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Shortened telomere length is associated with unfair treatment attributed to race in African Americans living in Tallahassee, Florida

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Abstract

Objectives: Experiences of interpersonal discrimination are pervasive stressors in the lives of African Americans. Increased discrimination stress may cause premature aging. Telomere length (TL) is a plastic genetic trait that is an emerging indicator of cellular health and aging. Short TL is a risk factor for the earlier onset of disease. TL shortens with age, a process that may be accelerated by psychosocial stress. Our study explores the relationship between TL and experiences of discrimination in the form of self-reported unfair treatment (UT).

Methods: Using a qPCR-based method, we measured TL in DNA from saliva samples provided by 135 African American adults from Tallahassee, FL. We developed discrimination measures using a modified survey that explores nine social domains of self-reported unfair treatment experienced both directly and indirectly. We used multiple regression to examine associations between UT and TL.

Results: We found that racial discrimination in the form of self-reported unfair treatment attributed to race (UT-Race-Self) is inversely associated with TL.

Conclusions: The significant association between increased UT-Race-Self and shorter telomeres supports the hypothesis that psychosocial stress stemming from racial discrimination may affect TL. The potential impact of discrimination on TL may contribute to premature biological aging and racial health inequalities seen in African Americans.

1 | INTRODUCTION

Racial health inequalities are well documented in the United States (National Center for Health Statistics, 2007), and there is substantial evidence that African Americans have both a higher chronic disease burden and lower life expectancies than other racial groups (ie, Hicken, Lee, Morenoff, House, & Williams, 2014; Williams & Mohammed, 2009; Wyatt et al., 2003). Geronimus' (1992) and Geronimus, Hicken, Keene, & Bound, 2006 "weathering" hypothesis provides a possible explanation for some of the observed differences in health outcomes. Geronimus posits that health inequalities between African Americans and European Americans are related to premature biological aging, or weathering, which stems from the cumulative impact of exposure to a range of stressors.

There is evidence to suggest that changes in telomere length (TL) may be an important pathway by which psychosocial stressors, including discrimination stress, could possibly cause premature biological aging and influence health outcomes. Telomeres comprise the ends of linear chromosomes and consist of noncoding repeated DNA

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(Blackburn & Gall, 1978). Since DNA replication machinery cannot bind to the ends of chromosomes, each round of cell division results in the loss of approximately 50-200 base-pairs of telomeric DNA (Wright, Piatyszek, Rainey, Byrd, & Shay, 1996). Consequently, telomeres function to protect chromosomes from the loss of coding DNA (Blackburn, 2000; Harley, Futcher, & Greider, 1990). Once telomeres reach a critical minimum length, cellular replication ceases, and cells become senescent (Armanios & Blackburn, 2012; Cong, Wright, & Shay, 2002; Palm & de Lange, 2008). Accordingly, TL is considered to be a marker of biological age (Ishii et al., 2006; Kimura et al., 2008; Olovnikov, 1973), as TL consistently inversely correlates with chronological age (reviewed by von Zglinicki & Martin-Ruiz, 2005), and short TL associates with a number of age-related diseases, immunosuppression, and early death (Al Khaldi, Mojiminiyi, AlMulla, & Abdella, 2015; Armanios et al., 2009; Carulli & Anzivino, 2014; Cawthon, Smith, O'Brien, Sivatchenko, & Kerber, 2003; D'Mello et al., 2014; Epel, Merkin, & Cawthon, 2008; Ma et al., 2011; Serrano & Andrés, 2004). These associations typically exist regardless of sex or racially defined group, but they tend to vary in strength between groups (eg, Carty et al., 2015; Chen et al., 2009).

It is important to point out that the association data linking TL and disease are correlational, not causal (Bischoff et al., 2006; Harris et al., 2006; Martin-Ruiz, Gussekloo, van Heemst, von Zglinicki, & Westendorp, 2005). However, based on known disease etiologies, causal relationships are plausible. For example, when cells have critically short telomeres, they are signaled to cease replication and become senescent. This signal is thought to be mediated through the uncapping of the structural protein shelterin from short telomeres (Cookson & Laughton, 2009; Lendvay, Morris, Sah, Balasubramanian, & Lundblad, 1996). Senescent leukocytes are subsequently a cause of atherosclerotic plaque buildup, which is a major contributor to heart disease (Childs et al., 2016), a condition that shows severe disparities between African and European American populations (Cooper et al., 2000).

