The Great Recession and health risks in African American youth

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Abstract

In the present study, we investigated associations of macro-economic conditions – the Great Recession – with cellular epigenetic aging, allostatic load, and self-reported health, in a group that experiences significant health disparities, African Americans. A sample of 330 African American adolescents in Georgia was followed from pre-recession (2007, M age = 16.6) to post-recession (2010, M age = 19.3). Economic data were collected in both 2007 and 2010. Three groups were formed to represent economic trajectories across the period of the Great Recession (stable low economic hardship, downward mobility, and stable high economic hardship). At age 19, measures of cellular epigenetic aging (derived from leukocyte DNA methylation profiles, reflecting the disparity between a person’s biological and chronological age), allostatic load (composite of blood pressure, C reactive protein, cortisol, epinephrine, norepinephrine, and body mass index), and adolescent self-report of health were obtained. Linear trend analyses documented significant differences across all outcomes. The more time adolescents spent under economic hardship, the higher their epigenetic aging [estimate = 1.421, SE = 0.466, \( p = .002 \)] and allostatic load [estimate = 1.151, SE = 0.375, \( p = .002 \)] scores, and the worse their self-report of health [estimate = 4.937, SE = 1.800, \( p = .006 \)]. Specific group comparisons revealed that adolescents in the downward mobility group had higher levels of allostatic load than adolescents in the stable low hardship group \( (p < .05) \). Overall, these findings suggest that the health profiles of African American youth may in part be shaped by environmental macro-economic societal conditions, and that effects on biological markers can be detected relatively early in life.

Introduction

Economic hardship has far-reaching effects on a number of life outcomes, including physical health (Adler and Rehkopf, 2008; Fiscella and Franks, 1997; McDonough et al., 2005). In fact, health in the U.S. is widely acknowledged to be unequally distributed by both poverty and race (Adler et al., 1993; Williams et al., 2010; Smedley et al., 2003), meaning that those who live in poverty or those who are racial/ethnic minorities experience worse health outcomes in many domains compared with those who are not. These individuals are more likely to die at younger ages, to experience higher rates of numerous chronic diseases, and to suffer a greater burden from these diseases (Williams et al., 2010; Adler and Rehkopf, 2008). Health disparities by income and race are present even in childhood, and persist over the lifecourse (Chen et al., 2002; Adler and Stewart, 2010). While this literature is extensive, the vast majority of studies on this topic documents epidemiologic associations of a family’s poverty level with health outcomes that are often self-reported. In contrast, macro-economic events can present an opportunity to investigate how an exogenously occurring societal-level event may be linked to a multitude of biological and health measures, particularly among groups (e.g., African Americans) that both are disproportionately at risk for disease and are often hardest hit by these events.

The Great Recession from December 2007 to June 2009 was the worst economic period in U.S. history since the Great Depression of 1929. 8.7 million jobs were lost during this period (Greenstone and Looney, 2013), as the national unemployment rate almost doubled from 5.0% to 9.5% (Statistics, 2012). Housing foreclosure rates quadrupled during the recession, with over 20% of homeowners finding themselves with mortgages that exceeded the value of their house (Gould Ellen and Dastrup, 2012). Over 40% of working Americans reported having to take a cut in pay or reduction in hours during the recession (Taylor et al., 2010). Moreover, African American and Hispanic families were hardest hit by this recession (Kochhar and Fry, 2014; Gould Ellen and Dastrup, 2012).

In this study, we took advantage of the Great Recession as an externally occurring macro-economic event that occurred in the...
midst of an ongoing, longitudinal study of African American youth that lasted from 2001 to 2010. Families’ economic circumstances were assessed repeatedly, allowing us to prospectively track which families experienced economic decline during the recession and which families did not.

