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Authors

Hailu, Elleni

Lewis, Tené

Needham, Belinda

et al.

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Longitudinal Associations Between Discrimination, Neighborhood Social Cohesion, and Telomere Length: The Multi-Ethnic Study of Atherosclerosis

Elleni M. Hailu, MPH,^{1,○} Tené T. Lewis, PhD,^{2,○} Belinda L. Needham, PhD,³ Jue Lin, PhD,⁴ Teresa E. Seeman, PhD,⁵ and Mahasin S. Mujahid, PhD¹

¹Division of Epidemiology, School of Public Health, University of California Berkeley, USA. ²Department of Epidemiology, Rollins School of Public Health, Emory University, Atlanta, Georgia, USA. ³Department of Epidemiology, School of Public Health, University of Michigan, Ann Arbor, USA. ⁴Department of Biochemistry and Biophysics, University of California San Francisco, USA. ⁵Department of Geriatrics, David Geffen School of Medicine, University of California Los Angeles, USA.

*Address correspondence to: Elleni M. Hailu, MPH, Division of Epidemiology, School of Public Health, University of California Berkeley, 2121 Berkeley Way, West #5302, Berkeley, CA 94720, USA. E-mail: ehailu@berkeley.edu

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Abstract

Background: We aimed to examine if neighborhood social cohesion moderated longitudinal associations between baseline reports of discrimination and 10-year changes in leukocyte telomere length (LTL).

Methods: Data are from the Multi-Ethnic Study of Atherosclerosis ($N = 1064$; age range 45–84 years). Baseline discrimination was measured using the Major Experiences of Discrimination Scale (MDS; none, 1 domain, ≥ 2 domains) and the Experiences of Discrimination Scale (EDS; none, moderate, high). Neighborhood social cohesion at baseline was assessed via a community survey within census tract–defined neighborhoods. 10-year change in LTL was defined as regression to the mean–corrected 10-year difference in the ratio of telomeric DNA to a single-copy gene (T/S).

Results: In linear mixed-effects models, we found that neighborhood social cohesion modified the effect of baseline reports of MDS on 10-year changes in LTL, independent of sociodemographic characteristics, health behaviors, and health conditions ($p(\chi^2) = .01$). Among those residing in neighborhoods with low social cohesion, experiencing major discrimination in ≥ 2 domains was associated with faster LTL attrition over 10 years, compared to reporting no discrimination ($\beta = -0.03$; 95% confidence interval: $-0.06, -0.003$). We found no main associations for either discrimination measure and no interaction between EDS and neighborhood social cohesion.

Conclusions: Results indicate that neighborhood social cohesion is an important dimension of the neighborhood context that may moderate the impact of major experiences of discrimination on telomere length attrition. These findings help advance our understanding of the integral role that neighborhood environments play in attenuating the effect of discrimination on accelerated cell aging.

Keywords: Discrimination, Multilevel stress, Neighborhood social cohesion, Telomeres

Discrimination, broadly defined as the unfair and unjust treatment of a group or an individual based on their sociopolitically ascribed identities, such as race, is a pervasive problem that continues to directly and indirectly hinder the health and well-being of marginalized populations (1). In addition to limiting an individual's access to material and psychosocial resources that promote healthy behaviors, discrimination has been shown to influence numerous ill-health indicators, such as diabetes mellitus (2), hypertension (3,4), cardio-

vascular diseases (5), inflammation (6), and a range of poor mental health conditions (7). Chronic experience of unfair treatment and the repeated activation of biologic stress response over time lead to physiologic wear and tear and the dysregulation of multiple organ systems, which then put individuals at an elevated risk for a myriad of adverse health outcomes (8,9).

One measure of physiologic wear and tear that may elucidate the biological pathways through which discrimination is embodied is

telomere length. Telomeres are protective caps made of DNA–protein complexes that prevent the ends of the human chromosome from deterioration. Telomere length is a known marker of biological aging, and short telomeres have been associated with increased risk of poor health, including several cardiometabolic disorders (10–12), cognitive decline (13), and even premature mortality (14–16). While telomeres naturally shorten due to age from cellular division, a significant body of research has consistently shown that telomere length may shorten as a result of sustained psychosocial stress via maladaptive physiologic stress response systems (17,18). Although findings for main-effects associations have been mixed, early evidence from studies investigating the link between discrimination and telomere length suggests that in specific population subgroups, experiencing discrimination may be associated with short telomeres (19). For example, in a recent cross-sectional study, Sullivan et al. (20) found that everyday discrimination was associated with short telomeres among Black and White women, while Lee et al. (21) found that major lifetime discrimination was cross-sectionally linked with short telomeres among older Black adults (mean age; 70 years). In addition to inconsistent findings, this body of work remains limited by the primary use of cross-sectional data, which restricts the ability to establish temporal relationships and explicate time-dependent mechanisms (19). Furthermore, the multilevel contexts that may play important roles in the relationship between discrimination and its adverse health consequences remain less well understood (1).

