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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 additional 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.

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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 exerts a weathering effect that results in premature aging.

Keywords

Biological aging; Accelerated aging; Financial pressure; Biological clock; Methylation and aging

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, 2005; Needham et al., 2013) and numerous investigations, as expected, found that LTL is related to factors such as childhood trauma, adult mental health disorders, health-related behaviors, and various chronic and age-related diseases (Shalev et al., 2013). However, most studies report modest associations, and in some cases, studies have reported inconsistent and rather puzzling findings. For example, several studies failed to find an association between LTL and SES (Batty et., 2009; Carroll et al., 2013; Cherkas et al., 2006; Steptoe et al., 2011; Surtees et al., 2011), or LTL and age among Black Americans (Needham et al., 2013), even though other research has shown that Black Americans have longer telomere length than White Americans of the same age (Needham et al., 2013; Rewak et al., 2014). Given the high rates of adversity, morbidity, and mortality suffered by Blacks compared to other ethnic groups living in the United States (US) (Thoits, 2011; Umberson et al., 2014; Williams, 2012), these findings highlight the need for additional research about telomeres, and raise questions about the validity of LTL as a predictor of healthy aging.

Therefore, the present study aimed to examine the link between income and premature aging using a recently developed epigenetic measure of biological aging (Hannum et al., 2013). Several longitudinal samples found this measure to be strongly correlated with chronological age and be a strong predictor of mortality (Mariano et al., 2015). There is emerging evidence that suggests 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. We used this instrument to investigate additional avenues (e.g., financial pressure, diet, exercise, smoking, access to health care) that might mediate how chronically low income accelerates 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).

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., 1992; Conger, Conger, & Martin, 2010). Hence, individuals tend to report that financial hardship is one of the most distressing and debilitating chronic stressors.

It is now widely posited that repeated and protracted stress contributes to premature aging (Epel et al., 2004; Geronimus et al., 2010; McEwen 2012), 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 supports that financial pressure can also accelerate biological aging.

Epigenetics and Aging

There is a growing body of research that has reported associations between epigenetic regulation and age (Fraga & Esteller, 2007; Weidner & 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 (Francis, 2011; Carey, 2012). Since the 1960s, researchers have been aware of the strong association between age and DNA methylation (Koch & 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). Despite the fact that 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). An individual's "biological clock" can be estimated by calculating the discrepancy between their chronological age and the age predicted with 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) also developed a methylomic measure of aging that is based on 353 sites. But unlike the Hannum et al. measure, which is designed to be used with blood assays, the Horvath instrument can be used with any type of biological tissue. Previous studies using blood assays were shown to be more strongly related to age and a better predictor of mortality than the Horvath instrument (Marioni et al., 2015). Further, the Horvath measure has not been validated among African Americans. Indeed, preliminary analyses indicated that only 67 of Horvath's 353 sites were significantly related to age in our sample of Black women. In contrast, all but 5 of the 71 sites identified by Hannum et al. were significantly related to age (see Appendix). Therefore, for the purposes of our study,

the Hannum et al. measure offers a more robust measure of biological aging that utilizes blood leukocytes, especially for our sample of Black women.

The Present Study

Using the biological clock developed by Hannum et al. (2013), the present study investigates additional avenues whereby low income might contribute to accelerated aging. Although an array of health-related behaviors may be important in this respect, we expect that the stress and strain of financial pressure will be an important pathway whereby low income accelerates aging. We test this idea using a sample of middle-aged Black women living in the US. Due to the high unemployment and incarceration rates experienced by Black men (Alexander, 2010; Western & Wildeman, 2009), economic survival of the family is often shouldered by Black women (Geronimus, 2013). Middle age is often a very challenging period for these women as they must contend with the stress of supporting multiple generations of dependents with resources provided by one or more low income jobs (Burton &Whitfield, 2003; Hicks-Bartlett, 2000; Jarrett & Burton, 1999). As a result, they may experience excess biological wear and tear, or as Geronimus (2013) has labeled, *biological weathering*. Geronimus argues that the likely outcome of such stressful circumstances for many Black women is accelerated biological aging (Geronimus et al., 2010), making it a particularly salient group for examining the association between economic hardship and aging.

Based upon this idea, we posit that the low income Black women in our sample will exhibit accelerated aging. Further, we expect that the association between low income and accelerated aging will persist, in large measure, even after controlling for a variety of health-related resources and behaviors such as diet, exercise, smoking, and access to medical care. Finally, we predict that much of the effect that low income has on accelerated aging will be explained (i.e., mediated) by financial pressure (i.e., difficulty paying bills, buying necessities, etc.).

