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Economic hardship and biological weathering: The epigenetics of aging in a U.S. sample of black women

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ABSTRACT

Background: Past research has linked low socio-economic status (SES) to inflammation, metabolic dysregulation, and various chronic and age-related diseases such as type 2 diabetes, coronary heart disease, stroke, and dementia. These studies suggest that the challenges and adversities associated with low SES may result in premature aging and increased risk of morbidity and mortality.
Objective: Building upon this research, the present study investigates various avenues whereby low income might accelerate biological aging.
Methods: Structural equation modeling and longitudinal data from a sample of 100 Black, middle-aged women residing in the United States was used to investigate the effect of income on a recently developed epigenetic measure of biological aging. This measure can be used as a "biological clock" to assess, at any point during adulthood, the extent to which an individual is experiencing accelerated or decelerated biological aging.
Results: Low income displayed a robust association with accelerated aging that was unaffected after controlling for other SES-related factors such as education, marital status, and childhood adversity. Further, our analyses indicated that the association between income and biological aging was not explained by health-related behaviors such as diet, exercise, smoking, alcohol consumption, or having health insurance. Rather, in large measure, it was financial pressure (difficulty paying bills, buying necessities, or meeting daily expenses) that accounted for the association between low income and accelerated aging.
Conclusions: These findings support the view that chronic financial pressures associated with low income exert a weathering effect that results in premature aging.

1. Introduction

In recent years, adverse conditions such as economic hardship, low education, and community disadvantage have been linked to biomarkers of inflammation and metabolic dysregulation, and to various chronic and age-related diseases such as type 2 diabetes, coronary heart disease, stroke, and dementia (Gruenewald et al., 2009; Hemingway et al., 2003; Koster et al., 2006; Loucks et al., 2007, 2010). This body of research suggests that exposure to chronic stress, especially the challenges and adversities associated with low socio-economic status (SES), can foster premature biological aging. More recently, several studies have tested this idea using leukocyte telomere length (LTL) as a measure of unhealthy aging.

LTL has been shown to be a strong marker of aging (Blackburn, 2014; Needham et al., 2013) and numerous investigations have found that, as expected, it is related to factors such as childhood trauma, adult mental health disorders, health-related behaviors,

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and various chronic and age-related diseases. However, most studies report modest associations, and in some cases, studies have reported inconsistent and rather puzzling findings. This includes, for example, several studies that fail to find an association between socioeconomic status and LTL (Carroll et al., 2013; Steptoe et al., 2011), or between age and LTL among black Americans (Needham et al., 2013). Further, there is research indicating that telomere length is longer among black Americans than white Americans of the same age (Needham et al., 2013; Rewak et al., 2014), a paradoxical finding given the high rates of adversity, morbidity, and mortality suffered by blacks compared to other ethnic groups living in the U.S. (Thoits, 2011; Umberson et al., 2014; Williams, 2012).

Such findings indicate that we still have much to learn about telomeres and that research needs to go beyond simply using LTL as an indicator of healthy aging. Toward that end, the present study examines the link between income and premature aging using a recently developed epigenetic measure of biological aging (Hannum et al., 2013). Across several longitudinal samples, this measure has been shown to be highly correlated with chronological age and to be a strong predictor of mortality (Mariano et al., 2015). Further, emerging evidence suggests that it can be used as a biological clock to assess, at any point during adulthood, the extent to which an individual is experiencing accelerated or decelerated biological aging. This instrument is used in the present study to investigate various avenues (e.g., financial pressure, diet, exercise, smoking, access to health care) whereby chronically low income might accelerate biological aging. We tested our models using longitudinal data from a large sample of middle-age Black women, a sample that is particularly relevant for the purposes of our study due to the high rates of poverty, morbidity, and mortality reported among this demographic in the US (Geronimus, 2013; Geronimus et al., 2010; Williams, 2012).