Neighborhood environments, especially neighborhood social environments, are important factors to consider in assessing the negative impacts of discrimination on disease risk and aging biomarkers. Positive neighborhood social environments, often characterized by strong social connectedness and collective efficacy, may have potential protective effects against chronic psychosocial stressors, including discrimination. Neighborhood social cohesion, a tenet of collective efficacy and a reflection of mutual trust within residents of a neighborhood, has been documented to have health rewards across a variety of outcomes (22,23). Neighborhoods with strong social cohesion allow their residents to garner a sense of community, belongingness, and support, creating a healthy coping environment from stressors both in and outside of their neighborhood (24,25). In other words, individuals who live in neighborhoods with high social cohesion may be able to practice beneficial coping strategies when faced with different social adversities, including discrimination, hence ameliorating their potential negative health consequences (26).

These findings underscore the need to more closely examine whether the relationship between discrimination and health manifests differently based on the multilevel contexts that surround individuals, in order to identify structural factors that may heighten the health impacts of discrimination, to promote healthy living and aging, and to ultimately eliminate pervasive health inequities. Furthermore, it is important to assess this link in racially and ethnically diverse populations using rich longitudinal data, to elucidate temporal associations, reduce study biases, and improve the generalizability of study findings (27,28). However, to the best of our knowledge, no study to date has assessed the relationship between experiences of discrimination, neighborhood social environments, and telomere length change over time. To address these important gaps, this study leveraged longitudinal and multilevel data from the Multi-Ethnic Study of Atherosclerosis (MESA) to determine whether the association between discrimination and the rate of leukocyte telomere length (LTL) attrition over 10 years was moderated by neighborhood social cohesion. We hypothesized that individuals reporting discrimination would experience faster telomere length attrition and that associations would be more pronounced among those

residing in neighborhoods with low social cohesion, compared to those living in highly cohesive neighborhoods.

Method

Study Sample

We utilized data from the MESA. Details about MESA's study design are described in depth elsewhere (29). In short, MESA is a cohort of 6814 older adults aged 45–84 years, recruited across 6 sites in the United States: Baltimore, MD; Chicago, IL; St. Paul, MN; Los Angeles, CA; New York, NY; and Forsyth County, NC, between the years 2000 and 2002. Participants were free of clinical cardiovascular disease at baseline and subsequent study waves occurred in approximate 2-year intervals. Data for the current analyses are from a random subset of MESA participants who were selected into the Stress Ancillary Study. This subset included White, Black, and Hispanic/Latinx participants from New York, NY and Los Angeles, CA with stored blood samples ($N = 1295$) at both Exam I (2000–2002) and Exam V (2010–2011). Participants were included in the final analytic subsample if they had 2 waves of data from both study waves (Exam I and Exam V) and did not have missing observations in the variables considered for these analyses ($N = 1064$). Across all study covariates, participants who had missing data had similar characteristics as those included in the analyses. The Institutional Review Boards of MESA field centers and the MESA coordinating center approved this study.

Study Variables

Change in LTL

Telomere length was assessed using quantitative polymerase chain reaction from leukocytes in peripheral blood samples collected at baseline (Exam I) and Exam V (30). Blood samples were stored at -80°C and assayed as a single batch at the University of California San Francisco. Telomere length was defined as the ratio of telomeric DNA (T) to a single-copy gene (S). Details regarding the protocol used to obtain telomere length are described elsewhere (31). Briefly, baseline and 10-year follow-up samples from each participant were assayed on the same plates and the same batch of assay reagents were used for all samples. Each of the DNA samples was assayed 3 different times on 3 separate days and each assay included 8 control DNA samples to normalize run-to-run variation. Each successfully assayed sample had 6 T/S values, of which the mean was obtained after removing outliers (0.2% of all the assayed observations). The intraassay coefficient of variation for the samples was 2.9%. Consistent with prior work, change in telomere length was calculated adjusting for regression to the mean, which could cause potential bias due to residual errors in baseline telomere length measurement (32). Given that controlling for baseline telomere length in our regression models may result in additional bias, we instead used the following formula developed by Verhulst et al. (33) to calculate 10-year changes in telomere length accounting for regression to the mean.

$$\Delta\text{LTL} = -1 \left[\rho \left(\text{LTL}_{\text{Exam I}} - \overline{\text{LTL}_{\text{Exam I}}} \right) - \left(\text{LTL}_{\text{Exam V}} - \overline{\text{LTL}_{\text{Exam V}}} \right) \right]$$

$$\rho = \frac{2rSD_{\text{Exam I}}SD_{\text{Exam V}}}{SD_{\text{Exam I}}^2 + SD_{\text{Exam V}}^2}$$

$$r = \text{corr}(\text{LTL}_{\text{Exam I}}, \text{LTL}_{\text{Exam V}})$$

SD = standard deviation; r = Pearson correlation coefficient.

Discrimination

To assess participants' reports of discrimination at baseline, we used modified versions of the Major Experiences of Discrimination Scale (MDS) and the Everyday Discrimination Scale (EDS), which were originally developed for the Detroit Area Study (34).