Methods

Sample

We tested our hypotheses using data from Waves 3, 4 and 5 for 100 of the primary caregivers (PCs) in the Family and Community Health Study (FACHS), a longitudinal study of several hundred African American/Black families initiated in 1997. A stratified random sampling procedure was used to intentionally generate a sample of families that represented a range in SES and neighborhood settings. Details regarding FACHS recruitment methods are described by Gibbons and colleagues (2004) and Simons and colleagues (2011). The first Wave of FACHS data were collected in 1997 to 1998 from 889 African American/ Black children and their PCs (829 women and 60 men). At the study's inception, all of the children were in the 5th grade, and about half of the sample resided in Georgia (n = 422) with the other half in Iowa (n = 467). At Wave 1, 36% of the families were below the poverty line, and 51% of the PCs identified as single parents. Data collection for Waves 3, 4, and 5 occurred during 2001 to 2002, 2004 to 2005, and 2007 to 2008 to capture information from the targeted youths at ages 14 to 15, 17 to 18, and 20 to 21 years, respectively. Of the

889 PCs interviewed at Wave 1, 693 were interviewed again at Wave 5 (77.3% of the original sample).

Given that population genetic admixture may confound genetic effects (Halder et al., 2009), we used the Structure program, Version 2.3.4 (Falush, Stephens, & Pritchard, 2007), with a panel of 24 ancestry information markers to gauge the number of ancestral populations in our sample and to estimate an ancestry proportion for each participant. On average, 94.7% of PCs in our sample had African ancestry (Lei et al., 2014). At Wave 5, 100 women were randomly selected from the roster of PCs that were identified as being of African American descent to participate in an epigenetic assessment. Due to the costs associated with the blood draws and epigenetic assays, the use of a subsample was necessary. At Waves 4 and 5, there were no missing values for any of the study variables. At Wave 3, 4% had missing values and the mean imputation method was used for these cases.

Study Procedures

The study protocol and all procedures were approved by the University institutional review board. At Wave 5, computer-assisted interviews were administered in the respondent's home and took on average two hours to complete. Interview questions were presented on laptop computers, which both the researcher and participant could see. The researcher read each question aloud and the participant entered an anonymous response using a separate keypad.

In addition, participants were also asked to provide a blood sample at Wave 5. A certified phlebotomist drew four tubes of blood (30 ml) from each participant; these were shipped on the same day to a laboratory for preparation. Upon receipt, the blood tubes were inspected to ensure anticoagulation and alloquots of blood were diluted 1:1 with phosphate buffered saline (pH 8.0). Mononuclear cell pellets were separated from the diluted blood specimen using a centrifuge with ficoll (400 g, 30 min). The mononuclear cell layer was removed from the tube using a transfer pipette, re-suspended in a phosphate buffered saline solution, and briefly centrifuged again. The resulting cell pellet was re-suspended in a 10% DMSO/RPMI solution and frozen at −8.0 Celsius until use. A typical yield for each pellet was between 10 and 15 μg of DNA.

DNA samples were replicated and included in each plate to aid in assessment of batch variation and to ensure correct handling of specimens. On average, the correlation between plates (using beta [β] values) was greater than .99 for the replicated samples. Prior to normalization, data were inspected for complete bisulfite conversion and cleaned to remove raw β values whose detection *p*-values, an index of the likelihood that the observed sequence represents random noise, were greater than .05. More than 99.76% of the 485,577 probes yielded statistically reliable data. Specifically, data were filtered based on these criteria: (1) samples containing 1% of CpG sites with a detection *p*-value > 0.05 were removed; (2) sites were removed if a beadcount of < 3 was present in 5% of samples; and (3) sites with a detection *p*-value of > 0.05 in 1% of samples were removed.

It should be noted, there is some evidence, primarily from animal studies, of circadian variation in DNA methylation for some genes. Unfortunately, in the present study we do not have information regarding the time of day that blood samples were collected. However,

none of the genes exhibiting circadian, methylomic variation in human studies (Powell & LaSalle, 2015) overlap with the 71 sites included in Hannum et al.'s (2013) measure of aging. Second, it is unlikely that this type of variance would contribute to a positive association between financial variables and biological aging, as noise almost always leads to a Type I error. Therefore, we do not believe that circadian, methylomic variation had a serious impact in the present study. Still, we need to acknowledge that our inability to control for the time of day that blood collection occurred is a limitation of our approach.