1.1. Potential links between income and accelerated aging

One of the most consistent and well documented associations reported in epidemiological health-focused studies is the inverse relationship between income and rates of morbidity and mortality (Thoits, 2010; Umberson et al., 2014). The link between low income and poor health has been attributed to a variety of factors that may directly or indirectly influence the relationship between income and biological aging. Low income is often chronic, lasting for years or an entire lifetime, and it appears to have deleterious effects on many other domains of everyday life. For example, low income restricts nutrition/dietary choices, participation in exercise or recreational activities, and access to health care. These restrictions, in turn, increase the risk of having an unhealthy weight (i.e., high body mass index [BMI]), and engaging in unhealthy stress-reducing activities such as smoking and heavy alcohol consumption. In addition, individuals with low income are more likely to be single and lack health insurance. Although we acknowledge that all of these factors likely contribute to accelerated aging, we expected that the financial worries and pressures associated with low income would also be powerful predictors of biological aging.

Chronically low income usually entails economic distress resulting from the financial challenges of meeting daily expenses, paying bills, and purchasing necessities. In addition, unanticipated negative events (e.g., automobile repair, job layoff) are more likely to occur and have more serious consequences among lower than higher income individuals. Finally, the vulnerability and insecurity associated with financial hardship often contributes to the development of secondary strains such as marital conflict, sleep disturbances or disorders, and child adjustment problems (Conger et al., 2010). Hence, individuals tend to report that financial hardship is one of the most distressing and debilitating of chronic stressors.

It is now widely posited that repeated and protracted stress contributes to premature aging (Epel et al., 2004; Geronimus et al., 2010), which in large part, could be the result of changes wrought in the immune system. Several studies have established that exposure to adversity causes the immune system to undergo a shift in gene expression; specifically, there is increased expression of pro-inflammatory genes and decreased expression of genes involved in antiviral processes and antibody synthesis (Cole, 2014). Importantly, this shift in gene expression results in chronically elevated levels of inflammation that have been linked to tissue damage, dysregulated metabolic processes, and increased risk for chronic and age-related conditions (Cole, 2014; Maggio et al., 2006). These findings demonstrate that adversity can have biological implications, and support the hypothesis that financial pressure likely accelerates biological aging.

1.2. Epigenetics and aging

There is a growing body of research that has reported associations between epigenetic regulation and age (Weidner and Wagner, 2014). Epigenetic regulation involves biochemical mechanisms that influence genome expression to either up-regulate or down-regulate particular genes. One of the most pervasive and well-studied mechanisms is methylation. This process occurs when a methyl group attaches to a segment of deoxyribonucleic acid (DNA) at a CpG site (i.e., a DNA region where a cytosine nucleotide is positioned next to a guanine nucleotide separated by one phosphate), which causes the inhibition of gene expression. Since the 1960s, researchers have been aware of the strong association between age and DNA methylation (Koch and Wagner, 2011).

Using blood leukocytes, Hannum et al. (2013) recently developed a measure of biological aging based upon the degree of methylation associated with 71 CpG sites scattered throughout the human genome. Methylation changes at these sites were strongly associated with chronological age; however, for some sites methylation increased with age, while it decreased with age at others. Nonetheless, in the sample used to develop the measure, the correlation between age and the weighted sum of methylation scores for all 71 sites exceeded. 90, and subsequent studies using this instrument reported correlations between .82 and .85 (Marioni et al., 2015). Nearly all 71 markers in their model lay within or near genes with known functions associated with age-related conditions, including Alzheimer's disease, cancer, tissue degradation, DNA damage, and oxidative stress (Hannum et al., 2013). Although Hannum et al.'s measure was developed using a White sample, their findings were recently replicated among a sample of Black Americans (Beach et al., in press).

After the age of 20, there appears to be a rather constant rate of methylation change in the 71 sites identified by Hannum and colleagues. Thus, their epigenetic measure can be used as a "biological clock" to assess, at any point during adulthood, the extent to which an individual is experiencing accelerated or decelerated biological aging (Hannum et al., 2013). This can be done by calculating the discrepancy between a person's chronological age and the age predicted using the epigenetic clock. The resulting difference indicates, in number of years, the extent to which an individual is biologically older or younger than their chronological age (i.e., accelerated or decelerated aging). A recent study by Marioni et al. (2015) found that this difference was a strong predictor of mortality across four longitudinal cohorts. Indeed, individuals with a predicted age five years greater than their chronological age showed a 21% increase in mortality risk.

It should be noted that Horvath (2013) has also developed a methylomic measure of aging. It is based on 353 sites and, unlike the Hannum et al. measure which is designed to be used with blood