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Socioeconomic status, health behavior, and leukocyte telomere length in the National Health and Nutrition Examination Survey, 1999–2002

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Abstract

The purpose of this study was to examine the association between socioeconomic status (SES) and leukocyte telomere length (LTL) – a marker of cell aging that has been linked to stressful life circumstances – in a nationally representative, socioeconomically and ethnically diverse sample of US adults aged 20–84. Using data from the National Health and Nutrition Examination Survey (NHANES), 1999–2002, we found that respondents who completed less than a high school education had significantly shorter telomeres than those who graduated from college. Income was not associated with LTL. African-Americans had significantly longer telomeres than whites, but there were no significant racial/ethnic differences in the association between education and telomere length. Finally, we found that the association between education and LTL was partially mediated by smoking and body mass index but not by drinking or sedentary behavior.

Keywords

socioeconomic status; cell aging; telomere length; health behavior; United States

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A large body of evidence links low socioeconomic status (SES) to the development of age-related diseases, such as cardiovascular disease, and earlier mortality (Adler & Rehkopf, 2008; Adler & Stewart, 2010). Several theoretical models share the assumption that the chronic stress associated with social disadvantage contributes to wear and tear on the body, which accelerates the rate of decline in physiological functioning (Geronimus et al., 2006; McEwen, 1998). Leukocyte telomere length (LTL), a biomarker of cell aging, may provide a link between the stress associated with social disadvantage and biological aging. Telomeres cap the ends of chromosomes and promote chromosomal stability. Telomere shortening, which tends to occur with advancing chronological age (Aubert & Lansdorp, 2008; Frenck et al., 1998; Iwama et al., 1998), causes cellular senescence in vitro (Blasco, 2005). Furthermore, a number of studies have found that LTL is associated with morbidity (e.g., Demissie et al., 2006; Samani et al., 2001; Zee et al., 2010) and mortality (e.g., Bakaysa et al., 2007; Cawthon et al., 2003; Weischer et al., 2012) independent of chronological age.

Although several studies have demonstrated an association between telomere length and stressful life circumstances (Damjanovic et al., 2007; Drury et al., 2011; Epel et al., 2004; Kananen et al., 2010; Tyrka et al., 2010), research examining the association between SES and LTL has produced mixed results (see Robertson et al., 2012a). While several studies have found a positive association between specific indicators of social status and telomere length (Adler et al., 2012; Carroll et al., 2013; Cherkas et al., 2006; Needham et al., 2012; Robertson et al., 2012b; Shiels et al., 2011; Steptoe et al., 2011; Surtees et al., 2012), others have found no association (Adams et al., 2007; Batty et al., 2009). One factor that could account for conflicting findings is inadequate statistical power due to small samples (Aviv, 2008). Using data from the National Health and Nutrition Examination Survey (NHANES) 1999–2002, the current study is the first to examine telomere length in a large, nationally representative, socioeconomically and ethnically diverse sample. Studies of LTL across large, heterogeneous samples, such as NHANES, can provide a deeper understanding of individual and group-level variation in the rate of biological aging.

The Telomere Hypothesis of Aging

Telomeres are the protective nucleoprotein structures capping the ends of eukaryotic chromosomes, consisting of a simple sequence of telomeric DNA (TTAGGG) tandemly repeated at each chromosome end. Telomeres naturally shorten with mitosis. Every time a cell divides, a portion of the telomeric DNA fails to replicate due to the “end replication problem” – that is, DNA polymerase does not completely copy the end of a linear DNA molecule (Blackburn, 2005; Olovnikov, 1973). Telomerase, the cellular ribonucleoprotein reverse transcriptase enzyme, can counteract shortening by elongating and protecting telomeres (Blackburn, 1997). However, telomerase is kept downregulated in normal human cells, and also can be decreased by aging and biochemical environment, such as oxidative stress levels and stress hormones (Choi et al., 2008; von Zglinicki et al., 1995).