The Epigenetic Measure of Biological Age

As previously described, biological age was assessed using the epigenetic clock by Hannum et al. (2013) that is based on the weighted methylation values at 71 CpG sites. Peripheral blood was used to perform methylation analysis. Illumina Human Methylation450 Beadchip (Bibikova et al., 2011) was used to determine the Methylation status for each these loci. We summed the values of the 71 weighted CpG sites (Hannum et al. 2013, Table S3) to form an index of biological age using the following equation:

$$Biological\ age_i = \sum_{j=1}^{71} (CpG_{ij} \times weighting_j)$$

Income Measurement

At Waves 3–5, respondents reported their annual household income from all sources (e.g. wages, interest, business profit, etc.). This variable was measured as an ordinal variable with 16 categories, ranging from 0 (less than $10,000) to 15 ($200,000 or more); total household income was based on the mid-point of these categories. Family per capita income was calculated by dividing the total household income by the number of family members. Scores were averaged across waves to form a measure of per capita income between 2002 and 2008 that indicated the extent to which the respondent had experienced chronically low income over a several-year period.

Financial Pressure

We used a four-item scale developed by Conger et al. (1992) to assess financial hardship at Waves 3–5. The items focused on the extent to which respondents had difficulty paying their monthly bills and were unable to afford the basic necessities of life such as food, clothing, housing, and medical care. Responses for these items ranged from 1 (*strongly disagree*) to 5 (*strongly agree*); scores were averaged across waves. The coefficient alpha was approximately .85 at each wave.

Health-Related Behaviors

The following health-related variables were assessed at Waves 4 and 5, and responses were then averaged across waves. Respondents reported how often during the prior 12 months they had *smoked cigarettes* (0 = never, 5 = everyday) or *consumed more than 3 drinks of alcohol* (0 = never, 5 = every day). *Exercise* was measured with two items: (1) On how many of the past 7 days did you exercise or participate in physical activity for at least 30

min that made you breathe hard such as running or riding a bicycle hard? and (2) On how many of the past 7 days did you exercise or participate in physical activity for at least 30 min that *did not* make you breathe hard, but was still exercise such as fast walking, slow bicycling, skating, pushing a lawn mower, or doing active household chores? Responses for these items ranged from 1 (0 days) to 5 (all 7 days). Responses to these two items were correlated ($r = .419$, $p < .001$), and scores were averaged to form the exercise variable. *Healthy diet* was assessed using two items that asked about frequency of fruit and vegetable consumption during the previous 7 days. Responses ranged from 1 (none) to 5 (twice a day or more). Responses to these two items were correlated ($r = .237$, $p = .016$), and scores were averaged to form the healthy diet variable. The respondents' height and weight were measured by the phlebotomist and used to calculate their BMI (kg/m^2).

Additional Control Variables

In addition to health-related behaviors, we controlled for childhood trauma, education, and marital status. Low family income increases the risk of childhood adversity, which may, according to some studies (e.g., Miller et al. 2011; Umberson, Williams, Thomas, Liu, & Thomeer, 2014), be associated with adult health and specifically accelerated aging (Beach, Lei, Brody, Yu, & Philibert, 2014). We assessed *childhood trauma* at Wave 1 using five retrospective items developed by Kessler (1997). Respondents were asked (1 = yes, 0 = no) whether they experienced stressful events during childhood (e.g., Did your parents divorce or separate permanently before your 17^{th} birthday? While you were growing up, was anyone in your family violent toward another family member?). Scores were summed across the five items to form a measure of childhood trauma. More than half (69%) of respondents reported they had experienced at least one stressful event while growing up. Spearman-Brown coefficient for this scale was .70. Education is strongly related to income, and some studies have also found education to be associated with both morbidity and aging (Adler et al., 2013; Needham et al., 2013; Steptoe et al., 2011). Therefore, we measured *education* by assessing whether the respondent had more than a high school education (1 = yes, 0 = no). Married individuals tend to have higher household incomes and lower rates of morbidity and mortality than those who are single (Thoits, 2011; Umberson & Montez, 2010). Respondents reported their *marital status* as 0 = unmarried, 1 = married.

Statistical Procedures

We conducted power analyses to ensure that our sample was adequate in size to detect statistical significance. According to the G*Power program, a regression model with one dependent variable and two predictors and an N of 100 has 80% power to detect an R^2 effect size of .04, which suggests that our sample size was indeed adequate to test our theoretical models.