To investigate the biological mechanisms potentially associated with macro-economic conditions, we focused on an epigenetic measure of aging in cells of the immune system (peripheral blood mononuclear cells, PBMCs). Epigenetics refers to modifications in DNA activity that do not involve changes to DNA sequence. The best studied epigenetic modification is DNA methylation, a process whereby methyl groups bind to cytosine residues that comprise DNA, and in doing so alter the cell’s ability to switch on particular genes. In the immune system, methylation is thought to be a dynamic process, helping cells prepare for and adapt to changing environmental demands. Across the lifespan, patterns of methylation change in ways that are relatively consistent across individuals (Horvath, 2013; Jones et al., 2015; Marioni et al., 2015). Based on these patterns, researchers have constructed methylation-based profiles of cellular aging.

The concept of cellular age is closely related to, but not isomorphic with chronological age. Indeed, research shows that some individuals show more rapid cellular aging than would be expected on the basis of their chronological age, whereas others show the reverse. Most of the research to date on cellular aging has focused on telomere biology (Epel et al., 2004; Blackburn and Epel, 2012; Shalev et al., 2013). But there is mounting evidence to suggest that methylation-based approaches provide some unique insights about cellular aging and health outcomes, and do so in a way that circumvents the methodological challenges of cellular heterogeneity in human blood (Horvath, 2013; Horvath et al., 2014). Indeed, faster epigenetic aging has been documented in tumor-derived cells from over 20 cancers, as well as in liver biopsies from obese patients (Horvath, 2013; Horvath et al., 2014). Epigenetic aging has also been studied in PBMCs: children with more “aged” cells show higher blood pressure (Simpkin et al., 2016), and adults with more “aged” cells show higher rates of all-cause mortality in longitudinal cohort studies (Marioni et al., 2015). Nevertheless, PBMC epigenetic aging is a relatively new metric, and confidence about its value would be enhanced if results converged with better established indicators of disease risk and health status. Thus, in this study we also measured allostatic load, a composite reflecting blood pressure, adiposity, stress hormones, and inflammation, and obtained self-reports of health. Using these outcomes, we examined the health profiles of African-American adolescents as a function of their families’ economic trajectories during the period of the Great Recession.

2. Materials and methods

2.1. Participants

The data for this study were drawn from the Strong African American Families Healthy Adult Panel (SHAPE) study. African American caregivers and one youth from each family participated, beginning when youth were in 5th grade ($M$ age = 11.2 years, $SD$ = 0.34; range from 11 to 13) in 2001. 53% were female. Families resided in nine rural counties in Georgia, in communities in which poverty rates are among the highest in the nation and unemployment rates are above the national average (Proctor and Dalaker, 2003). 667 families were selected randomly from lists of fifth-grade students from school directories, with permission from school superintendents (see Brody et al., 2004, for a full description). At age 18, 500 families were randomly selected to participate in biological data collection (necessary because of budgetary constraints). Of the 500 participants, economic hardship data in both 2007 and 2010 were available on 431, and urine and blood samples were obtained from 379 participants. Among the 500, there were no differences between those with and without economic hardship data, or between those with and without blood and urine data on demographic or study variables. However, when comparing demographic variables at the start of the study (2001), there were some differences between those included in the analyses below, and those who were not included in terms of parent education and parent marital status. See Online Supplemental Tables 1–3 for details. A total of 330 families both provided blood samples and had economic hardship data in 2007 and 2010 and constituted the sample for this study. See Online Fig. 1 for a diagram of participant flow through the longitudinal study.

Adolescents’ mean age was 16.6 ($SD$ = 0.53) in 2007 and 19.3 ($SD$ = 0.67) in 2010. Economically, these families are best characterized as working poor. Median household income was $1804/month, with 45.8% living below federal poverty thresholds. See Table 1.

2.2. Procedure

Data on demographic and socioeconomic characteristics were collected in participants’ homes using a standardized protocol in 2007 and 2010. Health and biological measures were only available in 2010. The original sample was randomized into a parenting intervention in 2001 (Brody et al., 2004), and hence intervention status was controlled in all analyses below. Caregivers provided consent, youth provided assent, and the university institutional review board approved study protocols. Families were paid $100 per visit.