In addition to age-dependent attrition, telomere shortening may be accelerated by exposure to psychosocial stress (reviewed by Quinlan et al., 2014). Chronic stress is thought to cause increased systemic and cumulative oxidative stress, weakened immunity, chronic inflammation, increased cortisol levels, and reduced telomerase activity—pathways that are also implicated in telomere shortening (Choi, Fauce, & Effros, 2008; Irie, Asami, Ikeda, & Kasai, 2003; Irie, Asami, Nagata, Miyata, & Kasai, 2001; Kawanishi & Oikawa, 2004; Oikawa & Kawanishi, 1999). The link between stress and TL is further supported by two recent meta-analyses that demonstrated an overall significant association between perceived stress and TL (Mathur et al., 2016; Pepper, Bateson, & Nettle, 2018). These findings indicate that by contributing to TL loss, psychosocial stress may have a durable effect on cellular health and aging.

The link between psychosocial stress and TL has been examined in different racial populations (eg, Epel et al., 2004; Needham et al., 2013). African Americans have longer telomeres on average than European Americans both at birth and during early adulthood (Hunt et al., 2008; Okuda et al., 2010), but they experience telomere shortening from infancy to middle adulthood at significantly faster rates (Chen et al., 2011; Rewak et al., 2014). This increased rate of telomere shortening may eventually overcome the longer telomeres seen in early life. One study found that African Americans had shorter telomeres on average than their European American counterparts by 61 years of age (Roux et al., 2009). These complex correlational results suggest a possible interaction between psychosocial and biological processes. Indeed, increased discrimination stress and the consistent strain of anticipating prejudice in interethnic interactions are associated with higher levels of anxiety and physiological reactivity across minority populations (Paradies, 2006; Sawyer, Major, Casad, Townsend, & Mendes, 2012). These factors are believed to increase overall oxidative stress and inflammation levels, both of which are presumed to contribute to premature biological aging and are suspected causal factors in the pathway between stress exposure and telomere attrition (Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010; McEwen & Seeman, 1999; Szanton et al., 2012).

Researchers have previously investigated the impact of discrimination stress on TL in African Americans. For example, the Health and Retirement Study (HRS) recently found that everyday discrimination (per the Everyday Discrimination Scale-Short Form; EDS-SF; Sternthal & Williams, 2011) was associated with shorter telomeres in African American adults (Lee, Kim, & Neblett, 2017; Liu & Kawachi, 2017). The same association was not observed among European Americans enrolled in the HRS (Liu & Kawachi, 2017). Additional findings suggest that discrimination stress may act in concert with other factors to shorten telomeres and subsequently accelerate the process of biological aging. For example, Chae et al. (2014, 2016) found that African American men who experienced more frequent racial discrimination (indexed using the situation version of the Experiences of Discrimination (EOD) questionnaire (Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005) had significantly shorter telomeres, but only when discrimination was included in a statistical interaction with either depression or an internalized anti-black racial bias.

In addition to personal experiences of unfair treatment, indirect exposure to prejudice and racial discrimination

experienced by family members, close friends, neighbors, and even strangers, may also influence health (Harrell, 2000). The adverse effects of perceived threats or maltreatment of others in a shared environment has mostly been studied in children (eg, Kelly, Becares, & Nazroo, 2013; Priest, Paradies, Stevens, & Bailie, 2012). Our research team developed a measure of vicarious discrimination while working with the same Tallahassee cohort as in the current study; we found that experiences of vicarious racism were associated with blood pressure through interactions with genetic markers involved in both cardiovascular disease and mental health (Boulter et al., 2015; Quinlan et al., 2016). As such, vicarious racism is an important stressor to include in investigations of racial inequalities.

Since experiences of interpersonal discrimination often cause feelings of fear, anger, and denial that can persist after the incident, they may have long-term effects on both physiological and mental health (Budhwani, Hearld, & Chavez-Yenter, 2015; English, Lambert, & Ialongo, 2014; Krieger, 2001; Krieger & Sidney, 1996; Kwate & Goodman, 2015). As such, another factor of interest that may combine with discrimination to influence TL and other health outcomes is emotional reactivity (Harrell, 2000). Interestingly, the face-valid emotional component of Cohen's Perceived Stress Scale ("feeling nervous or stressed") has previously been linked with short TL (Cohen, 1994; Epel, Daubenmier, Moskowitz, Folkman, & Blackburn, 2009). Thus, variation in the persistence of these emotions could affect TL directly or possibly influence the association between discrimination and TL and is worth investigating further.

