Socioeconomic status and cell aging in children

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Abstract

Theory suggests that chronic stress associated with disadvantaged social status may lead to acceleration in the rate of decline in physiological functioning. The purpose of this study is to examine the association between parental socioeconomic status (SES) and leukocyte telomere length (LTL), a marker of cell aging, in children. We examined SES and LTL in 70 white and black US children aged 7–13 who participated in the community-based AMERICO (Admixture Mapping for Ethnic and Racial Insulin Complex Outcomes) study. LTL was assessed using the polymerase chain reaction (PCR) method. Parental education was positively associated with child LTL, net of controls for sex, age, race/ethnicity, and family income. Compared to children with at least one college-educated parent, children whose parents never attended college had telomeres shorter by 1,178 base pairs, which is roughly equivalent to 6 years of additional aging. Socioeconomic disparities in cell aging are evident in early life, long before the onset of age-related diseases.

Introduction

A large body of research has demonstrated an association between socioeconomic status (SES) and morbidity and mortality (Adler & Rehkopf, 2008). Several theoretical models share the assumption that chronic stress associated with low social status leads to wear and tear on the body that accelerates the rate of decline in physiological functioning (Adams & White, 2004; Geronimus, Hicken, Keene, & Bound, 2006; McEwen, 1988; Pearlin, 1989). Leukocyte telomere length (LTL), a marker of cell aging, may provide a link between the stress associated with low SES and the risk of disease (Bauer, Jeckel, & Luz, 2009). The purpose of this study is to determine whether socioeconomic disparities in cell aging are evident early in life, before the onset of age-related diseases, such as cardiovascular disease, diabetes, stroke, and cancer.

Telomeres are repeat sequences of DNA that, together with associated protein factors, cap the ends of chromosomes and promote chromosomal stability. Telomeric DNA shortening tends to occur with advancing chronological age (French, Blackburn, & Shannon, 1998; Iwama et al., 1998; Lee, Nam, Terao, & Yoshikawa, 2002) and leads to cellular senescence in vitro (Blasco, 2005). Several studies have demonstrated that shorter LTL is associated with morbidity (Brouilette, Singh, Thompson, Goodall, & Samani, 2003; Samani, Boulby, Butler, Thompson, & Goodall, 2001) and mortality, independent of chronological age (Bakaysa et al., 2007; Cawthon, Smith, O’Brien, Sivatchenko, & Kerber, 2003; Kimura et al., 2008). Although previous research has demonstrated an association between LTL and stressful life circumstances (Drury et al., 2011; Entringer et al., 2011; Epel et al., 2004), studies examining the association between SES and LTL in adults have produced conflicting results. While Cherkas et al. (2006) found that LTL was significantly shorter in women of lower SES, several other studies have found little association of SES with cell aging, as measured by LTL (Adams et al., 2007; Batty et al., 2009). This is the first study to examine the association between SES and LTL in a sample of children.

Parental SES and child health

Research on parental SES and child health indicates that numerous health disparities are evident during childhood, including disparities in premature birth, injuries, respiratory illnesses, dental caries, hospitalization, and self-rated health (for a review, see Bradley & Corwyn, 2002). Compared to high SES children, those who live in low SES families may have worse health because (1) they are exposed to more negative life events, (2) they are more likely to experience psychological distress, (3) they are more likely to develop health-damaging personality traits, such as hostility and pessimism, and (4) they are less likely to engage in healthy behaviors, such as exercise (Chen, 2004). In addition,
childhood may represent a “critical” or “sensitive” period in development, whereby exposure to conditions associated with low SES may have long-term consequences for health and well-being, even among individuals who experience improvement in socioeconomic conditions over time (Cohen, Janicki-Deverts, Chen, & Matthews, 2010; Hertzman & Wiens, 1996; Shonkoff, Boyce, & McEwen, 2009).