The MDS assesses the occurrence of lifetime discrimination in participants' lives across 6 major domains: school, job/the workplace, housing, neighborhood, and encounters with the police. For example, participants are asked about being unfairly fired from a job or being physically threatened or abused by the police. Responses were recorded as yes/no, from which a summary score of participants' affirmative replies was summed to create a total score ranging from 0 to 6. Based on the distribution of responses and consistent with prior work, we then categorized this score into 3 groups (did not experience discrimination, experienced discrimination in 1 domain, and experienced discrimination in 2 or more domains) (2,5,6).

The EDS asks participants about the frequency of unfair treatment in their everyday lives, such as being threatened/harassed and being treated less than others, using 9 items on a Likert scale. Responses ranged from almost every day (1) to never (6). Mean scores, of which higher values indicated increased discrimination, were obtained after each of the items of the scale were reverse coded, summed, and averaged. In order to capture both potential linear and nonlinear relationships, we use both the continuous and categorical version (None [mean score = 1], Moderate [mean score >1 and ≤2], and High [mean score >2]) of the EDS in the following analyses.

The MDS and the EDS are widely used measures that have been validated in racially/ethnically diverse populations to capture experiences of discrimination (34). They have both demonstrated good reliability in capturing the latent construct and have demonstrated good internal consistency in this population (EDS Cronbach's alpha = 0.88).

Neighborhood social cohesion

Neighborhood social cohesion at baseline within census tracts was assessed using a community survey that asked individuals who lived in the same census tracts as MESA participants to rate the social environment within a mile from their place of residence using 4 items (35). Participants were asked about their perceptions on the presence of harmony, willingness to help, shared community values, and trust in their neighborhoods. Responses were on a Likert scale, ranged from strongly agree (1) to strongly disagree (5), and were reverse coded and summed. Conditional Empirical Bayes (CEB) estimates were utilized to average these items across all participants and to create a neighborhood-level measure of social cohesion, whereby higher values signified increased neighborhood social cohesion. The CEB estimate allows us to borrow information from other neighborhoods when data are sparse, shrinks less reliable scores on the scale toward the mean, and adjusts for respondent characteristics such as age, thereby increasing the validity of the measure. This measure has been shown to have good internal consistency, strong intraneighborhood correlation, and reliable ecometric property in appropriately capturing the area-level construct in this sample (Cronbach's alpha = 0.72) (22). To facilitate a clearer interpretation, we categorized this measure into 2 groups (low neighborhood social cohesion and high neighborhood social cohesion) using a median split (36).

Study Covariates

In order to account for possible confounding, based on prior literature, we adjusted our estimates for the following self-reported covariates that were collected via study questionnaire at baseline: age, gender (man, woman), race/ethnicity (White, Black/African American, Hispanic/Latinx), educational status (high school or less, some college/technical degree, university graduate), employment (unemployed, employed), household income in US dollars (<\$20 000, \$20 000–49 999, \$50 000–74 999, ≥\$75 000), and marital status (married, not married) (37).

Consistent with previous studies on the possible links between health behaviors and health conditions that may be conceptualized as potential mediators of the relationship between discrimination, neighborhood social cohesion, and telomere length (38,39), we also additionally included the following baseline covariates in our analyses: body mass index (kg/m²), diabetes mellitus (yes, no), hypertension (yes, no), cancer (yes, no), pack-years of smoking (the product of the self-reported number of packs of cigarette smoked per day and the number of years smoked), moderate to vigorous physical activity (the metabolic equivalent task of physical activity minutes per week), Chronic Burden Score (range = 1–5), and Center for Epidemiologic Studies—Depression score (range = 0–60).

Statistical Analyses

To examine bivariate relationships between telomere length measurements at the 2 study waves and values of study covariates, we calculated descriptive statistics. We also examined the distribution of study covariates across major discrimination categories and determined mean everyday discrimination scores across population characteristics at Exam I. Linear regression models were used to examine associations between discrimination measures and telomere length change. To assess if the relationship between reports of discrimination and telomere length change varied across neighborhood social cohesion categories, we used hierarchical linear mixed-effects models, in which individuals are nested within census tracts. The use of 2-level models, with telomere length change, discrimination measures, and study covariates as Level-1 variables, and neighborhood social cohesion as a Level-2 variable, allows us to ensure the independence of individual observations that are clustered within neighborhoods. For each discrimination measure (categorized major experiences of discrimination, categorized everyday discrimination, and continuous everyday discrimination), we ran separate models with a 2-way interaction term between the measure of discrimination and neighborhood social cohesion. In both linear and mixed-effects regressions, the initial models adjusted for sociodemographic covariates (Model 1), and the subsequent fully adjusted models additionally included the health behaviors and health conditions specified above (Model 2).

For example, to investigate how the association of major discrimination and change in LTL varies across neighborhood social cohesion, the following model is specified:

$$\begin{aligned} \Delta LTL_{ij} = & \beta_0 + \beta_1 \text{Major discrimination}_{ij} \\ & + \beta_2 \text{Neighborhood social cohesion}_j \\ & + \beta_3 \text{Major discrimination}_{ij} \\ & \times \text{Neighborhood social cohesion}_j \\ & + \beta X_{ij} + \zeta_{1j} + \varepsilon_{ij} \end{aligned}$$

$$\zeta_{1j} \sim N(0, \psi)$$

$$\varepsilon_{ij} \sim N(0, \theta)$$

where individuals (i) are nested within neighborhoods (j), with neighborhood-level (ζ_{1j}) and person-level (ε_{ij}) error terms, which are each normally distributed with mean zero and variances ψ and θ , respectively. \mathbf{X}_{ij} is a vector of all study covariates.