2.3. Measures

2.3.1. Family economic hardship

Family economic hardship was comprised of 3 objective and 3 subjective indicators collected in 2007 and again in 2010. Previous research has shown that the construct of economic hardship is best captured by both objective and subjective indicators (Conger and Elder, 1994; Sobolewski and Amato, 2005). For all indicators, scores of 1 were given if the family met the definition for hardship (as described in detail below), otherwise a 0 was given.

For the objective indicators, a score of 1 was assigned to each of the following characteristics that were endorsed: family poverty

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Low hardship</th>
<th>Downward mobility</th>
<th>High hardship</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adolescent age (in years)</strong></td>
<td>16.54 (0.54)</td>
<td>16.59 (0.51)</td>
<td>16.65 (0.50)</td>
</tr>
<tr>
<td><strong>Parent age (in years)</strong></td>
<td>42.14 (6.06)</td>
<td>43.56 (8.59)</td>
<td>43.61 (7.48)</td>
</tr>
<tr>
<td><strong>Adolescent gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>46.7%</td>
<td>45.5%</td>
<td>46.9%</td>
</tr>
<tr>
<td>Female</td>
<td>53.3%</td>
<td>54.5%</td>
<td>53.1%</td>
</tr>
<tr>
<td><strong>Parent education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;High school</td>
<td>13.0%</td>
<td>19.8%</td>
<td>38.8%</td>
</tr>
<tr>
<td>High school degree or GED</td>
<td>22.3%</td>
<td>36.6%</td>
<td>36.7%</td>
</tr>
<tr>
<td>Some college</td>
<td>53.3%</td>
<td>40.5%</td>
<td>21.4%</td>
</tr>
<tr>
<td>&gt; College graduate</td>
<td>11.4%</td>
<td>3.1%</td>
<td>3.1%</td>
</tr>
<tr>
<td><strong>Parent marital status</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married or partnered</td>
<td>44.8%</td>
<td>32.6%</td>
<td>18.4%</td>
</tr>
<tr>
<td>Single</td>
<td>55.2%</td>
<td>67.4%</td>
<td>81.6%</td>
</tr>
</tbody>
</table>
(Income-to-needs ratios were calculated as family income divided by the poverty threshold for that family size based on US Census Bureau guidelines. Income-to-needs ratios <1 were considered below poverty and assigned a score of 1); unemployment of the primary caregiver; and receipt of Temporary Assistance for Needy Families (TANF).

For the subjective indicators, adequacy of income was measured by a single item asking how adequate the primary caregiver felt his/her income was in meeting their needs, from 1 (much less adequate) to 3 (adequate to meet our needs) and to 5 (much more than adequate to meet our needs). A score of 1 was assigned to ratings lower than 3.

For unmet material needs, the 4-item unmet material needs scale was completed by caregivers (Conger and Elder, 1994). Caregivers indicated how much they agreed or disagreed with statements regarding their family’s financial situation, from 1 (strongly agree) to 4 (strongly disagree). Items included “My family has enough money to afford the kind of home we need.” Cronbach’s alphas were .89 in 2007 and .85 in 2010. The 4 items were summed, with higher scores indicating greater unmet needs. Scores ranged from 4 to 16 (M = 9.14, SD = 2.84 in 2007; M = 10.43, SD = 2.70 in 2010). A score of 1 was assigned to scores higher than 10, which was the mean score across two time points.

For inability to make ends meet, the 2-item cannot make ends meet scale was completed by caregivers (Conger and Elder, 1994). Caregivers were asked, for example, about how much difficulty they had during the past 12 months paying the bills, on a scale ranging from 1 (no difficulty at all) to 5 (a great deal of difficulty). Cronbach’s alphas were .89 in 2007 and .70 in 2010. The 2 items were summed, with higher scores indicating greater difficulty. Scores ranged from 2 to 10 (M = 5.15, SD = 2.23 in 2007; M = 6.31, SD = 2.08 in 2010). A score of 1 was assigned to scores higher than 6, which was the mean of the scale across two time points.