Despite evidence of socioeconomic disparities in child health, overall rates of morbidity and mortality are low during this stage of the life course. Furthermore, the types of health problems that are observable during childhood, such as accidental injuries, are not leading causes of adult morbidity and mortality. In order to demonstrate the link between childhood social conditions and adult health, it would be useful to identify an indicator of childhood health status that is related to the onset of diseases, such as cancer and cardiovascular disease, later in life. Leukocyte telomere length, a marker of cell aging that is associated with adult morbidity and cardiovascular disease, later in life. Leukocyte telomere length, health status that is related to the onset of diseases, such as cancer overall rates of morbidity and mortality are low during this stage of childhood, with college graduate as the reference category in all analyses. The original income variable ranged from 1 to 9 (in $10,000 increments from 1 = 0–9999 to 9 = 80,000+). Given that responses were not normally distributed, we constructed a three-category measure of income: <$40,000 per year (20%), $40,000–$69,000 per year (64%), and $70,000+ per year (16%). We then constructed dummy variables for income <$40,000 per year and $40,000–$69,000 per year with income of $70,000 or more per year as the reference category in all analyses.

Hypotheses

Given the evidence of socioeconomic health disparities during childhood (Bradley & Corwyn, 2002), we expected to find a positive association between parental SES and child telomere length. As proposed by Chen (2004), we also expected to find that the association between SES and LTL is partially mediated by health behavior. Based on previous research examining the association between telomere length and health behavior in adults, we examined fruit and vegetable consumption (Paul, 2011), physical activity (Cherkas et al., 2008), and body mass index (Valdes et al., 2005) as potential mediators.

Methods

Sample and procedures

The study sample includes 35 white and 35 black children selected at random from the full sample of 120 white, 120 black, and 120 Latino children who participated in the community-based AMERICO (Admixture Mapping for Ethnic and Racial Insulin Complex Outcomes) study. Data were collected in Birmingham, Alabama, between 2005 and 2008. Subjects were recruited through newspaper postings and fliers and recruitment activities at churches, schools, and community centers. The blood draw took place at 7:00 am, after a 12 h fast. Children were healthy and without observable illnesses at the time of the blood draw, according to parental report. Written, informed consent was obtained from each child’s parent or legal guardian. Human subjects approval for this study was granted by the Institutional Review Board at University of Alabama at Birmingham.

SES measurement

The SES measures included parental education (for the most highly educated parent) and total household income. The data set included a measure of educational attainment that ranged from 1 to 7 (1 = less than 8th grade, 2 = 9th—10th grade, 3 = 10th—11th grade, 4 = high school, 5 = partial college or special training, 6 = college, 7 = graduate or professional school). Because responses were not normally distributed, we constructed a three-category measure of educational attainment: high school graduate (13%), some college (17%), and college graduate (70%). We then constructed dummy variables for high school graduate and some college, with college graduate as the reference category in all analyses. The original income variable ranged from 1 to 9 (in $10,000 increments from 1 = 0–9999 to 9 = 80,000+). Given that responses were not normally distributed, we constructed a three-category measure of income: <$40,000 per year (20%), $40,000–$69,000 per year (64%), and $70,000+ per year (16%). We then constructed dummy variables for income <$40,000 per year and $40,000–$69,000 per year with income of $70,000 or more per year as the reference category in all analyses.

Measurement of mediators

Proposed mediators include fruit and vegetable consumption, physical activity, and body mass index. Respondents were asked to report the number of fruits and vegetables consumed per day. Responses ranged from 0 to 8 for fruits and 0–11 for vegetables. We used the Physical Activity Questionnaire for Older Children (PAQ-C) to assess physical activity (Crocker, Bailey, Falknner, Kowalski, & McGrath, 1997). The PAQ-C is a self-administered questionnaire that assesses moderate to vigorous physical activity during the past seven days. Previous studies have established validity and reliability of the PAQ-C (Kowalski, Crocker, & Faulkner, 1997). The activity composite score ranges from 1 (low physical activity) to 5 (high physical activity). Body mass index (weight (kg)/height (m²)) was calculated using measures of weight and height obtained by a trained interviewer.