Hierarchical regression models were used to investigate the effect of household income and financial pressure on accelerated aging. We used bootstrapping methods with 1,000 replications in Stata 13.0 (StataCorp, 2013) to test the indirect (i.e., mediation) effect of household income on accelerated aging through financial pressure.

Results

Initial Findings

Mean chronological age for the women (n = 100) was 48.5 years (standard deviation [*SD*] = 9.2), 17.8% had less than a 12th grade education, and 24.8% were married. The majority (68.5%) lived in large urban areas, 12.2% lived in the suburbs, and 19.3% lived in rural areas. When we compared this subsample of PCs to those who were not randomly selected for the methylation assessment, there were no significant differences between groups on any independent variables assessed at Wave 1 of the FACHS (household income: t = 1.042; financial pressure: t = 1.231; and chronological age: t = 1.133, *ps* > .05).

Mean biological age, calculated as the weighted sum of the 71 CpG sites identified by Hannum and colleagues (2013), was 49.64 (*SD* = 8.08). As expected, biological age was strongly correlated with chronological age (β = 0.82, p < 1e-25). Indeed, all but five of the 71 CpGs showed a significant association with chronological age in our sample of middle-aged Black women (see Appendix). When we compared the mean biological age of the sample to their chronological age (48.49, *SD* = 9.27), it indicated a slight tendency toward accelerated aging.

We then formulated a measure of accelerated aging using the residual scores from the regression of biological age on chronological age (Hannum et al., 2013; Marioni et al., 2015). These residuals had a mean of zero and represented both positive and negative deviations from chronological age (in years), with positive scores indicating accelerated aging.

Table 1 presents the correlations among the study variables. As expected, there was a strong inverse relationship between per capita income and financial pressure (r = −.431, p < .0001), and these two variables were found to strongly correlated with accelerated aging (r = −.300 and .342, *ps* < .0001, respectively). Associations between accelerated aging and other health-related and control variables were also observed that included health insurance (r = −.208, p < .01) and marital status (r = −.223, p < .01). Education, healthy diet, and tobacco use were related to household income and financial pressure, but their association with accelerated aging did not reach statistical significance.

Regression of Accelerated Aging on Income, Financial Pressure, and Health-related Behaviors

Table 2 presents the results from a series of hierarchical regression models used to determine the effect of household income, financial pressure, and various health-related behaviors on accelerated aging. Model 1 shows that per capita income has a strong effect on biological age (β = −.300, p < .0001). Model 2 reveals that this association is maintained (β = −.276, p < .0001) even after controlling for education, marital status, and childhood trauma. To aid in further interpretation of this finding, we graphed the estimated values of biological aging as a function of household income. As shown in Figure 1, when the predicted regression line and 95% confidence intervals (*CIs*) intersects with the line representing zero deviation (i.e., no difference) of biological from chronological age, the mean household income (and 95% *CI*) suggests that individuals with per capita incomes less

than $3,900 exhibit significant accelerated aging, whereas those with incomes greater than $15,000 exhibit significant decelerated aging. Indeed, approximately 68% of respondents with incomes less than $3,900 showed accelerated aging, and over 70% of individuals with incomes above $15,000 displayed decelerated aging (Figure 2).

Having established an association between household income and biological aging, we then focused our analyses on the various factors that might explain this relationship (Table 2). All of the health-related behaviors thought to mediate the association between income and aging were examined in Model 3. Only health insurance was marginally correlated with accelerated aging, which is consistent with the pattern of associations observed in the correlation matrix (Table 1). Importantly, the association between household income and accelerated aging remained robust even after controlling for a broad array of health-related behaviors ($\beta = -.262$, $p = .016$). Thus, the health-related behaviors listed in Table 2 do not explain the association between income and accelerated aging in our sample of Black women.

The final model in Table 2, Model 4, added financial pressure as a possible predictor, and as expected, financial pressure was significantly associated with accelerated aging ($\beta = .253$, $p < .05$). Furthermore, the relationship between income and accelerated aging was no longer significant once financial pressure was entered into the model. This suggests that financial pressure mediates much of the effect that income has on biological aging. The bootstrapping method with 1,000 replications, revealed a significant indirect effect of income on biological aging through financial pressure (indirect effect = −.306, $p < .05$), and accounted for approximately 25% of the total effect income has on accelerated aging (bottom of Table 2). This finding supports our initial hypothesis, in that the impact of low income on accelerated aging is primarily due to financial pressure.