The 3 objective and 3 subjective dichotomous measures were then summed to form an index of family economic hardship in 2007 and in 2010. Scores ranged from 0 to 6 (M = 1.68, SD = 1.47 in 2007; M = 2.72, SD = 1.71 in 2010), and we defined high vs. low economic hardship at each time point using a median split (≥3 vs. <3, same split used at each time point). The dichotomization and sum score approach is consistent with how we have测量ed family economic hardship in previous studies (Chen and Brody, 2015; Brody et al., 2013).

In ancillary analyses, we also tested the interaction of the two economic hardship indicators as continuous variables. However, we note that the hardship variables at the two time points were highly correlated (r = .55, p < .001), which can result in multicollinearity issues with analyses. Please see Online Supplement Tables 4–8.

2.3.2. Epigenetic aging

In 2010, blood samples were collected for DNA methylation profiling. PBMCs were isolated through density-gradient centrifugation (Ficoll-Paque Media PM 400). Genomic DNA was extracted with Qiagen DNA Mini Kits, and quality verified on an Agilent 2100 Bioanalyzer. DNA was then subjected to bisulfite conversion and global amplification (Infinium DNA Methylation Kit; Illumina, San Diego, CA). Methylation profiling was conducted by the University of Minnesota’s Genome Center, following manufacturer’s protocol for the Illumina Human Methylation 450 Beadchip. This chip contains 485,577 probes recognizing at least 20,216 transcripts, potential transcripts, or CpG islands. Average beta values for each targeted CpG residue was determined using the Illumina Genome Studio Methylation Module, Version 3.2. Beta values for each probe were calculated as the ratio of methylated probes to the sum of methylated and unmethylated probes, ranging from 0 = entirely unmethylated to 1 = fully methylated.

Two epigenetic aging metrics have been proposed in the literature, which use distinct targets, covariates, and formulas. We calculated both here. Horvath’s clock aggregates normalized methylation values from 353 CpG sites (Horvath, 2013). Hannum’s clock sums weighted methylation values from 71 CpG sites, using coefficients validated for PBMC (Hannum et al., 2013). The values derived from these metrics are residuals, that is, a continuous measure that reflects the discrepancy between an individual’s estimated cellular age and chronological age. Positive values reflect a relatively faster aging of cells. Faster epigenetic aging rates have been documented in tumor-derived cells, as well as in liver biopsies from obese patients (Horvath, 2013; Horvath et al., 2014), and predicted higher risk for all-cause mortality in four large cohorts (Marioni et al., 2015).

2.3.3. Allostatic load

The protocol for measuring allostatic load was based on procedures developed for field studies involving children and adolescents (Evans, 2003). Resting blood pressure was monitored with a Critikon Dinamap Pro 100. Three readings were taken every 2 min, and the average of the last two readings was used as the resting index (Kamarck et al., 1992). Overnight urinary catecholamines and cortisol were assayed. Total unbound cortisol was assayed with a radioimmune assay (Contreras et al., 1986). Epinephrine and norepinephrine were assayed with high-pressure liquid chromatography with electrochemical detection (Riggin and Kissinger, 1977). Creatinine was assayed to control for differences in body size and incomplete urine voiding (Tietz, 1976). C reactive protein (CRP), a marker of low-grade inflammation and later allostatic load, was assayed in serum using a Duoset Kit (DY1707; R&D Systems). Because CRP was characterized by a skewed distribution, we applied a log transformation to normalize the values (Howell, 2007; Tabachnick and Fidell, 2007).

The allostatic load composite was calculated by summing the standardized scores of seven indicators: cortisol; epinephrine; norepinephrine; diastolic blood pressure; systolic blood pressure; CRP; and body mass index. Prior studies in adults (Karlamangla et al., 2006), children (Evans, 2003), and adolescents (Brody et al., 2013) used similar metrics, combining multiple physiological indicators of risk into a composite.