Telomere shortening is not merely a marker of cellular aging – it appears also to have important functional consequences and is a mechanism of aging. Mitotic cells can undergo a limited number of cell divisions before they become senescent and lose the ability to grow and divide. Telomere shortening is a primary mechanism underlying this cellular senescence (although there are other mechanisms, such as stress-induced premature senescence (Sabin & Anderson, 2011)). If cells continue to divide, telomeres that are ‘too short’ lead to genomic instability, end-to-end chromosome fusion, less efficient mitosis, and loss of cellular proliferative capacity in vitro (Allsopp et al., 1992; Blackburn, 2000; Edo & Andres, 2005). When telomeres become critically shortened in leukocytes, they become senescent and secrete pro-inflammatory cytokines (Effros, 2004; Effros et al., 2005). New

experimental models in mice have shown that insufficient telomerase promotes accelerated aging and mortality, whereas high telomerase reverses aging deficits (Bernardes de Jesus et al., 2012; Jaskelioff et al., 2011). In addition, human mutations causing short telomeres cause a group of conditions collectively called "telomere syndromes" that resemble premature onset of common aging-related diseases (Armanios & Blackburn, 2012), consistent with a role of telomere shortening in human aging in the general population (Aubert & Lansdorp, 2008).[i]

Socioeconomic Status and Telomere Length

Socioeconomic status (also referred to as socioeconomic position, social class, or social status) includes the social and economic factors that shape an individual's position in society (Lynch & Kaplan, 2000). Commonly used indicators of SES include educational attainment, income, wealth, and occupational prestige. Individuals with low SES are exposed to more stressful conditions, such as chronic financial strain and exposure to hazardous work and home environments (Adler & Stewart, 2010). Low SES is also associated with decreased access to psychosocial resources, such as self-efficacy and social support, that can buffer the negative impact of stress on health (Adler & Stewart, 2010). Furthermore, given that stress responses are triggered by experiences in which individuals feel that the resources they have at hand are inadequate to deal with a threat, it is not surprising that people with low SES tend to demonstrate greater dysregulation of stress response systems, such as higher levels or altered diurnal patterns of stress hormones, such as cortisol (Cohen et al., 2006) and catecholamines (Janicki-Deverts et al., 2007), and lower heart rate variability (Sloan et al., 2005). Given the growing body of evidence demonstrating an association between telomere length and stressful life circumstances (Damjanovic et al., 2007; Drury et al., 2011; Epel et al., 2004; Kananen et al., 2010; Tyrka et al., 2010), LTL provides a potential biological link between low social status and morbidity and mortality.

Despite the plausibility of an association between SES and LTL, studies have produced mixed results (see Table 1 for a summary of findings). Cherkas and colleagues (2006) published the first paper in this area, which reported that women from manual social classes had shorter telomeres than women from non-manual classes. Two subsequent papers failed to replicate this finding (Adams et al., 2007; Batty et al., 2009), but more recent studies have found support for the hypothesis that low SES is associated with accelerated biological aging. For example, Shiels and colleagues (2011) found that renters (vs. home owners) and those with relatively low income experienced a faster rate of cell aging. Similarly, Robertson and colleagues (2012b) found that lower parental social class, lower educational attainment, and never owning a home were associated with shorter telomeres; while Carroll and colleagues (2013) found that father's education and current home ownership were associated with LTL. Four other studies have reported that low educational attainment (but not other measures of SES, such as income and occupation) is associated with shorter LTL in US and UK samples (Adler et al., 2012; Needham et al., 2012; Steptoe et al., 2011; Surtees et al., 2012).

With a few exceptions (see Adler et al., 2012; Carroll et al., 2013; Needham et al., 2012), previous research on SES and LTL has focused exclusively on populations of European ancestry. There are several reasons why it is important to examine racial/ethnic differences in the association between social status and telomere length. First, measures of SES may not be comparable across race/ethnic groups (Braveman et al., 2005; Williams & Collins, 1995). For example, levels of wealth, a potentially important determinant of morbidity and mortality, differ substantially between whites and non-whites at the same level of income;

[i]It should be noted, however, that some have questioned the utility of LTL as a biomarker of organismal aging (e.g., Der et al., 2012).

and this factor is not measured in most health datasets, including NHANES (Krieger et al., 1997). At an income of $15,000 per year, whites have on average $10,000 of net worth, while blacks have none (Conley, 1999). Thus a given income level will have different implications for health based on available resources for blacks versus whites. It is also well-documented that quality of education may differ substantially between race/ethnic groups (Card & Krueger, 1992). For this reason, a high school diploma or college degree may not provide the same health-related benefits for different racial/ethnic groups. Therefore, potential differences in the association between SES and LTL are critical to examine through analyses stratified by race/ethnicity.

Recent evidence suggests that telomere length may differ by race/ethnicity, but the direction has not been consistent. While some studies have found that blacks (Diez Roux et al., 2009; Geronimus et al., 2010) and Latinos (Diez Roux et al., 2009) have shorter LTL than whites, others have found just the opposite, with longer telomeres among blacks (Aviv et al., 2009; Hunt et al., 2008; Zhu et al., 2011). It is unclear if differences in findings are due to variations in characteristics of the samples and/or to other factors. Because NHANES oversampled African-Americans and Mexican-Americans, this study has adequate power to examine the association between SES and LTL within racial/ethnic categories.