Discussion

Past research has shown that stress exerts a disruptive effect on biological systems such as the sympathetic nervous system, hypothalamic-pituitary-adrenal (HPA) axis, immune system, and other metabolic processes (Cole, 2014; Deeman et al., 2010; McEwen, 2012). Thus, chronic exposure to stressful situations (i.e., adversity) is likely to result in biological weathering and premature aging (Geronimus, 2013; Geronimus et al., 2010). The present study tested this idea using a recently developed epigenetic measure of aging (Hannum et al., 2013). Hannum's measure focuses on methylation changes at 71 CpG sites that are strongly associated with chronological age. This instrument may be viewed as a biological clock, given that it can be used to assess whether individuals are aging biologically at a pace that is faster or slower than their chronological age. Recent research indicates that accelerated aging, as assessed with this measure, is a strong predictor of mortality (Marioni et al., 2013).

The current study tested two hypotheses. First, we predicted that chronic low income would be associated with accelerated aging, even after controlling for various health-related behaviors. Second, we posited that financial pressure would mediate much of the relation between low income and accelerated aging. Our sample of middle-aged, Black women was

particularly appropriate for testing these hypotheses given the high rates of poverty and poor health suffered by this group in the US. Indeed, there is evidence to support that mortality rates among Black women worsened after 1990 (Kindig and Cheng, 2013), and the most prominent differences in health between Black and White women occur in middle age (Geronimus et al., 2010).

The results from this study provided strong support for our hypotheses. As expected, we observed a robust association between income and accelerated aging that was unaffected by controlling for SES-related factors (i.e., education, marital status, and childhood adversity) that have been previously linked to biomarkers of health. When examined further, we found that 68% of women in our sample with per capita incomes less than $3,900 exhibited accelerated aging, whereas 70% of those with per capita incomes above $15,000 experienced decelerated aging.

Given this association, we examined the extent to which various health-related behaviors such as diet, exercise, smoking, alcohol consumption, and having health insurance could explain the effect of income on aging. Our analyses revealed no significant relationship between these variables and the speed to which aging occurred, and controlling for them had no impact on the association between income and biological aging. Although we expected these health-related behaviors to have a modest influence on aging, we were surprised to find no significant influence. This lack of association may be partially attributed to the limited nature of the measures we used to assess these variables. It could also be the case that few of the women in our sample engaged in diet and exercise habits sufficient to impact health and aging. Existing research suggests that individuals must make rather dramatic changes in their lifestyle in order to prevent or reverse the course of chronic disease(s) (Devries et al., 2014; Ornish et al., 2013). In our sample, only seven women reported exercising to the point of heavy breathing at least five times a week, and only 10 women reported eating fruits and vegetables at least eight times in the past week. Furthermore, virtually all of the women in the sample had a BMI well above the threshold for being overweight (>25 kg/m^2). Thus, it may appear that these variables had little effect on biological aging in the present sample; however, it is more likely that our sample lacked the variation and range required to detect any effect. Of note, Mariano et al. (2015) also found that health-related behaviors assessed in the four longitudinal studies were not associated with Hannum's measure of biological aging.

Although health-related behaviors had virtually no impact on biological aging, our analyses indicated that financial pressure had a strong effect on accelerated aging, and it mediated the influence low income had on aging. These findings are in support of existing research showing that prolonged exposure to chronic financial pressure exerts weathering effect that results in premature aging (Geronamus, 2010, 2013), and are particularly salient given the current income distribution within the US. Compared to other wealthy nations of the world, the US has long had a much greater, unequal income distribution (Wilkinson and Pickett, 2009), and this inequality has been amplified in recent years. Approximately 20% of the US population currently lives at or near the poverty line, defined as a household income of $23,000 for a family of four (U.S. Census Bureau, 2014). Our results suggest that these

individuals are at risk for premature aging and the morbidity and mortality risks that this portends.

Of course, Black Americans are more likely than other ethnic groups to suffer from low income and financial pressure (Massey, 2007). Almost one-third of Black Americans currently live near or below the US poverty line (U.S. Census, 2014), and this situation is even more direr for women-run households. In 2013, 42% of households headed by Black women were in poverty (U.S. Census, 2014). The financial pressure experienced by these households has undoubtedly been magnified in recent years by dramatic budget cuts in government programs such as food-stamps. When considering the current US income statistics in combination with our findings that link financial pressure and accelerated aging, it is not surprising that the average life expectancy in the US is shorter than virtually all other wealthy nations in the world (Murray et al., 2013; Wang et al., 2012). Within the US, the average life expectancy among Blacks is much shorter than other ethnic groups (Lantz et al., 1998; Williams, 2012).