2.3.4. Self-report of health

In 2010, adolescents reported on their physical health using the General Health Perceptions subscale from the RAND 36-Item Short-Form Health Survey (Hays et al., 1993). This is a continuous measure (range 0–100), in which higher scores indicate poorer health (relative to those with lower scores). Self-report ratings of health predict overall mortality rates (Idler and Benyamini, 1997).

2.3.5. Statistical analyses

All analyses controlled for standard socio-demographic variables that were collected, including adolescent gender (race was not controlled because all were African American, and age was not controlled because all were in the same grade when recruited), parent age, parent education, and parent marital status (parent age and marital status are controlled given that there were differences between those with and without data on these variables, see Online Supplement). In addition, given the design of the original study, intervention status (intervention vs. control group) was also controlled. We first conducted orthogonal polynomial contrasts. The polynomial contrasts test whether means increase across the three groups (stable low hardship, downward mobility, stable high hardship) in a linear way (linear comparison) or whether the pattern of means is curvilinear (quadratic comparison). Second,
univariate between-group analyses of variance (ANOVA) were used to test specific comparisons across the three groups. We note that our outcome measures reflect continuous distributions, and hence that we are comparing scores in one group to another, rather than drawing conclusions about ‘poor health’ or ‘accelerated aging’ in any absolute sense, particularly given the young and healthy sample.

3. Results

3.1. Family economic hardship groups

In 2007, 28.5% (n = 94) of families experienced high economic hardship. In 2010, 56.7% (n = 187) experienced high economic hardship. 39.7% (n = 131) of families were experiencing low economic hardship in both 2007 and 2010 (stable low hardship group). 24.8% (n = 82) were experiencing high economic hardship in both 2007 and 2010 (stable high hardship group). A small number (n = 12, 3.6%) experienced upward mobility (high economic hardship in 2007, low economic hardship in 2010). Because the sample size of 12 was deemed inadequate for meaningful follow-up analyses, these cases were dropped from further analyses.

Table 1 presents sample characteristics. The three economic groups described above did not differ on the demographic variables of adolescent age, adolescent gender, or parent age (all p’s > .2). As expected, at baseline (pre-recession, in 2007), the stable high hardship group had lower levels of parent education and were more likely to be single parents compared to the stable low hardship and the downward mobility (who started off as low hardship but moved to high hardship) groups (all p’s < .001). The stable low hardship and downward mobility groups did not differ from each other on parent education or marital status at baseline.

Table 2 presents mean values for the economic hardship indicators by group.

3.2. Differences among economic hardship groups in adolescent outcomes in 2010

Using the polynomial contrasts, significant linear effects were found for all outcomes, meaning that as the amount of time spent under economic hardship increased, the higher their epigenetic aging and allostatic load scores, and the worse their self-reported health; linear contrast estimate = 1.431, SE = 0.558, p = .011 for epigenetic aging using Hannum’s method; estimate = 1.421, SE = 0.466, p = .002 for epigenetic aging using Horvath’s method; estimate = 1.151, SE = 0.375, p = .002 for allostatic load; estimate = 4.957, SE = 1.800, p = .006 for self-report of health. No quadratic effects were found. See Table 3.

ANOVA revealed that for epigenetic aging scores using the Horvath method, a significant omnibus difference across groups was detected, F(2,310) = 4.88, p = .008. Post-hoc comparisons revealed that adolescents in the stable high hardship group exhibited greater epigenetic aging values relative to adolescents in the stable low hardship group (Cohen’s d = .44, p < .05) and adolescents in the downward mobility group (Cohen’s d = .35, p < .05). Using the Hannum method, a significant omnibus difference across groups was also detected, F(2,310) = 3.29, p = .039. Post-hoc comparisons revealed that adolescents in the stable high hardship group exhibited greater epigenetic aging values relative to adolescents in the stable low hardship group (Cohen’s d = .37, p < .05), with adolescents in the downward mobility group falling in the middle and not differing from the two other groups. See Table 4 and Fig. 1.