Socioeconomic Status, Health Behavior, and Telomere Length

Health behavior is a major determinant of morbidity and mortality in the US (Ford et al., 2011) and the UK (Khaw et al., 2008). Recent studies also suggest that cigarette smoking (McGrath et al., 2007; Strandberg et al., 2011; Valdes et al., 2005), heavy drinking (Pavanello et al., 2011), sedentary behavior (Du et al., 2012; Ludlow & Roth, 2011), and obesity (Kim et al., 2009; Lee et al., 2011; Tzanetakou et al., 2012; Valdes et al., 2005) are associated with reduced telomere length. Because health behavior and obesity (which results from behaviors related to diet and activity) are strongly patterned by SES (Adler & Stewart, 2010), the current study examined these factors as potential mediators of the association between SES and LTL.

Hypotheses

Evidence is beginning to accumulate that individuals with lower social status have shorter telomeres than similar individuals with higher status. Use of larger, more representative data sets is needed to make sense of conflicting findings of studies on SES and LTL from smaller, isolated populations, and to contextualize these findings by examining potential moderators and mediators of the association. Based on the theory and evidence reviewed here, we examined the following hypotheses:

Hypothesis 1 SES, as measured by educational level and household income, will be positively associated with LTL.

Hypothesis 2 The association between SES and LTL will differ (be moderated) by race/ethnicity.

Hypothesis 3 Health behaviors (smoking, alcohol consumption, physical activity) and BMI will partially mediate the association between SES and LTL.

METHODS

Sample and Procedures

Since 1960, the National Center for Health Statistics of the Centers for Disease Control and Prevention (CDC) has conducted The National Health and Nutrition Examination Survey (NHANES) to provide national estimates of the health and nutritional status of the US

civilian non-institutionalized population. NHANES 1999–2002 is a cross-sectional, nationally representative sample of 21,004 individuals aged 2 months and older. NHANES 1999–2002 utilized a 4 stage sampling design: 1) primary sampling units (PSUs) consisting primarily of single counties, 2) area segments within PSUs, 3) households within segment areas, and 4) persons within households. On average, 2–3 individuals per household were sampled. NHANES 1999–2002 oversampled low-income persons, those aged 12–19, persons aged 60 and over, African-Americans, and Mexican-Americans in order to obtain more accurate estimates in these populations.

During two NHANES surveys (i.e., NHANES III and NHANES 1999–2002), DNA specimens were collected in order to establish a national probability sample of genetic material for future research. DNA samples from NHANES III are not suitable for the examination of telomere length, since the DNA is only available in the form of crude lysates of cell lines. The DNA collected in NHANES 1999–2002, however, is purified from whole blood, and is therefore suitable for the examination of telomere length.

All NHANES 1999–2002 respondents aged 20 and over were asked to provide DNA samples. Of the 10,291 respondents who were eligible to provide DNA, 7,825 (76%) both provided DNA and consented specifically to future genetic research. We excluded 653 respondents whose self-reported race/ethnicity was “other” or “other Hispanic,” since a goal of this study was to examine race/ethnic differences in the association between SES and LTL, and these groups are too diverse for our purposes. Given the possibility of survival bias among the extreme elderly (Aviv et al., 2006), we also excluded 225 respondents aged 85+. An additional 1,587 respondents were excluded from the analytic sample due to missing data on one or more variables included in the models (final n=5,360). The amount of missing data for each variable ranged from 0–8.8%, and there were no significant sociodemographic differences between the full sample and the final analytic sample. To account for oversampling and non-response bias, sampling weights must be used in the analysis. This helps ensure that estimates are representative of the general US population. Human subjects approval for this study was provided by the Institutional Review Board at the CDC.

Telomere Length Assay

Aliquots of purified DNA were provided by the laboratory at the Division of Health and Nutrition Examination Surveys, National Center for Health Statistics, Centers for Disease Control and Prevention. Using standardized procedures, DNA was extracted from whole blood and stored at −80°. The LTL assay was performed in the laboratory of Dr. Elizabeth Blackburn at the University of California, San Francisco, using the quantitative polymerase chain reaction (PCR) method to measure telomere length relative to standard reference DNA (T/S ratio), as described in detail elsewhere (Cawthon, 2002; Lin et al., 2010). The single-copy gene used as a control to normalize input DNA was human beta-globin. Each sample was assayed at least twice. T/S ratios that fell into the 7% variability range were accepted, and the average of the two was taken as the final value. A third assay was run for samples with greater than 7% variability, and the average of the two closest T/S values was used. The inter-assay coefficient of variation (CV) was 4.4%.