Limitations

Although we cannot think of any reason to believe that our findings are specific to Black women only, our results need to be replicated with larger and more diverse samples that involve both men and women, and other ethnic groups. Second, our epigenetic measure of aging was obtained at a single point in time. Future research should assess biological aging and environmental conditions at multiple time-points so that changes in the environment can be examined in relation to changes in aging speed. Such an approach would provide more compelling evidence for the role of the environment in accelerating or decelerating biological aging.

Future Research

In addition to addressing these limitations, we recommend that future research expand its focus to include a wider array of stressors than those investigated in the current study. Our analyses indicated that financial pressure explains much of the impact low income has on accelerated aging; yet, it would be interesting to see if other strains associated with low income, such as unemployment or living in a disadvantaged neighborhood, also influence aging. Finally, future studies should also investigate the extent to which social resources (i.e., supportive partner) and psychological traits (i.e., optimism and religiosity) serve to decelerate aging, or provide a buffering effect against stressors such as low income and financial pressure. Our findings indicate that these factors should receive similar attention as determinants of biological aging to health-related behaviors (diet, exercise), which are the primary targets of most treatment and policy interventions.

At this point, Hannum's (2013) epigenetic measure of aging has little direct clinical utility. The expense of methylation analyses would prevent most clinicians from employing it as a diagnostic tool. Rather, its importance is as a research instrument that can be used to detect the individual variability in the speed of aging, with the goal of linking this variability to lifestyle and environmental factors. These outcomes would be helpful in identifying all possible targets for health promotion policies and programs, and, as in the case of the current

study, they may help our society recognize that the underlying causes of premature aging and chronic diseases are often inextricably tied to broader social forces, which also need to be addressed if we are to improve the health of certain subgroups of the population.

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Research Highlights

- We investigated the effect of financial hardship on healthy aging among a large sample of Black women.
- Aging was assessed using an epigenetic measure that predicts chronological age and mortality.
- Low income predicted accelerated aging whereas high income predicted decelerated aging.
- The effect of low income on aging was mediated by financial pressures (unmet needs, unpaid bills).
- These findings suggest that chronic financial strain exerts a weathering effect that results in premature aging.

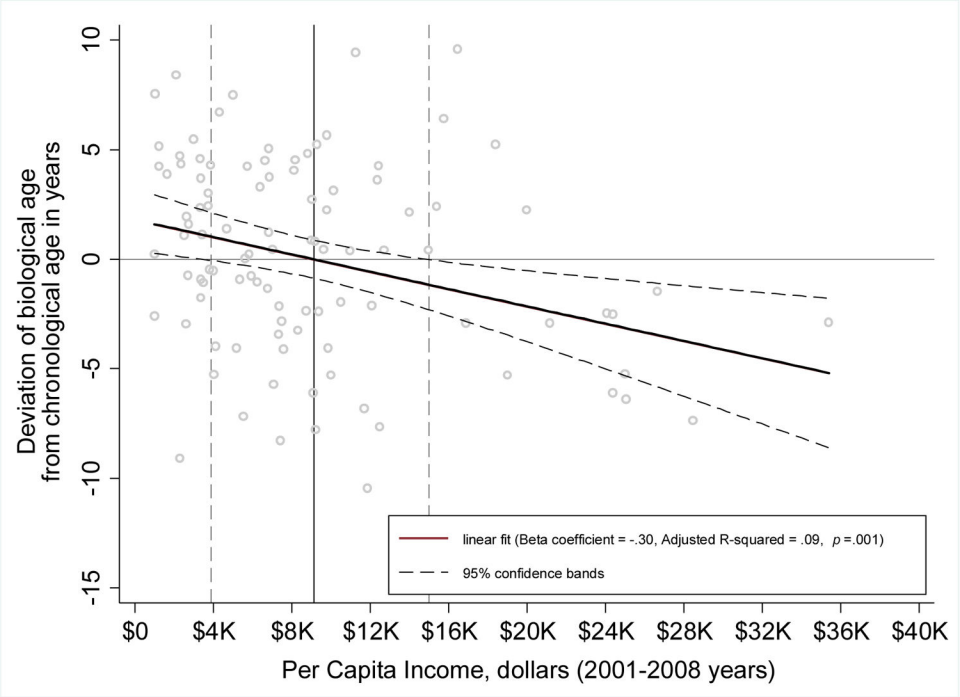

Figure 1. Scatter plot representing the association between per capita income (waves 3, 4 and 5) and biological aging using Hannum's weights

The solid line displays the predicted regression line, and the dashed lines are the 95% confidence bands for the fitted line. Predicted scores represent residual biological age after controlling for chronological age.