With respect to allostatic load, a significant difference across groups was also detected, F(2,310) = 5.78, p = .003. Post-hoc LSD comparisons revealed that adolescents in the stable high hardship group and the downward mobility group evinced higher allostatic load scores compared to adolescents in the stable low hardship group (all p’s < .05); effect size (Cohen’s d) = .45 and .36, respectively. See Table 4 and Fig. 1.

With respect to self-report of health, a significant difference across groups was also detected, F(2,310) = 5.16, p = .006. Post-hoc comparisons revealed that adolescents in the stable high hardship group reported poorer overall health compared to adolescents in the stable low hardship group (Cohen’s d = .40, p < .05) and downward mobility groups (Cohen’s d = .43, p < .05). See Table 4 and Fig. 1.

Table 3 Polynomial contrasts for means differences in adolescents’ health outcomes by family economic hardship groups.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Polynomial contrasts Linear</th>
<th>Estimation</th>
<th>SE</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allostatic load</td>
<td></td>
<td>1.151</td>
<td>0.375</td>
<td>.002</td>
</tr>
<tr>
<td>Epigenetic aging (Hannum)</td>
<td></td>
<td>1.431</td>
<td>0.558</td>
<td>.011</td>
</tr>
<tr>
<td>Epigenetic aging (Horvath)</td>
<td></td>
<td>1.421</td>
<td>0.466</td>
<td>.002</td>
</tr>
<tr>
<td>Overall health status</td>
<td></td>
<td>4.957</td>
<td>1.800</td>
<td>.006</td>
</tr>
</tbody>
</table>

Note. The polynomial contrasts test whether the means increase across groups in a linear way (linear comparison) or whether the pattern of means is curvilinear (quadratic comparison). For the linear trend, the estimates for all the variables are significant (p < .05), indicating that as family economic hardship increased from stable low hardship to downward mobility to stable high hardship, health outcomes worsened in adolescents. Economic hardship groups were formed by first summing hardship variables and using a median split to define low and high hardship, and then creating groups over time of those who remained in the low hardship group, those who remained in the high hardship group, and those who moved from low hardship to high hardship over time. None of the quadratic estimates was significant.
4. Discussion

This study demonstrated that the more time African American adolescents spent experiencing economic hardship during the Great Recession, the worse their health profiles, as reflected in PBMC epigenetic aging, allostatic load, and self-reported health. The pattern of results differed somewhat depending on whether analyses considered linear trends vs. group comparisons, so in the sections that follow we consider each separately.

The results of linear trend analyses indicate that the dynamics of families’ economic circumstances over the Great Recession are associated with markers of cellular epigenetic aging and multi-systemic indicators of physiological risk (allostatic load). These findings are consistent with previous research demonstrating that the accumulation of economic hardship over time is associated with higher risk of myocardial infarction, greater difficulties with activities of daily living in adults, and various inflammatory processes thought to underlie these outcomes (Halvqvist et al., 2004; Tabassum et al., 2008; Lynch et al., 1997; Pollitt et al., 2008). The present study extends this research on the accumulation of hardship to a younger, minority group population, and to novel markers of PBMC aging based on methylation profiling.

The specific group comparisons demonstrated that the stable low economic hardship group had worse health than the stable low economic hardship group on all outcomes, from epigenetic aging to allostatic load to self-reported health. These findings are consistent with an established literature on the association between economic indicators such as income, and health outcomes (McDonough et al., 1997; Poulton et al., 2002). In addition, studies have documented different effects of socioeconomic status across points in the lifecourse (Smith et al., 1998; Lynch et al., 1994; Smith et al., 2010; Kurian and Cardarelli, 2007). Previous research has investigated the impact of low socioeconomic status at different stages in the lifecourse (Smith et al., 1998; Lynch et al., 1994; Kittleson et al., 2006; Kuh et al., 2002), as well as the effects of change in income over time in longitudinal studies of health (McDonough et al., 1997; Poulton et al., 2002). In addition, studies have documented different effects of socioeconomic status across periods by racial groups (Maty et al., 2010). However, most of these studies investigate naturally occurring changes over time in a family’s socioeconomic status, which may be more open to selection interpretations than are macroeconomic downturns.