The conversion from T/S ratio to base pairs (bp) was calculated based on comparison of telomeric restriction fragment (TRF) length from Southern blot analysis and T/S ratios using DNA samples from the human diploid fibroblast cell line IMR90 at different population doublings. The formula to convert T/S ratio to bp was 3,274 + 2,413 * (T/S). DNA samples were coded and the lab was blinded to all other measurements in the study. The CDC conducted a quality control review before linking the telomere data to the NHANES 1999–2002 public use data files.

SES Measurement

The SES measures were education and income. The education measure included dummy variables for less than high school, high school, and some college, with college degree as the reference category. The income measure was the Poverty Income Ratio (PIR), calculated as the ratio of income to the poverty threshold for a household of a given size and composition. PIR values below 1.00 are below the official poverty threshold. PIR was examined as a continuous measure and as a dichotomous measure (below the poverty threshold=1; at or above the poverty threshold=0).

Measurement of Mediators

Hypothesized mediators included smoking, drinking, physical inactivity, and obesity. The smoking measure indicated cumulative exposure to tobacco smoke in pack-years, calculated as the average number of cigarettes smoked per day times the number of years smoked, divided by 20 (dummy variables for less than 30 pack-years, more than 30 but less than 60 pack-years, and 60 or more pack-years, with never smoker as the reference category) (Mannino et al., 2003). Alcohol use included dummy variables for abstainers (women and men who reported no alcohol consumption in the past 12 months) and heavy drinkers (women who reported having drunk 2 or more alcoholic beverages per day in the past 12 months and men who reported having drunk 3 or more alcoholic beverages per day in the past 12 months), with moderate drinkers (women who reported having drunk more than 0 but less than 2 drinks per day in the past 12 months and men who reported having drunk more than 0 but less than 3 drinks per day in the past 12 months) as the reference category (Ford et al., 2011). Physical inactivity was defined as participating in less than 10 minutes of moderate or vigorous activity or strength training in the previous 30 days. Body mass index (BMI) was the ratio of weight/height^2 in kg/m^2. BMI was examined as a continuous measure.

Data Analysis

Telomere length was transformed by natural logarithm prior to regression modeling. Therefore we report the percentage change in the average value of telomere length (the outcome variable) for a one-unit change in an explanatory variable. Because the absolute value of all parameter estimates was <.10, the percentage change in the outcome was estimated by multiplying the parameter estimate (*b*) by 100 (Vittinghoff et al., 2012). All regression models accommodated the complex sampling design of NHANES by incorporating strata and PSU indicators, as well as sample weights for the genetic subsample (National Center for Health Statistics, 2006). Analyses were conducted on-site at the CDC Research Data Center in Atlanta and remotely using ANDRE, the CDC's remote access system for the analysis of restricted data.

The hypothesis that SES is positively associated with telomere length was tested with a linear model regressing log LTL (T/S ratio) on education, income, and a core set of covariates described below. We only present results for the continuous measure of PIR, since the continuous and dichotomous measures produced substantively equivalent results. To test the hypothesis that the association between SES and LTL is moderated by race/ethnicity, we examined the model separately for whites, African-Americans, and Mexican-Americans. After estimating stratified models, we compared corresponding parameter estimates across racial/ethnic groups using *z*-tests (African American versus white, Mexican American versus white, and African-American versus Mexican-American) (Paternoster et al., 1998). Significant *z*-tests suggest moderating effects of race/ethnicity. Finally, the third hypothesis, which stated that health behaviors and BMI partially mediate the association between SES and LTL, was tested with a series of regression models (Baron & Kenny, 1986) using methodological extensions to accommodate categorical mediators (Iacobucci,

2012). We calculated Aroian tests of the indirect effects of SES on LTL through smoking, drinking, physical inactivity, and BMI. A significant Aroian test suggests a significant indirect effect of SES on LTL via a candidate mediator (Iacobucci, 2012).

All models included controls for the following potential confounders: age (in years), sex/gender (female=1; male=0), nativity (foreign-born=1; US-born=0), and marital status (married=1; never married, separated, divorced, or widowed=0). All models also included an age-squared term to account for potential nonlinearity in the association between age and LTL. Models fit to data pooled across race/ethnicity included dummy variables for African-American and Mexican-American, with white as the reference category.

