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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.

Introduction

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, Hicken, Keene, & Bound, 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, Blackburn, & Shannon, 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, Boultby, Butler, Thompson, & Goodall, 2001; Zee, Castonguay, Barton, Germer, & Martin, 2010) and mortality (e.g., Bakaysa et al., 2007; Cawthon, Smith, O'Brien, Sivatchenko, & Kerber, 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., 2012; 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, Batty, Der, Fenton, et al., 2012). While several studies have found a positive association between specific indicators of social status and telomere length (Adler et al., 2013; Carroll, Diez-Roux, Adler, & Seeman, 2013; Cherkas et al., 2006; Needham, Fernandez, Lin, Epel, & Blackburn, 2012; Robertson, Batty, Der, Green, et al., 2012; 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.

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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, Fauce, & Effros, 2008; von Zglinicki, Saretzki, Docke, & Lotze, 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, Vaziri, Patterson, & 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, Dagarag, Spaulding, & Man, 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).[1]

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., 2012; 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 et al. (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 et al. (2011) found that renters (vs. home owners) and those with relatively low income experienced a faster rate of cell aging. Similarly, Robertson, Batty, Der, Green, et al. (2012) found that lower parental social class, lower educational attainment, and never owning a home were associated with shorter telomeres; while Carroll et al. (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., 2013; Needham et al., 2012; Steptoe et al., 2011; Surtees et al., 2012).

With a few exceptions (see Adler et al., 2013; 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; and this factor is not measured in most health datasets, including NHANES (Krieger, Williams, & Moss, 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, Zhao, Tsai, & Li, 2011) and the UK (Khaw et al., 2008). Recent studies also suggest that cigarette smoking (McGrath, Wong, Michaud, Hunter, & De Vivo, 2007; Strandberg et al., 2011; Valdes, Andrew, Gardner, & et al., 2005), heavy drinking (Pavanello et al., 2011), sedentary behavior (Du et al., 2012; Ludlow & Roth,

[1] 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).