There are fewer studies that have investigated effects of exogenous changes in economic conditions or policies on health outcomes. In general, these studies find benefits to physical health when policies or programs are put into place that improve the economic circumstances of households (Ludwig et al., 2011; Ospuyuk et al., 2014), or a worsening of individual health when economic conditions in a society worsen (Burgard et al., 2013, 2012; Modrek and Cullen, 2013). However, these studies have typically relied on self-reported health outcomes (Burgard et al., 2013, 2012), or have focused on adult health (Ludwig et al., 2011), with the few studies that have investigated effects on children focusing on mental health.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Low hardship (n = 131)</th>
<th>Downward mobility (n = 105)</th>
<th>High hardship (n = 82)</th>
<th>F (2,310)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allostatic load</td>
<td>0.74a</td>
<td>0.73b</td>
<td>0.89b</td>
<td>5.778</td>
<td>.003</td>
</tr>
<tr>
<td>Epigenetic aging (Horvath)</td>
<td>0.73a</td>
<td>0.12a</td>
<td>0.26a</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall health status</td>
<td>23.33a</td>
<td>22.87a</td>
<td>30.34b</td>
<td>5.155</td>
<td>.006</td>
</tr>
</tbody>
</table>

Note. Means with different superscripts are significantly different from each other (p < .05). Economic hardship groups were formed by first summing hardship variables and using a median split to define low and high hardship, and then creating groups over time of those who remained in the low hardship group, those who remained in the high hardship group, and those who moved from low hardship to high hardship over time (downward mobility).

Note that analyses of each individual economic indicator did not produce as consistent a pattern of results, suggesting that the accumulation of economic risk factors is important to consider. See Online Supplement Results for findings with individual economic indicators.

The specific group comparisons revealed that the group of adolescents whose families experienced economic decline during the Great Recession had greater allostatic load profiles compared to the group of adolescents whose families experienced stable low economic hardship during the Great Recession. We note that the patterns for the downward mobility group varied by outcome, such that the downward mobility group looked more like the high economic hardship group on allostatic load, but more like the low economic hardship group on self-reported health and epigenetic aging. It is possible that the experience of economic decline may have the most immediate effects on risk markers for cardiovascular diseases, such as blood pressure, obesity, and low-grade inflammation (since adolescents in the downward mobility group were most similar to adolescents from the stable high hardship group, and distinct from adolescents in the stable low hardship group, on these indicators immediately after the recession). The experience of economic troubles may heighten stress in the family or conflict between parents (Evans, 2004; Conger et al., 1994; McLoey, 1990), which in turn may have effects on allostatic load measures (Repetti et al., 2011; Evans and Kim, 2012; Danese and McEwen, 2012). Or it may be the case that downward mobility itself is stressful because of relative deprivation comparisons. There is a growing literature suggesting that the experience of perceiving oneself to be lower in status relative to others in around you is detrimental to health (Adler et al., 2000; Singh-Manoux et al., 2003; Marmot and Wilkinson, 2001; Manuck et al., 2010), and one form of relative deprivation may occur when families experiencing downward mobility compare what they currently have to what they used to have.

In contrast, disparities in PBMC epigenetic aging may take longer to develop, as suggested by the fact that adolescents in the downward mobility group were in between the stable low and stable high hardship groups on this indicator post-recession. In addition, it appears that adolescents may not perceive the early effects of changing economic conditions on their health, as self-report of health in the downward mobility group remained similar to the stable low hardship group following the recession.