Here we tested for associations between TL and multiple measures of unfair treatment (UT), including those explicitly attributed to race, experienced by study participants or individuals close to the study participant (measures adapted from Williams & Jackson, 2005; Williams, Yu, Jackson, & Anderson, 1997) in 135 African Americans sampled from Tallahassee, FL. We also examined if the effects of UT persisted after the experiences themselves, specifically, if UT combined with emotional reactivity to predict TL. The setting for this study was selected because there is a large, vibrant African American community in Tallahassee with substantial variation in many of our covariates, such as measures of socioeconomic status (SES), including education level.

2 | MATERIALS AND METHODS

2.1 | Study participants

Details of recruitment and sampling strategies are outlined by Boulter et al. (2015). Briefly, we drew a multistage probability sample in which Census block groups were first clustered by indicators of racial composition and material deprivation. We derived material deprivation from the US Census Bureau's American Community Survey, which assesses the percent of vacant housing units, median household income, percent of female-headed households, and percent of the population in poverty. We then selected block groups within each cluster at random, followed by a random selection of households, and finally of one eligible adult within each household. Informed consent was obtained from all participants prior to sample collection. All personal information was de-identified prior to inclusion in analysis. This study was approved by the University of Florida IRB-01 (#364-2008) and IRB-02 (#2007-U-469). The initial number of participants recruited to this was 185. Twenty individuals did not provide saliva, while saliva extracts from another 30 did not yield DNA of sufficient quality and/or quantity for TL measurement. The final number of participants included in this study was 135.

2.2 | Sociocultural data

Initial ethnographic interviews (N = 34) revealed that participants commonly cited racial discrimination experienced by both themselves and by close friends and family members as sources of stress (see Boulter et al., 2015). Therefore, for the subsequent survey sample, we adapted the widely used measure of everyday discrimination developed by Williams et al. (1997) (EDS). Specifically, the original measure asks only about unfair treatment (UT) to self. The measure was expanded to ask, "Have you, or someone close to you, been unfairly treated..." in each of nine domains (Table 1). If respondents answered "Yes," we followed-up by asking whether (a) the participant and (b) whether people in seven other social roles (spouse or partner, parents, child, sibling, grandchild, other relative, or close friend) had experienced unfair treatment in a given domain.

Based on participant responses, we developed two measures of UT to test for associations with TL. The first measure, UT-Self, counted the number of UT domains that participants reported experiencing directly. The second measure, UT-Other, counted domains of UT that were experienced by someone close to the participant in the absence of a report of UT-Self (Table 1). Unless otherwise stated, all UT measures were unweighted counts of affirmative answers per social domain (scores range from 0 to 9).

Next, we created measures of UT-Self and UT-Other that only included instances of unfair treatment attributed to race (UT-Race-Self and UT-Race-Other). Specifically, if participants answered "Yes" to "Have you or someone close to you ever been unfairly treated...?", they

TABLE 1 Unfair treatment (UT) domains used to develop UT measures

Unfair Treatment Domains
Have you or anyone close to you been treated unfairly concerning work?
Have you or anyone close to you been treated unfairly by the police?
Have you or anyone close to you been treated unfairly by the courts?
Have you or anyone close to you been treated unfairly at school?
Have you or someone close to you been treated unfairly in getting a house?
Have you or someone close to you been treated unfairly in getting money?
Have you or someone close to you been treated unfairly in getting health care?
Have you or someone close to you received worse service than other people?
Have you or someone close to you ever been unfairly rejected or not accepted by your in-laws?
Who was treated unfairly?
Yourself, your spouse, your parents, your child, your sibling, your grandchild, other relative, close friend

were asked the follow-up question "What do you think was the main reason for this experience?" and could choose as many as they liked of the following 12 possibilities: Your race, gender, age, weight, shade of skin, sexual orientation, income or social class, education level, hair style, way of speaking, way of dress, and physical disability. We then counted the number of domains where participants exclusively attributed UT to race, as opposed to the other possibilities because it more directly queried the participants' experiences of race-based discrimination.

We derived all UT measures from a validated measure of unfair treatment (Williams et al., 1997; Williams & Jackson, 2005). For each individual UT measure, we determined internal consistency by calculating Cronbach's alpha using the "psych" package in R (Cronbach, 1951; Revelle, 2018): UT-Self—$\alpha = 0.76$; UT-Other—$\alpha = 0.67$ (we tested if removing any of the nine items would improve the α, but it did not achieve $\alpha > 0.70$); UT-Race-Self—$\alpha = 0.81$; UT-Race-Other—$\alpha = 0.74$ (Williams et al.'s original UT measure - $\alpha = 0.88$).