It is not clear whether chronic health conditions are a predictor or an outcome of reduced telomere length. If they are, in fact, a predictor of reduced LTL, then chronic conditions could potentially confound the association between SES and LTL. For this reason, we examined models with and without controls for the following chronic health conditions: cardiovascular disease (participant reported being told by their physician that they ever had a heart attack or stroke, or participant reported being told by their physician that they had coronary heart disease=1; no heart attack, stroke, or coronary heart disease=0), diabetes (participant reported current use of medication for diabetes, or participant had a glycosylated hemoglobin level greater than 7%=1; no diabetes medication or glycosylated hemoglobin less than or equal to 7%=0), hypertension [participant reported being told by their physician that they had high blood pressure, or participant reported current use of medications to treat high blood pressure, or average measured blood pressure (the average of all measurements taken after dropping the first measurement) was 140 mmHg systolic or 90 mmHg diastolic=1; no high blood pressure or blood pressure medication=0], and high cholesterol (participant reported physician diagnosis of high cholesterol, or participant reported current use of medication to treat high cholesterol, or participant had a total cholesterol level of 240 mg/dL or higher=1; no high cholesterol or cholesterol medication=0). Results were the substantively equivalent regardless of the inclusion of controls for chronic conditions. For the sake of parsimony, we present the results of models that do not include controls for cardiovascular disease, diabetes, hypertension, or high cholesterol. Unweighted descriptive statistics for all study variables are shown in Table 2.

RESULTS

Telomere length was inversely associated with age. As shown in Panel A of Table 3, one year of additional age was associated with a .6% decrease in log T/S ratio in the full sample ($b = -.006$, $p < .01$). In addition, the telomeres of respondents who did not graduate from high school were approximately 4% shorter than the telomeres of those who graduated from college ($b = -.042$; $p < .01$). Holding income, race/ethnicity, age, sex, nativity, and marital status constant, the average log-transformed T/S ratio for those with the lowest level of education was −.042 compared to .010 for those with the highest level of education. This corresponds to a difference of 102 bp. Given the model-based estimate of the age-associated rate of telomere shortening in this study of 14.60 bp per year, the difference between low and high SES respondents of the same chronological age was roughly equivalent to 7 years of additional aging.

In addition to age and education, we also found that race/ethnicity was a significant predictor of LTL in the full sample. The telomeres of African-Americans were approximately 5% longer than the telomeres of whites of the same age and sex ($b = .048$, $p < .01$). The difference in the average log-transformed T/S ratio for whites compared to African-Americans corresponds to a difference of 116 bp, which, if no other factors invalidate the comparison, would translate to about 8 years of additional aging for whites

compared to African-Americans. In contrast to these results, we found no significant difference in LTL between whites and Mexican-Americans.

In models stratified by race/ethnicity (see Panels B-D of Table 3), we found that age was significantly inversely associated with LTL among whites (b = −.005, p < .01) and Mexican-Americans (b = −.010, p < .001) but not among African-Americans. The difference in the association between age and LTL was significant for African-Americans and Mexican-Americans ($z_{AAvs.MA}$ = 2.31, p = .02), but not for whites and African-Americans ($z_{Wvs.AA}$ = .93, p = .35) or for whites and Mexican Americans ($z_{Wvs.MA}$ = 1.46, p = .14). In addition, gender was associated with LTL only in the African-American sample, in which women's telomeres were approximately 6% longer than those of men (b = .055, p < .01). The association between gender and LTL was significantly different between African-Americans and whites ($z_{Wvs.AA}$ = 2.23, p = .03) but not between African-Americans and Mexican-Americans ($z_{AAvs.MA}$ = 1.61, p = .11). Finally, we found that white respondents who did not graduate from high school had significantly shorter LTL than white college graduates (b = −.050, p < .01) while education was not related to LTL among African-Americans and Mexican-Americans. However, race/ethnic differences in the association between education and LTL were not statistically significant ($z_{Wvs.AA}$ = 1.23, p = .22; $z_{Wvs.MA}$ = 1.29, p = .20; $z_{AAvs.MA}$ = .13, p = .90).