Based on these models, we then estimated the link between discrimination measures and telomere length within categories of neighborhood social cohesion.

In exploratory analyses, we examined if relationships between discrimination, neighborhood social cohesion, and telomere length change varied by race/ethnicity, given the fact that discrimination is often tied to characteristics such as race/ethnicity. For these analyses, we used linear mixed-effects models stratified by race/ethnicity and separately examined the interaction between each discrimination measure and neighborhood social cohesion.

All analyses were conducted using R statistical software version 3.5.0 at the University of California Berkeley. Statistical significance was defined at $p < .05$.

Results

Descriptive Results

Table 1 presents the overall distribution of population characteristics as well as the mean and standard deviation of LTL during both study waves (Exams I and V) within categories of baseline study covariates. The mean age in our study population was 60.6 years ($SD = 9.37$). The sample consisted of 30.1% Black/African American, 43.3% Hispanic/Latinx, and 26.6% White participants. 53.4% of our respondents were women and 46.6% were men.

Mean telomere length (T/S ratio) at Exam I in this population was 0.92 ($SD = 0.20$) and was 0.71 ($SD = 0.14$) 10 years later during Exam V. At baseline, those experiencing major discrimination in one domain, those reporting no everyday discrimination, and those residing in neighborhoods with low social cohesion had the shortest telomere length. This pattern remained relatively consistent during the follow-up period (Table 1).

The majority of our sample reported not having experienced major discrimination (53.2%), and the distribution of remaining study participants was relatively evenly split among those experiencing discrimination in one domain (24.4%) and in 2 or more domains (22.4%; Table 1). While distributions for most other study covariates were similar, compared to those reporting no major discrimination, participants who reported discrimination in 2 or more domains were younger (aged 45–54 years; 37.8% vs 29.2%), were more likely to identify as Black/African American (45.8% vs 22.2%), and were more likely to be a man (56.7% vs 40.8%; Table 2). Most study participants reported experiencing moderate everyday discrimination (48.6%), whereas 29.5% reported no everyday discrimination and 21.9% reported high everyday discrimination (Table 1). Mean everyday discrimination scores were higher for those aged 45–54, Black/African American participants, those who earned between \$50 000 and \$75 000, employed individuals, and those who resided in neighborhoods with low social cohesion (Table 2).

Regression Results

In the overall linear regression models, there was no association between major discrimination (1 domain; $\beta = 0.0005$; 95% CI: $-0.02, 0.02$; ≥ 2 domains; $\beta = -0.01$; 95% CI: $-0.03, 0.02$) or everyday discrimination (mean EDS; $\beta = 0.004$; 95% CI: $-0.01, 0.02$) and LTL change, after adjusting for all study covariates (Table 3). However, we did find that neighborhood social cohesion moderated

the link between major discrimination and 10-year changes in LTL ($p(\chi^2) = .01$; Table 3). In models adjusting for sociodemographic characteristics, among those residing in low social cohesion neighborhoods, individuals reporting major discrimination in 2 or more domains, experienced a 0.03-unit faster telomere length attrition after 10 years ($\beta = -0.03$; 95% CI: $-0.06, -0.002$) than those reporting no major discrimination (Model 1, Table 3). This association persisted after additional adjustment for health behaviors and health conditions ($\beta = -0.03$; 95% CI: $-0.06, -0.003$; Model 2, Table 3; Figure 1). Although not statistically significant, our findings were in the opposite direction for those residing in neighborhoods with high social cohesion, where neighborhoods with high social cohesion appeared to be protective against telomere attrition for those experiencing major discrimination in 2 or more domains ($\beta = 0.03$; 95% CI: $-0.0005, 0.06$; Model 2, Table 3; Figure 1).

We found no association between everyday discrimination and LTL change in the overall models or within strata of neighborhood social cohesion (Table 3).

Results of our exploratory analyses models, where we stratified by race/ethnicity and tested 2-way interactions between discrimination measures and neighborhood social cohesion, did not reveal any significant differential associations between either discrimination measure, neighborhood social cohesion, and LTL change by race/ethnicity (data not shown).

Discussion

Despite early evidence documenting the negative health consequences of discrimination, the multilevel and time-dependent contexts that influence its effect on physiological wear and tear are not well understood. Utilizing longitudinal data on a multiethnic sample of older adults, this study investigated whether neighborhood social cohesion moderated the association between discrimination and 10-year telomere length attrition. Independent of a wide range of sociodemographic factors, health behaviors, and health conditions, we found that the link between major experiences of discrimination across multiple domains and 10-year changes in telomere length varied across levels of neighborhood social cohesion. Among those residing in neighborhoods with low social cohesion, experiencing discrimination in 2 or more domains resulted in faster telomere length attrition over 10 years, compared to those who reported no discrimination, and this decline in LTL was equivalent to the decline associated with a 15-year increase in age from our models. These results highlight the neighborhood social environment, more specifically, neighborhood social cohesion, as an important buffer that may mitigate the adverse and cumulative health impacts of discrimination on accelerated aging.