Harrell (2000) posits that individual differences in emotional reactions to stress may influence how discrimination shapes health outcomes. One potential source of this variation is emotional persistence. To allow us to assess if the effects of UT persisted after the experiences themselves, participants were asked: "During the past year, have you felt emotionally upset, for example angry, sad, or frustrated, as a result of how you were treated based on your race?" This question, based on one developed by the CDC Measures of Racism Working Group (Jones et al., 2009), was coded as the binary variable "Race-Upset."

In addition to discrimination, chronic stress stemming from differences in SES is often investigated as a possible explanation for disparities in rates of telomere shortening between African Americans and European Americans (Gebreab et al., 2016; Geronimus et al., 2015; Needham et al., 2013; Theall, Brett, Shirtcliff, Dunn, & Drury, 2013). Financial strain, education years completed, and household income are some of the most commonly tested SES measures, and all have been associated with either shorter TL or increased telomere attrition in African American cohort studies (Needham et al., 2013; Schrock et al., 2018; Shiels et al., 2011). Thus, we decided to use these three measures of SES as covariates. Financial strain measured how much trouble the participants had in paying their bills (scored 1-4, with 1 being not difficult at all and 4 being very difficult). The yearly household income measures were binned by $5000 increments, which extended from $0 to >$90 000 (scores range from 1 to 19).

2.3 | Biological data

Saliva samples were collected and DNA was extracted using Oragene saliva kits (DNA Genotek, Ottawa, Canada). DNA quality and quantity were determined using a NanoDrop 1000 Spectrophotometer (Thermo Fisher Scientific, Waltham, MA). Minimum 260/280 ratios of 1.8 and 260/230 ratios of 1.5 were required for inclusion in the analysis.

TL measured from DNA extracted from Oragene saliva kits consistently associates with blood TL in the literature; published Pearson's correlation coefficients range from $r = 0.48$-0.72 (Goldman et al., 2017; Mitchell et al., 2014; Stout et al., 2017). Variation in correlation values is likely reflective of cellular heterogeneity in saliva samples, as they consist of roughly 80% leukocytes and 20% epithelial cells on average (Theall et al., 2013; Thiede, Prange-Krex, Freiberg-Richter, Bornhauser, & Ehninger, 2000). In spite of the reported cellular heterogeneity, salivary TL reliably satisfies measures of external and internal validity that are regularly used when measuring TL. For example, significant negative associations with age in salivary TL estimates are almost universally reported in the literature, while intra-sample QC is regularly within acceptable bounds (Chen et al., 2015; Goldman et al., 2017; Nelson, Varcin, Coman, Devivo, & Tager-Flusberg, 2015).

Data on age and sex were also collected. Measurements on height (using a stadiometer) and weight (using a digital scale) were collected by the interviewers trained in anthropometric methods and used to calculate body mass index (BMI) (kg/m^2). Participants were also asked if they had ever regularly smoked cigarettes (ie, smoking status).

2.4 | Measuring relative TL

Relative TL was measured on a BioRad CFX 96 real time PCR detection system (BioRad, Hercules, CA) using a modified version of Cawthon's (2009) monochrome multiplex qPCR (MMQPCR) assay. Telomere (T) primer pair "telg" and "telc" (Cawthon, 2009) and single copy gene (S) primers "albdgcr2" and "albugcr2" (Tackney, Cawthon, Coxworth, & Hawkes, 2014), purchased from Eurofins MWF Operon (Huntsville, AL), were used along with Maxima SYBR Green/ROX qPCR Master Mix (2X) (Invitrogen, Carlsbad, CA). We used the following thermocycling profile: stage 1:15 min at 95°C; stage 2:2 cycles of 2 s at 49°C; stage 3:34 cycles of 2 s at 97°C, 30 s at 59°C, 15 s at 74°C with signal acquisition, 30 s at 84°C, 15 s at 85°C with signal acquisition (Cawthon, 2009; Tackney et al., 2014). Primer sequences, along with qPCR reagent concentrations and volumes, are listed in Table S1.

High quality human genomic DNA purchased from Aldevron (Madison, WI, product number 5070-25) was used to construct a five-point standard reference curve (Cawthon, 2009), using a 3-fold serial dilution with initial concentration of standard 1 = 5 ng/μL. Sample DNA was diluted to 1.25 ng/μL. All samples and standards were included in triplicate on each plate. Three negative controls and two positive controls (concentration 1.25 ng/μL) were included on every plate. Total reaction volume was 11 μL. Minimum plate quality control requirements included a standard curve correlation of $R^2 > 0.98$ and qPCR efficiency between 85% and 115%.