Next, we examined the potential mediating role of health behaviors and BMI on the association between SES and LTL. Because the association between education and LTL was not significantly different across race/ethnic groups, we performed tests of mediation in the full sample. As shown in Model 2 of Table 4, those reporting 60 pack-years of smoking or more had significantly shorter telomeres than never-smokers (b = −.039, p < .05), and BMI was inversely associated with log T/S ratio (b = −.002, p < .01). Alcohol use and sedentary behavior were not significantly associated with telomere length. The association between education and LTL was partially mediated by smoking ($z_{smoking}$ = 2.80, p = .01) and BMI (z_{bmi} = 2.21, p = .03) but not by drinking ($z_{drinking}$=.09, p = .93) or sedentary behavior ($z_{sedentary}$ = 1.50, p = .14).

DISCUSSION

Although socioeconomic disparities in morbidity and mortality are well-documented, we know relatively little about the biological mechanisms underlying the association between social status and health. Based on theory and research suggesting that chronic stress associated with disadvantaged social status may lead to acceleration in the rate of decline in physiological functioning (Geronimus et al., 2006; McEwen, 1998), we hypothesized that SES would be positively associated with telomere length, a biomarker of cell aging. Using data from the National Health and Nutrition Examination Survey (NHANES), 1999–2002, we found that respondents who completed less than a high school education had significantly shorter telomeres than those who graduated from college. We found no association between income and telomere length. Consistent with some prior studies, but contrary to the stress hypothesis, we found that African-Americans had significantly longer telomeres than whites; and we found no evidence of racial/ethnic differences in the association between SES and LTL. Finally, we found that smoking and body mass index partially mediated the association between education and LTL.

Strengths and Limitations

These results from a large, nationally representative sample are consistent with some previous studies of SES and LTL (Adler et al., 2012; Needham et al., 2012; Robertson et al., 2012b; Steptoe et al., 2011; Surtees et al., 2012) but inconsistent with others (Adams et al., 2007; Batty et al., 2009; Carroll et al., 2013; Cherkas et al., 2006; Shiels et al., 2011).

Discrepant findings in the literature on telomere epidemiology may be due, in part, to the use of small and/or non-representative samples (Aviv, 2008). Despite the advantages of using a large, nationally representative, socioeconomically and ethnically diverse sample to examine the association between SES and LTL, limitations should be noted. First, SES is indexed by occupation as well as by income and education, and British studies have shown associations of occupational class and telomere length. Although NHANES asked respondents about their longest-held occupation, the data were missing for 2,216 out of the 5,360 respondents in our analytic sample (41% missing). In the subsample of respondents with data on occupation, we found no significant association between occupation and LTL. Given the large amount of missing data and the lack of evidence for an association between occupation and LTL, we decided not to include occupation in the models. Second, the analyses are cross-sectional. While we were able to examine the relationship between SES and current telomere length, the findings do not tell us when the effects occur; longitudinal data would allow for an analysis of the rate of telomere shortening as well as current length. Finally, we did not have a direct assessment of stress exposure and cannot estimate the extent to which SES effects on LTL operate through stress physiology.

Directions for Future Research

This study produced several intriguing findings that highlight the need for additional research. First, work is needed to determine the extent to which socioeconomic differences in mortality are explained by differences in telomere length. Additional work is also needed to understand race/ethnic differences in LTL. African-Americans in this study had longer telomeres than whites, consistent with several other studies (Aviv et al., 2009; Hunt et al., 2008; Zhu et al., 2011). This finding is somewhat perplexing, given that African-Americans have higher morbidity and mortality than whites. The measure of LTL used in this study is an average of telomere length across all leukocyte cell types, including neutrophils, eosinophils, basophils, lymphocytes, and monocytes. Previous research suggests that, within the same individual, telomere length in different cell types varies (Lin et al., 2010). Given evidence of black/white differences in leukocyte cell subpopulations (Freedman et al., 1997), future work should attempt to assess LTL in a single cell type, such as monocytes, to determine whether racial/ethnic heterogeneity in cell types explains why African-Americans have longer *overall* LTL than whites. Additionally, more work is needed to determine whether the association between gender and LTL is in fact different for African-Americans and whites. Contrary to most previous studies, we found no significant association between gender and LTL in the full sample (Barrett & Richardson, 2011); a significant gender difference was only observed in the African-American subsample. Finally, we found that smoking and BMI partially mediated the association between education and LTL while alcohol use and lack of exercise did not. This may be rationalized in that drinking is a complex behavior which is protective in low doses, and exercise may operate through BMI to affect health. Future studies should explore other potential mediators, such as diet, history of infection, and exposure to environmental toxins, as well as exposure to stressful environments.