Rich psychosocial resources that come with residing in neighborhoods with high social cohesion are thought to reinforce healthy behaviors, the collective will to establish and maintain healthy physical environments, and strong community bonding (23,40). As such, studies have documented various physical and psychological health benefits associated with residing in socially cohesive neighborhoods (22,26,41). More specifically, in the context of experiencing discrimination, residing in neighborhoods with high social cohesion is thought to reinforce residents' self-esteem and sense of belongingness, thus providing healthy coping and stress-buffering environments within which individuals are able to reappraise such unfair encounters as less threatening, recognize them as a collective experience, and distract themselves from the distresses that arise from them (42). Rather than engaging in maladaptive coping strategies

Table 1. Distribution of Baseline Population Characteristics and Leukocyte Telomere Length, the Multi-Ethnic Study of Atherosclerosis, 2000–2011

	Overall (N = 1064)	LTL Exam I	LTL Exam V
	N (%)	Mean (SD)	Mean (SD)
Major Discrimination Scale (mean = 60.59, SD = 9.37)			
None	566 (53.20)	0.92 (0.21)	0.71 (0.14)
One domain	260 (24.40)	0.90 (0.20)	0.70 (0.14)
≥2 domains	238 (22.40)	0.92 (0.21)	0.71 (0.16)
Everyday Discrimination Scale (mean = 1.61, SD = 0.71)			
None	314 (29.50)	0.91 (0.21)	0.69 (0.15)
Moderate	517 (48.60)	0.92 (0.20)	0.71 (0.14)
High	233 (21.90)	0.92 (0.21)	0.72 (0.14)
Neighborhood social cohesion			
Low	525 (49.30)	0.90 (0.21)	0.71 (0.15)
High	539 (50.70)	0.94 (0.20)	0.70 (0.14)
Age (years)			
45–54	333 (31.30)	0.98 (0.21)	0.76 (0.15)
55–64	337 (31.70)	0.94 (0.19)	0.71 (0.13)
65 and older	394 (37.00)	0.85 (0.19)	0.66 (0.13)
Race/ethnicity			
White	283 (26.60)	0.94 (0.20)	0.71 (0.15)
Black/African American	320 (30.10)	0.90 (0.20)	0.72 (0.15)
Hispanic/Latinx	461 (43.30)	0.92 (0.21)	0.70 (0.14)
Gender			
Women	568 (53.40)	0.94 (0.20)	0.72 (0.15)
Men	496 (46.60)	0.89 (0.20)	0.69 (0.14)
Education			
High school or less	425 (39.90)	0.92 (0.21)	0.70 (0.15)
Some college/Technical school	325 (30.50)	0.93 (0.21)	0.71 (0.14)
University graduate	314 (29.50)	0.91 (0.19)	0.72 (0.14)
Income			
Less than \$20 000	253 (23.80)	0.91 (0.21)	0.69 (0.13)
\$20 000–49 999	453 (42.60)	0.91 (0.21)	0.71 (0.14)
\$50 000–74 999	166 (15.60)	0.94 (0.20)	0.72 (0.15)
More than \$75 000	192 (18.00)	0.93 (0.19)	0.73 (0.16)
Employment status			
Unemployed	442 (41.50)	0.90 (0.20)	0.68 (0.14)
Employed	622 (58.50)	0.93 (0.21)	0.73 (0.14)
Marital status			
Not married	461 (43.30)	0.89 (0.20)	0.70 (0.14)
Married	603 (56.70)	0.94 (0.20)	0.72 (0.15)
Diabetes status			
No	930 (87.40)	0.92 (0.20)	0.71 (0.15)
Yes	134 (12.60)	0.91 (0.21)	0.68 (0.13)
Hypertension			
No	600 (56.40)	0.93 (0.21)	0.72 (0.15)
Yes	464 (43.60)	0.90 (0.20)	0.69 (0.14)
Cancer			
No	995 (93.50)	0.92 (0.21)	0.71 (0.14)
Yes	69 (6.50)	0.86 (0.17)	0.69 (0.14)
Chronic Burden Score (mean = 1.31, SD = 1.18)			
Low (0–1)	666 (62.60)	0.92 (0.21)	0.71 (0.14)
Moderate (2)	230 (21.60)	0.91 (0.20)	0.70 (0.14)
High (3 or more)	168 (15.80)	0.92 (0.21)	0.72 (0.16)
BMI (mean = 29.08, SD = 5.47)			
≤24.9	241 (22.70)	0.92 (0.21)	0.72 (0.15)
25.0–29.9	431 (40.50)	0.92 (0.20)	0.71 (0.15)
≥30	392 (36.80)	0.92 (0.21)	0.70 (0.14)
CES-D (mean = 7.89, SD = 8.00)			
No	920 (86.50)	0.92 (0.20)	0.71 (0.15)
Yes	144 (13.50)	0.93 (0.21)	0.70 (0.14)
Pack-years of smoking (mean = 8.70, SD = 15.94)			
Nonsmoker (0)	565 (53.10)	0.93 (0.21)	0.72 (0.14)
1–10	220 (20.70)	0.91 (0.18)	0.71 (0.14)