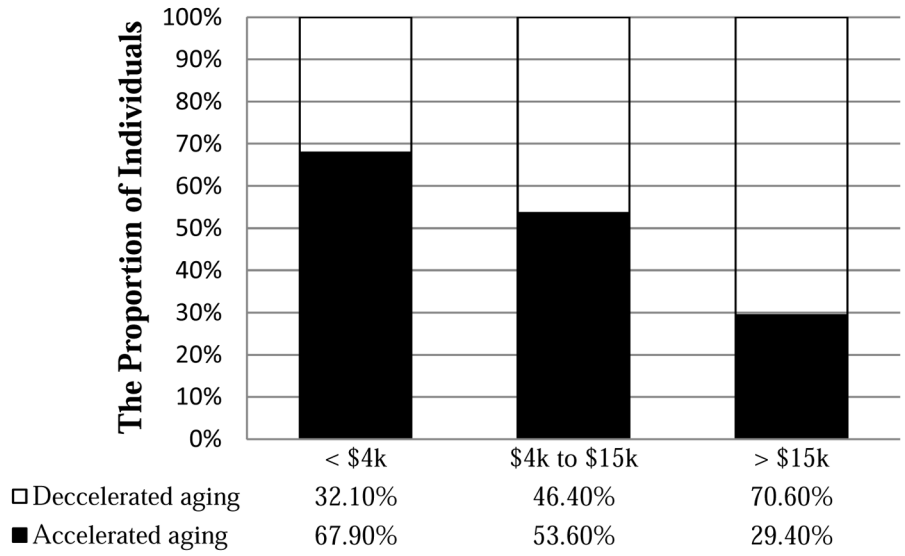

Figure 2.

The proportion of respondents displaying accelerated versus decelerated aging at various levels of per capita income (N = 100).

Table 1

Correlation matrix of the study variables (N = 100).

Variables [a]	1	2	3	4	5	6	7	8	9	10	11	12
1. Accelerated biological aging	——											
2. Per capita income	−.300**	——										
3. Financial pressure	.342**	−.431**	——									
4. Education (≥High school)	−.087	.333**	−.375**	——								
5. Childhood trauma	−.053	−.075	.083	−.118	——							
6. Married	−.223 *	.236*	−.336**	.207 *	−.005	——						
7. Tobacco use	.100	−.006	.171†	.157	.035	−.283	——					
8. Alcohol use	−.076	.131	−.023	.104	−.003	−.086	.361**	——				
9. Healthy diet	−.016	.164	−.200*	−.023	−.092	.102	−.017	−.054	——			
10. Exercise	−.021	−.002	−.069	.110	−.087	.019	−.027	−.074	.249*	——		
11. Body Mass Index (kg/m^2)	−.101	−.049	.089	−.187 †	.022	.095	−.241*	−.075	−.049	.089	——	
12. Health insurance	−.208 *	.101	−.220*	.113	−.074	.164	−.217*	−.197 *	−.046	−.155	.009	——
Mean	.000	9129.405	.000	.822	1.812	.248	.446	.327	2.040	1.302	33.475	.861
Standard Deviation	4.607	6987.795	1.000	.385	1.405	.434	.500	.471	.905	1.166	7.737	.347

Note.

[a] All study variables were assessed at Wave 5, with the exception of: Per capita income and Financial pressure (Waves 3, 4, 5); Childhood trauma (Wave 1),

† p<.10;

* $p \leq .05$;

** $p \leq .01$ (two-tailed tests).

Table 2

Regression models examining household income, financial pressure, and health behaviors as predictors of residual change scores between Hannum's biological age and chronological age (N = 100).