The present study is the first to our knowledge to utilize an externally occurring economic event as a background context from which to investigate links with multiple immune and health measures among African Americans, a sample that experiences a disproportionate share of disease burden in our society (Williams et al., 2010; Kurian and Cardarelli, 2007). Previous research has investigated the impact of low socioeconomic status at different points in the lifecourse (Smith et al., 1998; Lynch et al., 1994; Kittleson et al., 2006; Kuh et al., 2002), as well as the effects of change in income over time in longitudinal studies of health (McDonough et al., 1997; Poulton et al., 2002). In addition, studies have documented different effects of socioeconomic status across periods by racial groups (Maty et al., 2010). However, most of these studies investigate naturally occurring changes over time in a family’s socioeconomic status, which may be more open to selection interpretations than are macroeconomic downturns.

There are fewer studies that have investigated effects of exogenous changes in economic conditions or policies on health outcomes. In general, these studies find benefits to physical health when policies or programs are put into place that improve the economic circumstances of households (Ludwig et al., 2011; Ospuyuk et al., 2014), or a worsening of individual health when economic conditions in a society worsen (Burgard et al., 2013, 2012; Modrek and Cullen, 2013). However, these studies have typically relied on self-reported health outcomes (Burgard et al., 2013, 2012), or have focused on adult health (Ludwig et al., 2011), with the few studies that have investigated effects on children focusing on mental health.
and social behaviors (Chase-Lansdale et al., 2003; Costello et al., 2003; Morris and Gennetian, 2003; Huston et al., 2001). This is the first study that we are aware of to focus on biological mechanisms including cellular epigenetic aging as well as allostatic load in African American adolescents in the context of changing macro-economic conditions, allowing us to better understand how broader societal-level economic conditions may be related to immune and physiological mechanisms with implications for health in an at-risk sample.

Strengths of this study include the investigation of PBMC epigenetic aging derived from DNA methylation profiles that have been shown to predict all-cause mortality (Marioni et al., 2015); the focus on African Americans, a group that experiences striking disparities in health outcomes in this society (Williams et al., 2010; Bibbins-Domingo et al., 2009); and the ability to utilize a significant macro-economic event, the Great Recession, that occurred in the context of an ongoing longitudinal study. One major limitation is the lack of any health or biological measures at baseline; this study was not initially designed with a focus on health, and so unfortunately, there were no health measures or biological specimens collected at baseline. Another important limitation is that because we were not able to experimentally manipulate economic conditions, we cannot know for certain that downward mobility in our sample was due to the Great Recession; however, economic indicators in our longitudinal sample were relatively stable prior to the Great Recession, and we believe that there are important potential public health and policy implications of situating cellular epigenetic and allostatic load profiles in a broader societal context. Other limitations include the lack of medical record data on diagnoses of medical conditions. We also note that there are no accepted norms for clinically significant levels of epigenetic aging, allostatic load, or the self-reported health measure, so it is difficult to discern the implications of these findings for clinical morbidity or mortality outcomes. Finally, because this study was conducted in rural African American adolescents, future research is needed testing whether patterns would be different in urban African American adolescents, in other minority groups, or in Caucasian adolescents.

In sum, this study documented that the more time spent under economic hardship during the period of the Great Recession, the relatively greater African American adolescents' cellular epigenetic aging and allostatic load, and the relatively poorer their overall health. As well, the group that experienced economic decline during the period of the Great Recession (starting off under low hardship conditions but changing to high hardship conditions) had greater allostatic load compared to the group who lived under low hardship conditions throughout the Great Recession. These findings have potential implications in highlighting the role that macro-economic conditions can play in the health of youth, but future research studies would need to monitor trajectories of change in cellular epigenetic and allostatic load measures over time and determine what magnitude of economic shock can produce changes in these markers in youth. Taken together, these findings suggest that changing the economic circumstances of families may have the potential to change biological and health trajectories of African American youth over time.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.bbi.2015.12.015.

References