Table 1. Continued

	Overall (N = 1064)	LTL Exam I	LTL Exam V
	N (%)	Mean (SD)	Mean (SD)
>10–20	107 (10.10)	0.93 (0.21)	0.71 (0.16)
>20	172 (16.20)	0.90 (0.20)	0.68 (0.14)
Moderate to vigorous physical activity (mean = 6022.11, SD = 6353.43)			
Low	355 (33.40)	0.92 (0.20)	0.70 (0.14)
Moderate	355 (33.40)	0.91 (0.21)	0.70 (0.15)
High	354 (33.30)	0.92 (0.21)	0.72 (0.14)

Note: LTL = leukocyte telomere length; SD = standard deviation; BMI = body mass index; CES-D = Center for Epidemiologic Studies—Depression.

such as self-blame, rumination, and attributing experiences of discrimination as warranted, strong social cohesion facilitates a venue for processing and contextualizing discrimination, and for seeking guidance on how to contend with these experiences, attenuating its psychological and physiological impacts (43,44). Although not statistically significant, our findings for those residing neighborhoods with high social cohesion highlight this potentially protective nature of social cohesion against the adverse health impacts of discrimination. However, more research is needed to more closely assess highly socially cohesive neighborhoods in relation to discrimination and telomere length attrition.

On the other hand, neighborhoods characterized by low social cohesion may present additional challenges to their residents and exacerbate the effect of discrimination on health. As a result of historic inequitable policies and practices in the United States that have orchestrated racial and economic residential segregation and subsequent sustained disinvestment, individuals who live in such neighborhoods bear the clustering of spatial disenfranchisement. In these settings, the lack of protective social environments, particularly low social cohesion, may pose as a source of chronic stress through its links with increased social disorder, weakened social fabric, and the lack of health-promoting physical environments (eg, parks) (24,36,45). Such concentration of stressful neighborhood environments can then compound together with discrimination to heighten physiological wear and tear (38,46–48). These types of environments may also constrain individuals' opportunities to process and cope with discrimination. The limited availability of social support networks, with whom to garner a sense of social capital and connectedness in less socially cohesive neighborhoods, leaves individuals with depleted psychosocial resources and therefore may lead them to internalize unfair treatment and accept it as a deserved treatment, further enhancing its ill-health effects. In a prior cross-sectional study, although we did not find associations between discrimination and baseline telomere length in the full sample, we showed that among participants with low individual-level social support, reporting moderate and high everyday discrimination versus no discrimination was associated with shorter telomeres. In these analyses, there was no association between discrimination and telomere length for those with moderate and high social support (37). The current study extends this work by including longitudinal measurements of telomere length to assess the influence of discrimination on telomere length attrition over time and by additionally examining neighborhood social cohesion, which is integral in shaping stress-coping mechanisms. Our results are also consistent with Saleem et al. (49), who found that neighborhood social cohesion attenuated the impact of racial discrimination on depressive symptoms among African American adolescents.

The findings of this study provided evidence that neighborhood social cohesion moderated the impact of major discrimination, rather than everyday discrimination, on telomere length change. While contrary to prior studies that have found significant associations between everyday hassles and poor health outcomes (7), our results are consistent with studies that have documented inverse links between lifetime (major) discrimination and telomere length, as well as with previous findings in MESA showing negative relationships between major discrimination and incident cardiovascular disease and diabetes (2,5,19,21,39). More specifically, our findings are aligned with a recent study by Chae et al. (39) that used latent change score analyses to document that African Americans reporting racial discrimination across multiple major domains experienced faster telomere length attrition. There are also several reasons that may explain why we saw stronger associations for major discrimination instead of everyday unfair treatment. First, major experiences of discrimination capture the presence of lifetime adversity across multiple institutions that lead to sustained socioeconomic and psychosocial deprivation. Experiences of discrimination that are more blatant, such as unfairly being denied housing, losing a job or a promotion, and being harassed by law enforcement, have a long-lasting impact on quality of life and limit the availability of resources that aid in establishing adaptive coping behaviors and consequently healthy living and aging (1). By assessing the presence of unjust treatment across a broader set of institutions, the major discrimination scale captures deep-seated inequalities that burden marginalized populations. Such inequitable and unfair practices can also be conceptualized as more upstream indicators of day-to-day hassles, as they reinforce social norms that manifest in experiences of interpersonal discrimination (50). Alternatively, because we were only able to assess discrimination at baseline, the major experiences of discrimination may have been better suited to assess long-term unfair treatment, as opposed to responses to the everyday discrimination scale, which may have changed throughout the 10 years. Finally, because our sample primarily includes individuals who were alive during the Jim Crow era, when *de jure* discrimination was widely practiced, the major discrimination scale may be more salient in capturing the health effects of such extreme experiences of discrimination. As such, more research is warranted to further understand the complex nature of major versus everyday experiences of discrimination and their health effects.

