.34	.040	.013
Sex × Lifetime Discrimination Burden **	0.23	.07	.001	.031
SES × Lifetime Discrimination Burden **	0.20	.07	.003	.162
Sex × SES × Lifetime Discrimination Burden *	-0.23	.09	.011	.020

(b) Sex × SES × Gender Discrimination

Model predictors	*b*	*se*	*p*	$\eta^2_{partial}$
Age *	-0.01	.004	.014	.019
Race	-0.11	.08	.196	.005
Depressive symptoms	<0.001	.004	.922	<.001
Waist circumference	-0.002	.002	.262	.004

Lifetime substance use burden	0.004	.03	.883	<.001
Sex	0.07	.15	.634	.001
SES	-0.21	.13	.112	.008
Gender discrimination **	-0.30	.09	.001	.035
Sex × SES	0.12	.18	.524	.001
Sex × Gender Discrimination **	0.39	.12	.001	.032
SES × Gender Discrimination *	0.25	.10	.013	.019
Sex × SES × Gender Discrimination *	-0.29	.14	.040	.013

(c) Sex × SES × Racial Discrimination

Model predictors	*b*	*se*	*p*	$\eta^2_{partial}$
Age *	-0.01	.004	.016	.018
Race	-0.12	.09	.168	.006
Depressive symptoms	-0.001	.004	.751	<.001
Waist circumference	-0.002	.002	.284	.004
Lifetime substance use burden	0.02	.03	.628	.001
Sex	0.10	.15	.515	.001
SES *	-0.26	.13	.048	.012
Racial discrimination ***	-0.30	.08	<.001	.048
Sex × SES	0.06	.19	.767	<.001
Sex × Racial Discrimination ***	0.31	.09	<.001	.038
SES × Racial Discrimination **	0.30	.09	.001	.036
Sex × SES × Racial Discrimination *	-0.24	.10	.023	.016

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 3

Multiple Regression Model Estimating the Two-way Interaction of Age × Sources of Discrimination with Telomere Length

Model predictors	*b*	*se*	*p*	$\eta^2_{partial}$
Age **	-0.04	.01	.001	.032
Sex ***	0.30	.08	<.001	.040
Race	-0.11	.08	.114	.006
SES *	-0.10	.08	.250	.004
Depressive symptoms	0.001	.004	.890	<.001
Waist circumference	-0.004	.002	.107	.008
Lifetime substance use burden	0.002	.03	.941	<.001
Sources of discrimination **	-0.11	.04	.007	.022
Age × Sources of Discrimination **	0.002	.001	.008	.021

Note. * $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 4

Multiple Regression Model Estimating the Two-way Interaction of Sex × Race and the Main Effect of Age with Telomere Length

Model predictors	*b*	*se*	*p*	$\eta^2_{partial}$
Age **	-0.12	.004	.008	.021
Sex	0.10	.11	.353	.003
Race *	-0.27	.11	.015	.018
SES	-0.09	.08	.300	.003
Depressive symptoms	<0.001	.004	.924	<.001
Waist circumference	-0.002	.002	.341	.052
Lifetime substance use burden	-0.01	.03	.831	<.001
Everyday discrimination	-0.01	.01	.342	.003
Sex × Race *	0.35	.16	.025	.015

Note. * $p < .05$, ** $p < .01$, *** $p < .001$