Conclusions

This study adds to the growing body of literature demonstrating an association between disadvantaged social status and cell aging. Because we examined data from a large (n=5,360), nationally representative data set, the results of this work are generalizable to the US adult population. Together with the results of other recent work on stress and cell aging, this study suggests that LTL is one biological mechanism by which social conditions “get under the skin” to affect health.

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Research Highlights

- Leukocyte telomere length (LTL) is a marker of cell aging
- Previous research on socioeconomic status (SES) and LTL has produced mixed results
- This is the first study to examine SES and LTL in a nationally representative sample of US adults
- We found that education was positively associated with LTL
- This association was partially mediated by smoking and body mass index

Table 1

Summary of the Main Study Findings Regarding the Association between SES and Telomere Length

Study	Sample	Method	Major Findings
Cherkas et al. 2006	1552 white female twins from the St. Thomas' (TwinsUK) Twin Registry, aged 18–75	Southern blot	Women with lower occupational status (self or husband) had shorter telomeres than women with higher occupational status (no association with education or income)
Adams et al. 2007	318 white women and men from the Newcastle Thousand Families Study, aged 50	PCR	No evidence of an association between social class (occupation and income) and telomere length
Batty et al. 2009	1542 white men from the West of Scotland Coronary Prevention Study, aged 45–64	PCR	Unemployed men had shorter telomeres than employed men (no association with education or area-based deprivation)
Shiels et al. 2011	382 white women and men from the pSoBid Study, aged 35–64	PCR	The rate of age-related telomere shortening was greater among renters and those with low income (no association with education or occupation)
Steptoe et al. 2011	448 white women and men from the Whitehall II Study, aged 53–76	PCR	Lower educational attainment was associated with shorter telomere length (no association with household income and employment grade)
Surtees et al. 2012	4441 white women from the (Epic)-Norfolk Study, aged 41–80	PCR	Lower educational attainment was associated with shorter telomere length (no association with occupation)
Needham et al. 2012	70 white and black girls and boys from the AMERICO Study, aged 7–13	PCR	Lower parental educational attainment was associated with shorter telomere length in children (no association with family income)
Adler et al. 2012	2599 white and black adults from the Healthy Aging, and Body Composition Study, aged 70–79	PCR	Lower educational attainment was associated with shorter telomere length (no association with income); the association was stronger for blacks than whites
Robertson et al. 2012	2185 white women and men from the West of Scotland Twenty-07 Study, aged 35–75	PCR	Lower parental social class, lower educational attainment, and never owning a home were associated with shorter telomere length among the younger adults only (no association with occupation, income, area deprivation, employment status, self-rated child or adult SES, childhood income or wealth, social class mobility, or accumulated SES)
Carroll et al. 2013	963 white, black, and Hispanic adults from the Multi-Ethnic Study of Atherosclerosis, aged 45–84	PCR	Father's education was positively associated with telomere length; among whites and Hispanics only, renters had shorter telomeres than home owners (no association with adult education or income)

Table 2

Unweighted Descriptive Statistics for All Study Variables

	Full Sample (n=5,360)		Whites (n=2,997)		African-Americans (n=986)		Mexican-Americans (n=1,377)	
	Prop.	**Mean (SD)**	**Prop.**	**Mean (SD)**	**Prop.**	**Mean (SD)**	**Prop.**	**Mean (SD)**
Leukocyte telomere length (T/S ratio)	---	1.02 (.26)	---	1.02 (.27)	---	1.09 (.29)	---	1.00 (.23)
Educational Attainment								
Less than high school	0.32	---	0.16	---	0.38	---	0.62	---
High school	0.23	---	0.28	---	0.22	---	0.15	---
Some college	0.25	---	0.28	---	0.27	---	0.17	---
College degree	0.2	---	0.28	---	0.13	---	0.06	---
Poverty income ratio	---	2.75 (1.62)	---		---		---	
Age	---	48.58 (17.91)	---	50.84 (18.32)	---	46.31 (16.63)	---	45.27 (17.13)
Sex/gender								
Female	0.52	---	0.51	---	0.53	---	0.54	---
Male	0.48	---	0.49	---	0.47	---	0.46	---
Nativity								
Foreign-born	0.2	---	0.05	---	0.1	---	0.4	---
US-born	0.8	---	0.95	---	0.9	---	0.6	---
Marital status								
Married	0.61	---	0.66	---	0.38	---	0.66	---
Never married, separated, divorced, or widowed	0.39	---	0.34	---	0.62	---	0.34	---
Pack years smoking								
0 pack years	0.57	---	0.52	---	0.6	---	0.66	---
< 30 pack years	0.31	---	0.31	---	0.31	---	0.28	---
30–59 pack years	0.09	---	0.11	---	0.07	---	0.04	---
60+ pack years	0.04	---	0.06	---	0.02	---	0.02	---
Drinking								
Abstainer	0.38	---	0.33	---	0.46	---	0.43	---
Moderate drinker	0.59	---	0.64	---	0.5	---	0.55	---
Heavy drinker	0.03	---	0.03	---	0.04	---	0.02	---