Limitations

The results of this study should be interpreted within the context of its limitations. First, our study sample may not be representative of all older adults in the United States, as our participants were selected

Table 2. Distribution of Baseline Population Characteristics and Across Categories of Major Experiences of Discrimination, the Multi-Ethnic Study of Atherosclerosis, 2000–2011

	MDS (%)			Mean EDS N = 1064
	None	1 Domain	≥2 Domains	
	N = 566	N = 260	N = 238	
Age (years)				
45–54	29.20	30.00	37.80	1.83
55–64	30.20	35.00	31.50	1.64
65 and older	40.60	35.00	30.70	1.39
Race/ethnicity				
White	29.00	26.90	20.60	1.56
Black/African American	22.30	32.70	45.80	1.91
Hispanic/Latinx	48.80	40.40	33.60	1.44
Gender				
Women	59.20	50.00	43.30	1.6
Men	40.80	50.00	56.70	1.61
Education				
High school or less	47.20	37.30	25.60	1.48
Some college/Technical school	26.70	32.70	37.40	1.72
University graduate	26.10	30.00	37.00	1.67
Income				
Less than \$20 000	25.80	21.90	21.00	1.43
\$20 000–49 999	42.40	46.90	38.20	1.63
\$50 000–74 999	14.30	14.20	20.20	1.74
More than \$75 000	17.50	16.90	20.60	1.69
Employment status				
Unemployed	45.40	41.20	32.80	1.45
Employed	54.60	58.80	67.20	1.72
Marital status				
Not married	40.80	46.90	45.40	1.64
Married	59.20	53.10	54.60	1.58
Diabetes status				
No	89.20	83.50	87.40	1.62
Yes	10.80	16.50	12.60	1.55
Hypertension				
No	54.80	59.20	57.10	1.64
Yes	45.20	40.80	42.90	1.57
Cancer				
No	93.50	92.70	94.50	1.62
Yes	6.50	7.30	5.50	1.48
Chronic Burden Score				
Low (0–1)	73.00	55.40	45.80	1.50
Moderate (2)	17.70	26.20	26.10	1.66
High (3 or more)	9.40	18.50	28.20	1.95
BMI				
≤24.9	25.10	22.30	17.20	1.6
25.0–29.9	39.90	40.40	42.00	1.59
≥30	35.00	37.30	40.80	1.64
CES-D				
No	87.50	91.20	79.00	1.56
Yes	12.50	8.80	21.00	1.96
Pack-years of smoking				
Nonsmoker (0)	58.00	46.20	49.20	1.54
1–10	18.90	27.30	17.60	1.63
>10–20	10.20	10.40	9.20	1.66
>20	12.90	16.20	23.90	1.77
Moderate to vigorous physical activity				
Low	35.50	32.30	29.40	1.54
Moderate	32.90	35.00	32.80	1.56
High	31.60	32.70	37.80	1.73
Neighborhood social cohesion				
Low	50.20	48.80	47.90	1.67
High	49.80	51.20	52.10	1.55

Note: MDS = Major Discrimination Scale; EDS = Everyday Discrimination Scale; BMI = body mass index; CES-D = Center for Epidemiologic Studies—Depression.

Table 3. Associations Between Baseline Experiences of Discrimination and 10-Year Change in Telomere Length Across Categories of Baseline Neighborhood Social Cohesion, the Multi-Ethnic Study of Atherosclerosis, 2000–2011

	Model 1	Model 2
	Beta (95% CI)	Beta (95% CI)
<i>Overall (N = 1064)^a</i>		
Major Discrimination Scale		
None	Ref	Ref
One domain	0.002 (–0.02, 0.02)	0.0005 (–0.02, 0.02)
≥2 domains	–0.005 (–0.02, 0.02)	–0.01 (–0.03, 0.02)
Everyday Discrimination Scale		
None	Ref	Ref
Low and moderate	–0.003 (–0.02, 0.02)	–0.001 (–0.02, 0.02)
High	–0.0004 (–0.02, 0.02)	–0.003 (–0.03, 0.02)
Mean Everyday Discrimination	0.005 (–0.01, 0.02)	0.004 (–0.01, 0.02)
<i>Low social cohesion (N = 525)^b</i>		
Major Discrimination Scale*		
None	Ref	Ref
One domain	–0.004 (–0.03, 0.02)	–0.01 (–0.03, 0.02)
≥2 domains	–0.03 (–0.06, –0.002)	–0.03 (–0.06, –0.003)
Everyday Discrimination Scale		
None	Ref	Ref
Low and Moderate	–0.01 (–0.04, 0.02)	–0.01 (–0.04, 0.02)
High	–0.01 (–0.04, 0.02)	–0.01 (–0.04, 0.03)
Mean Everyday Discrimination	0.001 (–0.01, 0.02)	0.002 (–0.01, 0.02)
<i>High social cohesion (N = 539)^b</i>		
Major Discrimination Scale*		
None	Ref	Ref
One domain	0.01 (–0.02, 0.03)	0.004 (–0.02, 0.03)
2 or more domains	0.03 (–0.002, 0.06)	0.03 (–0.0005, 0.06)
Everyday Discrimination Scale		
None	Ref	Ref
Low and moderate	–0.001 (–0.03, 0.03)	0.003 (–0.02, 0.03)
High	–0.01 (–0.04, 0.03)	–0.004 (–0.04, 0.03)
Mean Everyday Discrimination	0.003 (–0.02, 0.02)	0.004 (–0.01, 0.02)

Notes: CI = confidence interval. Model 1: adjusting for baseline sociodemographic covariates (age, race, gender, education, income, employment status, and marital status). Model 2: Model 1 + baseline health behaviors and conditions (exercise, smoking, body mass index, chronic burden, depression, diabetes, hypertension, and cancer).