Relative TL was measured by comparing the number of cycles required to amplify the telomeric DNA to that of the single copy DNA, that is, the T/S ratio. T/S ratio was calculated using a variation of Pfaffl's (2001) equation for relative qPCR quantification (Ehrlenbach et al., 2009). This equation had the tightest correlation with age. T/S ratio was averaged across three replicates for each sample. The coefficient of variance (CV) was calculated to determine precision of TL measurements. Samples were re-assayed if the CV between replicates was greater than 0.05. Significant outliers were identified using Dixon's-Q test and were excluded from analysis.

Raw T/S ratio data were visibly skewed right. Shapiro-Wilk test was strongly significant ($W = 0.12$) and Box Cox test results recommended log transformation of T/S ($\lambda = -0.06$). Transformation normalized the data ($W = 0.98$) and improved all regression models.

2.5 | Statistical analysis

Summary and inferential statistics were calculated using R 3.6.1 (R Development Core Team, 2019). Before running our OLS regressions, we incorporated our outcome, predictor, and control variables into a bivariate correlation analysis. Multiple regression models were specified to investigate associations between TL and UT. All models included the following covariates: age, sex, BMI, and smoking status. Chronological age is the most consistent predictor of TL, which warranted its inclusion in our regressions. Sex was included in our models as a covariate as women tend to have longer telomeres than age-matched men (Gardner et al., 2013). Higher BMI frequently predicts shorter TL (Muezzinler, Zaineddin, & Brenner, 2014), while smokers are more likely to have shorter TL

TABLE 2 Sample characteristics

Sociodemographic (N = 135)	Mean or n	SD or %
Age (years), mean (SD)	40.75	(11.89)
Sex, *n* (%)		
Male	47	(34.81)
Female	88	(65.19)
Health phenotype		
BMI (kg/m^2), mean (SD)	32.32	(9.52)
Smoking, n (%)		
Yes	57	(42.22)
No	78	(57.78)
Unfair treatment (UT)		
UT-Self, mean (SD)	1.56	(1.97)
UT-Other, mean (SD)	1.53	(1.70)
UT-Race-Self, mean (SD)	1.20	(1.91)
UT-Race-Other, mean (SD)	1.16	(1.67)
Race-Upset (yes), *n* (%)		
Yes	66	(48.89)
No	61	(45.19)
NA	8	(5.92)
Socioeconomic status		
Financial strain[a], mean (SD)	2.38	(1.06)
Education (years), mean (SD)	13.17	(2.49)
Household income[b], mean (SD)	5.73	(4.37)

[a]Financial strain (difficulty paying bills) is scored 1-4, with 1 being not difficult at all and 4 being very difficult.
[b]Household Income was binned by $5000 increments that range from $0 to >$90 000 (scored 1-19).

(Astuti, Wardhana, Watkins, & Wulaningsih, 2017). Because both are chronic stressors, and we wanted to test for associations between UT and TL independently of SES, we ran our models with and without the SES covariates (financial strain, education years completed, and household income).

Our first two models tested included the unattributed UT variables (UT-Self, UT-Other). Models 3 and 4 included the UT variables attributed to race (UT-Race-Self, UT-Race-Other) and an interaction between UT-Race-Self and Race-Upset. The interaction was included to test whether the association between UT-Race-Self and TL varied by the persistence of negative emotions. We tested for multicollinearity in all four of our models. Variance inflation factors (VIFs) ranged from 1.021 to 2.063, all of which were in the acceptable range.

3 | RESULTS

Sample characteristics (N = 135) are presented in Table 2. The average age of participants was 41 years, and the sex ratio was roughly two women for every man. Average BMI (32 kg/m^2) fell in the moderately obese range (a BMI between 30 and 35) according to the Center for Disease Control. Roughly 42% of the sample reported that they were smokers at some point in their life. Participants reported experiences of unfair treatment, both personally (UT-Self) and by someone close to them (UT-Other) in ~1.5 domains on average. UT-Self and UT-Other were attributed to race in slightly more than one domain on average. Approximately half of the participants reported that they felt emotionally upset during the year prior to completing the survey as a result of how they were treated based on race. Participants most commonly reported financial strain (FS) at levels between "not much of a problem" (FS = 2) and "somewhat of a problem" (FS = 3). Mean education level completed was 1 year of college. Average household income was between $20000 and 25 000.