	Accelerated Aging							
	Model 1		Model 2		Model 3		Model 4	
Predictors[a]	***b***	**β**	***b***	**β**	***b***	**β**	***b***	**β**
Intercept	0.000 (0.439) **		0.553 (1.286)		4.592 (3.194)		3.614 (3.163)	
Per capita income (W3,4,5)	−1.380 (0.442) **	−.300	−1.273 (0.476) **	−.276	−1.206 (0.491) *	−.262	−0.900 (0.502) †	−.195
Financial pressure (W3,4,5)							1.167 (0.539) *	.253
Education (≥HS) (W5)			0.365 (1.235)	.031	0.458 (1.324)	.038	1.212 (1.343)	.101
Childhood trauma (W1)			−0.232 (0.316)	−.071	−0.282 (0.321)	−.086	−0.282 (0.314)	−.086
Married (W5)			−1.747 (1.057)	−.164	−1.397 (1.131)	−.132	−0.997 (1.124)	−.094
Tobacco use (W5)					0.349 (1.038)	.038	−0.071 (1.036)	−.008
Alcohol use (W5)					−1.164 (1.035)	−.119	−0.990 (1.018)	−.101
Healthy diet (W5)					0.164 (0.523)	.032	0.348 (0.519)	.068
Exercise (W5)					−0.274 (0.408)	−.069	−0.243 (0.400)	−.061
Body Mass Index (W5)					−0.049 (0.060)	−.083	−0.061 (0.059)	−.103
Health insurance (W5)					−2.574 (1.369) †	−.194	−2.144 (1.357)	−.162
R^2	**.090**		**.121**		**.172**		**.213**	
Indirect effect [b]							**−.306* (−.774, −.052)**	
Direct effect [b]							**−.900 (−1.768, .096)**	
The total effect mediated by financial pressure							**25.373%**	

Note. Unstandardized (b) and standardized coefficients (β) are shown with standard errors in parentheses. HS = High school. W1, 3, 4, 5 = Waves 1, 3, 4, and 5; represents the study wave(s) the variable was assessed.

[a] Per capita income and financial pressure are standardized (z-transformation: mean = 0 and standard deviation = 1).

[b] Indirect and direct effects are shown with their 95% confidence intervals.

** $p \leq .01$;

* $p \leq .05$,

† $p < .10$ (two-tailed tests)

Appendix

Correlations between chronological age and methylation for each of the 71 CpG sites identified by Hannum et al.'s (2013) epigenetic measure of aging.

	Chronological Age	
Y = Aging-elated markers	**β**	***p*-value**
cg16867657	.788	.000
cg06639320	.623	.000
cg22454769	.643	.000
cg24079702	.493	.000
cg07553761	.309	.002
cg04875128	.512	.000
cg14692377	.210	.035
cg22736354	.554	.000
cg07547549	.523	.000
cg02650266	.479	.000
cg23500537	.294	.003
cg03032497	.299	.002
cg08097417	.672	.000
cg14361627	.644	.000
cg16419235	.469	.000
cg22285878	.169	.091
cg03607117	.339	.001
cg06493994	.488	.000
cg04400972	.374	.000
cg23091758	.381	.000
cg07955995	.541	.000
cg22158769	.496	.000
cg20426994	.410	.000
cg14556683	.501	.000
cg00748589	.480	.000
cg21296230	.525	.000
cg07927379	.284	.004
cg25410668	.430	.000
cg22213242	.282	.004
cg23606718	.534	.000
cg03399905	.513	.000
cg25478614	.509	.000
cg06419846	.229	.021
cg00481951	.553	.000
cg08540945	.252	.011
cg18473521	.497	.000
cg11067179	.181	.070

	Chronological Age	
Y = Aging-elated markers	**β**	***p*-value**
cg04940570	.529	.000
cg21139312	.329	.001
cg19935065	.251	.011
ch1339564907R	−.398	.000
cg09651136	−.067	.508
ch230415474F	−.324	.001
cg13001142	−.277	.005
cg05442902	−.355	.000
cg02867102	−.308	.002
cg00486113	−.330	.001
cg20052760	−.247	.013
cg19722847	−.363	.000
cg06874016	−.378	.000
cg02046143	−.192	.055
cg25428494	−.262	.008
cg04474832	−.195	.050
cg02085953	−.337	.001
cg04416734	−.325	.001
cg22512670	−.123	.221
cg06685111	−.298	.002
cg03473532	−.537	.000
cg22016779	−.484	.000
cg20822990	−.435	.000
cg08415592	−.159	.113
cg07583137	−.409	.000
cg09809672	−.413	.000
cg01528542	−.458	.000
cg07082267	−.312	.001
cg22796704	−.218	.029
cg23744638	−.323	.001
cg16054275	−.197	.048
cg08234504	−.314	.001
cg19283806	−.504	.000
cg10501210	−.481	.000

Note. Number of significant = 66 (92.95%). β=Beta.