^aLinear regression models.

^bLinear mixed-effects regression models.

* $p(\chi^2) < .05$; boldface: $p < .05$.

from 2 study sites, and are more educated, healthier, and wealthier than the general US population in the same age range. Hence, more nationally representative studies should be conducted to corroborate our findings. Second, utilizing census tracts as a way to define neighborhoods may have limited our ability to capture participants' geographic definition of a neighborhood that may be more relevant to their health. Our results are also subject to possible biases such as neighborhood self-selection. In order to address this possibility, consistent with prior work, we adjusted for a range of sociodemographic characteristics that may influence self-selection. However, we cannot rule out residual confounding due to unmeasured factors. Our study was also not powered to detect the potential differential manifestation of discrimination and neighborhood environments across racial/ethnic groups, which is needed for a more nuanced comprehension of their joint health impacts. Furthermore, because discrimination measures were only available at baseline, we were not able to assess how changes in reports of discrimination may influence changes in telomere length. We also only examined baseline measures of social cohesion in this study. Thus, future work should assess how longitudinal

changes in discrimination and neighborhood social cohesion may be related to telomere length change. Additionally, in this study, we did not examine the attribution of experiences of discrimination or more explicit forms of unfair treatment such as racism. Hence, studies should more closely examine how experiences of discrimination that are attributed to social identities (eg, race/ethnicity) influence telomere length attrition. Lastly, the measure of telomere length available in this sample is specific to leukocytes and may not be generalizable to other tissue samples. Further investigations are needed to corroborate our results using other markers of aging that may further elucidate the biological embodiment of discrimination.

Strengths

Despite these limitations, our study has several strengths. By assessing telomere length at 2 time points with enough time to observe meaningful changes, this study provides evidence on how discrimination may be related to the rate of cellular aging over time. This study also additionally incorporates information on neighborhood social cohesion, a strong indicator of social connectedness at the neighborhood level. To the best of our

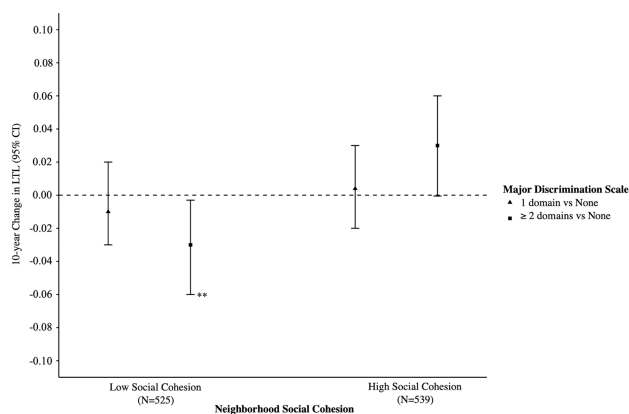


Figure 1. Associations between baseline reports of Major Experiences of Discrimination Scale (MDS) and 10-year change in telomere length across categories of baseline neighborhood social cohesion, the Multi-Ethnic Study of Atherosclerosis (MESA), 2000–2011. Note: ** $p < .05$. Estimates are from linear mixed-effects regression models adjusted for baseline sociodemographic characteristics, health behaviors, and health conditions.

knowledge, this is the first study to examine the extent to which neighborhood contexts may moderate the link between discrimination and longitudinal changes in telomere length. Studies such as this one that jointly investigate individual-level stressors and neighborhood environments more accurately represent the complex multilevel realities of people, as beings nested within both time and space and interacting with structural and environmental factors in their day-to-day lives. They also further explicate the multifaceted psychosocial processes through which discrimination, across multiple institutions, affects health. By accounting for a range of covariates, including sociodemographic characteristics, health behaviors, and health conditions, the results of this study are able to better explain the differential consequences of discrimination on telomere length across neighborhood contexts. Additionally, the rigor of measures utilized in this study increases the validity of our findings. Both our discrimination and neighborhood social cohesion measures have been previously validated and have demonstrated good test–retest reliability (33,34). By utilizing CEB estimates, our neighborhood social cohesion measure additionally accounts for data sparsity and differential self-report. Furthermore, MESA is a well-characterized cohort that is racially and socioeconomically diverse, making the results of this study notable.

Conclusions

By leveraging longitudinal and neighborhood-level data in a well-ascertained and racially/ethnically diverse cohort, our study is the first of its kind to show that residing in neighborhoods with low social cohesion may make individuals vulnerable to the adverse effects of discrimination on telomere length. In addition to highlighting the need for transformational change across institutions that harbor discriminatory practices, this study provides evidence for intervening at the neighborhood level with the intent to promote social connectedness and collective agency as a way to mitigate the negative health consequences of discrimination.

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Conflict of Interest

None declared.

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