Pearson correlation testing revealed that UT-Race-Self (r = −0.172, 95%CI = −0.332, −0.004, P = .046) and age (r = −0.188, 95%CI = −0.346, −0.019, P = .029) were the only variables that correlated with TL in our dataset (Figure S1). The effect size of the age correlation is comfortably within the 95% confidence interval of most published values between blood TL and age (e.g., Eisenberg, Hayes, & Kuzawa, 2012; Rej, Tennyson, Lee, & Eisenberg, 2019).

Results of our multiple regression models are presented in Table 3. Of the covariates tested in all models (age, sex, BMI, and smoking status), only age was significantly associated with TL. In models 1 and 2, neither

TABLE 3 Multiple linear regression models including unfair treatment variables to predict salivary telomere length (log T/S ratio)

	Dependent variable: relative telomere length			
	Model 1 $\beta \pm$ SE (P value)	**Model 2 $\beta \pm$ SE (P value)**	**Model 3 $\beta \pm$ SE (P value)**	**Model 4 $\beta \pm$ SE (P value)**
Age	**−0.027 ± 0.013* (.038)**	**−0.028 ± 0.013* (.034)**	*−0.024 ± 0.013† (.071)*	*−0.026 ± 0.014† (.060)*
Sex (female)	−0.118 ± 0.350 (.737)	0.029 ± 0.363 (.937)	−0.129 ± 0.351 (.714)	0.019 ± 0.365 (.959)
BMI	0.001 ± 0.017 (.939)	0.001 ± 0.017 (.978)	−0.002 ± 0.017 (.916)	−0.000 ± 0.018 (.985)
Smoking (no)	−0.220 ± 0.319 (.491)	−0.146 ± 0.349 (.675)	−0.130 ± 0.328 (.693)	−0.054 ± 0.362 (.882)
UT-Self	−0.114 ± 0.082 (.164)	−0.095 ± 0.086 (.275)	-	-
UT-Other	0.059 ± 0.090 (.511)	0.046 ± 0.093 (.619)	-	-
UT-Race-Self	-	-	**−0.235 ± 0.107* (.030)**	**−0.239 ± 0.112* (.036)**
UT-Race-Other	-	-	−0.087 ± 0.099 (.383)	−0.067 ± 0.101 (.511)
Race-Upset (Yes)	-	-	−0.262 ± 0.390 (.503)	−0.375 ± 0.408 (.360)
UT-Race-Self × Race-Upset (Yes)	-	-	0.193 ± 0.171 (.261)	0.198 ± 0.175 (.260)
Financial strain	-	−0.005 ± 0.159 (.977)	-	0.009 ± 0.164 (.956)
Education (years)	-	−0.124 ± 0.077 (.109)	-	*−0.137 ± 0.081† (.091)*
Household income	-	0.048 ± 0.045 (.290)	-	0.054 ± 0.046 (.240)
R^2	0.055	0.070	0.083	0.100
Adjusted R^2	0.011	<0.000	0.021	0.009
Number of observations	135	129	127	121

*P < .05; †P < .1.

UT-Self nor UT-Other was significantly associated with TL. For model 3, we observed a significant association between UT-Race-Self and TL ($\beta = -0.235$, SE = 0.107, $P = .030$). When we ran model 3 independent of the interaction term (Table S2 and model S1), the strength of the primary association attenuated slightly ($\beta = -0.162$, SE = 0.085, $P = .059$). Inclusion of the interaction between UT-Race-Self and Race-Upset resulted in the modest improvement of model 3 (adjusted-R^2 increased to 0.021 from 0.019 and R^2 increased to 0.083 from 0.073). None of the SES variables included in models 2 and 4 significantly associated with TL. However, after accounting for SES, UT-Race-Self maintained its significant association with TL (Model 4: $\beta = -0.239$, SE = 0.112, $P = .036$). We also tested for associations between SES and TL independent of the unfair treatment variables and did not observe any significant results (Table S2 and Model S2).

4 | DISCUSSION

Experiences of discrimination stress may accelerate telomere shortening, and subsequently, hasten cellular aging and increase disease risk. As such, short TL is cited as a potential biomarker for studying the weathering hypothesis (Geronimus et al., 2015), which posits that chronic exposure to socially structured stressors accelerates biological aging in African Americans, thus unequally increasing vulnerability to diseases and other related conditions compared to European Americans (Geronimus et al., 2006). In this study, we examined the relationship between TL and racial discrimination, a psychosocial stressor that is pervasive in the lives of many African Americans. Working with data from a community-based research project in Tallahassee, FL, we observed an inverse association between TL and racial discrimination, as measured by unfair treatment to self and attributed to race (UT-Self-Race). Neither unattributed unfair treatment (UT-Self) nor unfair treatment experienced by a close friend or family member (UT-Other, UT-Race-Other) associated with TL, which suggests that personal experiences of UT that are perceived to be racially based might be particularly relevant to TL in this population.