Telomere length assay

Using standardized procedures, DNA was extracted from whole blood and stored for 2–5 years at −80°C. The LTL assay was performed in the laboratory of Dr. Elizabeth Blackburn at the University of California, San Francisco, by Dr. Jue Lin, 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., 2009). 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 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 3274 + 2413 * (T/S). DNA samples were coded and the lab was blinded to all other measures in the study.

Data analysis

Because LTL is normally distributed in this sample, we used ordinary least squares (OLS) regression to examine the association between parental SES and child telomere length. The model included controls for sex (1 = female, 0 = male), age (in years), and race/ethnicity (1 = black, 0 = white). See Table 1 for descriptive statistics for all study variables.

Results

As shown in Table 2, age was inversely associated with LTL, but we found no sex or race/ethnic differences in telomere length. Parental education was associated with child telomere length, while family income was only significant at the p < .10 level. Compared to those with the highest family income ($70,000+ per year), children living in low-income households (<$40,000 per
year) had a lower average T/S ratio ($b = -3.82$; 95% Confidence Interval $= -3.30$, $p = .007$) than children living in high-income households. Compared to children with at least one college-educated parent, those whose most highly educated parent never attended college had a lower average T/S ratio ($b = -4.49$; 95% Confidence Interval $= -4.88$, $p = .002$).

As shown in Table 3, we found that none of the proposed mediators was associated with LTL. Therefore, the association between parental education and income and child telomere length was not mediated, or explained, by fruit or vegetable consumption, physical activity, or body mass index (Baron & Kenny, 1986).

### Discussion

This preliminary study suggests that socioeconomic disparities in telomere length, a marker of cell aging, are evident in early life, long before the onset of age-related diseases. This disparity does not appear to be attributable to differences in fruit or vegetable consumption, physical activity, or body mass index. Although previous research on SES and LTL in adults has produced mixed results (see Adams et al., 2007; Batty et al., 2009; Cherkas et al., 2006), the current study suggests that low social status may contribute to accelerated cell aging during childhood.

Controlling for age, sex, race/ethnicity, and family income, the average T/S ratio for children whose parent completed college was no more than a high school education was 1.77 compared to 2.26 for children whose parent completed college. This corresponds to a difference of 1178 bp. Given the observed cross-sectional rate of telomere shortening in this study of 196 bp per year, which is consistent with previous research on children using Southern blot analysis (Frenck et al., 1998), the difference in bp between low and high SES respondents of the same chronological age is roughly equivalent to 6 years of additional aging.

The main strength of this study is the availability of data on SES and LTL for children. To our knowledge, no previous studies have examined the association between childhood socioeconomic conditions and child telomere length, although several retrospective studies have considered whether childhood social conditions are associated with adult telomere length (Adams et al., 2007; Kananen et al., 2010; Surtees et al., 2011). Despite the fact that every child in the sample had at least one parent who completed high school (indicating that the sample is skewed toward higher SES), we were still able to detect a significant association between parental education and child telomere length. This suggests that childhood socioeconomic conditions may have lasting consequences for aging and health throughout the life course. To determine whether this is the case, we will need to conduct prospective studies that include measures of childhood and adult SES and LTL.

Despite its strengths, this study has several limitations that should be addressed in future research. First, since data are from a convenience sample, the results are not generalizable. Moreover, the sample size is very small, which limits statistical power. In addition to examining a larger, more representative sample of children, future studies should also examine longitudinal associations between social status and LTL. Longitudinal studies will enable researchers to determine whether social status is related to the rate of change in cell aging. Finally, because this study used secondary data, we were limited in the choice of mediators. Future studies are necessary to elucidate the mechanisms underlying group differences in LTL and to identify resources that attenuate the association between low social status and cell aging.

Studies examining mediators and moderators of the association between parental SES and child LTL may one day be used to develop interventions to prevent accelerated aging among low SES children. Given the association between shorter LTL and earlier morbidity and mortality in adults, reducing disparities in cell aging among children could lead to a significant reduction in adult health disparities.
References


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