	Full Sample (n=5,360)		Whites (n=2,997)		African-Americans (n=986)		Mexican-Americans (n=1,377)	
	Prop.	**Mean (SD)**	**Prop.**	**Mean (SD)**	**Prop.**	**Mean (SD)**	**Prop.**	**Mean (SD)**
Physical activity								
Sedentary	0.39	---	0.3	---	0.5	---	0.5	---
Active	0.61	---	0.7	---	0.5	---	0.5	---
Body mass index	---	28.45 (6.20)	---	27.90 (5.90)	---	29.84 (7.56)	---	28.66 (5.55)

Notes: Prop. = Proportion; SD = Standard Deviation.

Table 3

OLS Regression of Log-Transformed Telomere Length (T/S Ratio) on Education and Income

	A. Full Sample (n=5,360)		B. Whites (n=2,997)		C. African-Americans (n=986)		D. Mexican-Americans (n=1,377)	
	Est.	**SE**	**Est.**	**SE**	**Est.**	**SE**	**Est.**	**SE**
Education (College degree)								
Less than high school	−.042**	0.015	−.050**	0.018	−0.009	0.028	−0.004	0.031
High school	−0.016	0.015	−0.016	0.016	−0.012	0.036	0.02	0.029
Some college	−0.016	0.015	−0.015	0.017	−0.015	0.028	0.021	0.03
Poverty income ratio	0.004	0.005	0.005	0.006	−0.002	0.006	0.001	0.01
Race/ethnicity (White)								
African-American	.048**	0.016	---	---	---	---	---	---
Mexican-American	−0.034	0.022	---	---	---	---	---	---
Age	−.006**	0.002	−.005*	0.002	−0.003	0.002	−.010***	0.003
Age^2	0	0	0	0	0	0	0	0
Female (Male)	0.017	0.009	0.011	0.011	.055**	0.016	0.023	0.012
Foreign-born (US-born)	0.019	0.017	0.019	0.024	0.05	0.04	−0.004	0.024
Married (Never married, separated, divorced, or widowed)	−0.01	0.011	−0.012	0.013	−0.001	0.019	0.007	0.02
Intercept	.274***	0.039	.274***	0.049	.246***	0.057	.310***	0.079
R^2	0.17		0.17		0.16		0.15	

*p<.05;

**p<.01;

***p<.001

Notes: OLS = Ordinary Least Squares; Est. = Estimate; SE = Standard Error.

Table 4

OLS Regression of Log-Transformed Telomere Length (T/S Ratio) on Education, Income, Health Behavior, and BMI (n=5,360)

	Model 1		Model 2	
	Est.	**SE**	**Est.**	**SE**
Education (College degree)				
Less than high school	−.042**	0.015	−.033*	0.013
High school	−0.016	0.015	−0.01	0.014
Some college	−0.016	0.015	−0.011	0.014
Poverty income ratio	0.004	0.005	0.003	0.005
Race/ethnicity (White)				
African-American	.048**	0.016	.051**	0.017
Mexican-American	−0.034	0.022	−0.033	0.022
Age	−.006**	0.002	−.004*	0.002
Age^2	0	0	0	0
Female (Male)	0.017	0.009	0.016	0.009
Foreign-born (US-born)	0.019	0.017	0.014	0.016
Married (Never married, separated, divorced, or widowed)	−0.01	0.011	−0.01	0.011
Pack years smoking (0 pack years)				
< 30 pack years	---	---	0.006	0.007
30–59 pack years	---	---	−0.03	0.017
60+ pack years	---	---	−.039*	0.018
Drinking (Moderate drinker)				
Abstainer	---	---	0.004	0.011
Heavy drinker	---	---	0.001	0.023
Sedentary (Active)	---	---	−0.012	0.009
Body mass index	---	---	−.002**	0.001
Intercept	.274***	0.039	.300***	0.041
R^2	0.17		0.18	

* p<.05;

** p<.01;

*** p<.001

Notes: OLS = Ordinary Least Squares; Est. = Estimate; SE = Standard Error.