It is interesting to speculate why only UT-Self-Race associated with TL. UT-Self did not associate with TL, prompting the interpretation that only unfair treatment attributed to race is processed in a pathway that leads to shortened TL. Neither of the UT-Other measures associated with TL, although we previously found associations of UT-Other and UT-Self with blood pressure through different biological pathways (Boulter et al., 2015; Quinlan et al., 2016). Specifically, in Quinlan et al. (2016), UT-Self associated with BP through interactions with genetic variants involved in cardiovascular disease and UT-Other associated with BP through interactions with genetic variants involved in mental health. The meaning and impact of unfair treatment may vary across dimensions like Self vs Other and Race-related vs general, and these differences may explain why we only find an association between TL and UT-Race-Self.

The amount of variation explained by our regression models (R^2 = 0.055-0.100; adjusted-R^2 = 0.000-0.021, Table 3) is small but comparable to those in other TL studies (eg, Barha et al., 2016; Bethancourt et al., 2017; Hill, Ellison, Burdette, Taylor, & Friedman, 2016). These studies included similar covariates as ours, including age, sex, BMI, SES (household income), and smoking status. After including the UT-Race variables in place of the unattributed UT variables (models 3 and 4 vs 1 and 2), we observed a modest improvement in the model; while adding the SES variables reduced adjusted-R^2 (models 2 and 4 vs models 1 and 3).

Other studies have also recently found associations between TL and discrimination (Chae et al., 2016, 2014; Lee et al., 2017; Liu & Kawachi, 2017). Two of these studies only reported significant findings after they added discrimination in a statistical interaction with either depressive symptoms (Chae et al., 2016) or an internalized anti-black racial bias (Chae et al., 2014). Meanwhile, two other studies found direct inverse associations between everyday discrimination and TL in African Americans enrolled in the Health Retirement Study (HRS) (Lee et al., 2017; Liu & Kawachi, 2017). An association between everyday discrimination at TL was not observed in the European Americans enrollees in the HRS. Across all of these studies, different measures were used to index discrimination including Krieger's Experiences of Discrimination (EOD) questionnaire (2001; used by the HRS) and the Everyday Discrimination Scale-Short Form (EDS-SF) by Sternthal and Williams (2011; used by Chae et al.). In our case, we used a modified version of the EDS-Long Form (Williams et al., 1997; Williams & Jackson, 2005) that accounted for social and cultural context. Thus, while each study captures slightly different aspects of the discrimination experience, all identify significant associations between TL and discrimination. Together, these results further suggest that discrimination is an important factor to include when investigating TL variation in African Americans.

Low SES is believed to increase stress levels, and differences in SES are commonly cited as a possible explanation for differences in TL between African Americans and European Americans (eg, Gebreab et al., 2016; Geronimus et al., 2015; Needham et al., 2013; Theall et al., 2013). While many of these studies have found

inverse associations between measures of SES and both TL and rate of telomere shortening in African Americans (eg, Needham et al., 2013; Schrock et al., 2018), others have failed to do so, while simultaneously reporting associations that link TL with discrimination (Chae et al., 2016; Lee et al., 2017; this study). We also did not observe any associations between SES and TL, either with or without the unfair treatment data. Together, these results suggest that discrimination should be considered as an important psychosocial stressor when predicting TL (or that other, uncollected measures of SES may better capture TL differences in our sample population).

While the inclusion of Race-Upset (or the emotional impact of UT-Race) and its interaction with UT-Race-Self improved our statistical models, neither was significant. While not significant, the association for the interaction term is in the expected direction, whereby individuals who experienced more UT-Race-Self and reported being upset by how they were treated based on race tended to have shorter telomeres than those who did not indicate that they were upset (Figure S2). Considering that individuals who answered yes to the Race-Upset question tended to report more experiences of UT-Race-Self ($F = 3.154$, $P = .078$) and UT-Race in general ($F = 8.488$, $P = .005$), as demonstrated by post hoc ANOVAs, it makes sense that, after controlling for the interaction, we observed an increase in association strength between UT-Race-Self and TL (from $\beta = -0.162$ to -0.235).

Still, our binary measure of the emotional impact of race-based unfair treatment is crude, as it fails to capture either the duration or the severity of the long-term effects of discrimination stress. Moreover, emotional responses to unfair treatment are complex. For example, Krieger, Sidney, and Coakley (1998) found only moderate associations between exposure to unfair treatment and ways of handling anger, and these associations also varied by social class and gender. For example, African American professionals who indicated that they were "very likely" to handle anger by keeping it to themselves were 4-5 times more likely to keep unfair treatment to themselves (Krieger et al., 1998). Working-class women, by contrast, were 3 times more likely more likely to keep it to themselves, while working-class men were 1.5 times more likely (Ibid). Previous research also suggests that internalized oppression could lead stigmatized groups to underreport discrimination or to downplay the emotional impact of such exposure (Krieger et al., 1998; Krieger & Sidney, 1996; Williams & Mohammed, 2013). Considering the evidence linking racial discrimination to increased inflammation and increased oxidative stress levels, as well as subsequent adverse mental and physiological health outcomes (Lewis et al., 2010; Szanton et al., 2012), the inclusion of more sophisticated measures of the emotional impact of discrimination, measures of internalized oppression, and larger sample sizes to detect complex interactions, are important future directions for this work.

One of the strengths of our study was that we worked with a community that was actively engaged in all aspects of research design (Boston, Mitchell, Collum, & Gravlee, 2015; Gravlee, Szurek, & Mitchell, 2015). While we cannot say if UT-Other, UT-Race-Self, and UT-Race-Other can be applied more broadly to African American communities outside of Tallahassee, we can stress the importance of active participant engagement in the development of these measures. In addition to TL, we have reported associations between our ethnographically informed, modified UT measures and blood pressure through interactions with genetic markers involved in both cardiovascular disease and mental health (Boulter et al., 2015; Quinlan et al., 2016). As such, we encourage other research groups to adopt a community-based participatory framework on both ethical and empirical grounds (Gravlee et al., 2015; Israel, Schulz, Parker, & Becker, 1998).

While we observed a significant association between TL and UT-Race-Self, there were several limitations to our study. First, our results were consistent with other studies that found associations between discrimination and TL, but we did not test all of the same variables, such as racial bias, and we used slightly different measures of discrimination and depressive symptoms (Chae et al., 2016, 2014; Lee et al., 2017; Liu & Kawachi, 2017). Second, we were limited by our sample size and the lack of information on other health behaviors previously included in studies that examined stress and TL (eg, diet, exercise, sleep, and general perceived stress) so our results should be investigated in a larger sample with additional covariates. Third, our measures of Race-Upset and Smoking are each based on a single question and lack information on duration and intensity. Race-Upset, in particular, could benefit from a more sophisticated measure of emotion sensitivity, intensity, and persistence, such as the Emotional Reactivity Scale or the Perth Emotional Reactivity Scale (Becerra, Preece, Campitelli, & Scott-Pillow, 2019; Nock, Wedig, Holmberg, & Hooley, 2008). Finally, we used DNA from saliva samples. While saliva is primarily composed of leukocytes, it is more heterogeneous than whole blood (the most common source of DNA for TL studies) (Theall et al., 2013; Thiede et al., 2000). Regardless, previous studies of salivary TL have shown moderate to strong associations with blood TL, the expected negative associations with age and low intra-sample variance, suggesting that saliva samples are a viable option for TL measurement (Chen et al., 2015; Goldman et al., 2017; Mitchell et al., 2014; Nelson et al., 2015; Stout et al., 2017).

In sum, our study revealed a significant negative association between self-reported unfair treatment attributed to race (UT-Race-Self) and TL in African Americans living in Tallahassee, FL. TL is a biomarker of aging, and short TL is associated with an increased risk of heart disease as well as with general mortality risk. Experiences of psychosocial stress are posited to hasten telomere shortening; a link that has been confirmed by multiple meta-analyses (Mathur et al., 2016; Pepper et al., 2018). The observed association may reflect the "weathering" caused by discrimination that possibly contributes to premature biological aging and racial health inequalities seen in African Americans. Further study of the stress caused by discrimination that is experienced by minorities should improve our understanding of the underlying cause and mechanism of racial inequalities in disease.

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AUTHOR CONTRIBUTIONS

P.H.R.: generated TL data, performed analyses, acquired funding, wrote the paper. H.S.C.: conceptualized the project, developed unfair treatment measures, edited the paper. C.C.G.: conceptualized the project, developed unfair treatment measures, acquired funding, edited the paper. C.J.M.: conceptualized the project, supervised the current study, acquired funding, wrote the paper.

Data Availability Statement

TL, anthropometric data, and socioeconomic and unfair treatment variables are freely available on the Open Science Framework public database. The URL to access this data is https://osf.io/3x9v7/.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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