Published in final edited form as:

Brain Behav Immun. 2016 March ; 53: 234–241. doi:10.1016/j.bbi.2015.12.015.

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=4.957, 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.

Keywords

socioeconomic status; epigenetic aging; adolescent health

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All authors declare no conflicts of interest.

INTRODUCTION

Economic hardship has far-reaching effects on a number of life outcomes, including physical health (Adler & Rehkopf, 2008; Fiscella & Franks, 1997; McDonough, Sacker, & Wiggins, 2005). In fact, health in the U.S. is widely acknowledged to be unequally distributed by both poverty and race (Adler, Boyce, Chesney, Folkman, & Syme, 1993; Williams, Mohammed, Leavell, & Collins, 2010; Smedley, Stith, & Nelson, 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 & Rehkopf, 2008). Health disparities by income and race are present even in childhood, and persist over the lifecourse (Chen, Matthews, & Boyce, 2002; Adler & 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 & 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 & 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 & Fry, 2014; Gould Ellen & 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 lifecourse, patterns of methylation change in ways that are relatively consistent across individuals (Horvath, 2013;

Jones, Goodman, & Kobor, 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 & 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., in press), 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.

MATERIALS AND METHODS

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 11–13) 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 & 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, 2, and 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 Figure 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 \$1,804/month, with 45.8% living below federal poverty thresholds. See Table 1.

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.

Measures

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 & Elder, 1994; Sobolewski & 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 (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 & 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 & 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 0–6 ($M = 1.68$, $SD = 1.47$ 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 measured family economic hardship in previous studies (Chen & 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.

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).

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 minutes, 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, Hane, & Tyrrell, 1986). Epinephrine and norepinephrine were assayed with high-pressure liquid chromatography with electrochemical detection (Riggin & 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 Duo Set 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 & 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 (Karlman, Singer, & Seeman, 2006), children (Evans, 2003), and adolescents (Brody et al., 2013) used similar metrics, combining multiple physiological indicators of risk into a composite.

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, Sherbourne, & Mazel, 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 & Benyamini, 1997).

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 (ANOVAs) 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.

RESULTS

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). 31.8% (n=105) experienced downward mobility, meaning that they were experiencing low economic hardship in 2007 but high economic hardship in 2010. 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.

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.

ANOVAs 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 Figure 1.

With respect to allostatic load, a significant difference across groups was 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 Figure 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 (Cohen's $d=.40$, $p<.05$) and downward mobility groups (Cohen's $d=.43$, $p<.05$). See Table 4 and Figure 1.

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.

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 (Hallqvist, Lynch, Bartley, Lang, & Blane, 2004; Tabassum et al., 2008; Lynch, Kaplan, & Shema, 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 high 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-relevant biological mediators including epinephrine, norepinephrine, cortisol, and inflammation (Ranjit et al., 2007; Cohen, Doyle, & Baum, 2006; Cohen et al., 2006; Deverts, Cohen, Kalra, & Matthews, 2012; Janicki-Deverts et al., 2007), but now also demonstrate the phenomenon with a novel measure of PBMC aging.

In addition, 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, Ge, Elder, & Lorenz, 1994; McLoyd, 1990), which in turn may have effects on allostatic load measures (Repetti, Robles, & Reynolds, 2011; Evans & Kim, 2012; Danese & 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, Epel, Castellazzo, & Ickovics, 2000; Singh-Manoux, Adler, & Marmot, 2003; Marmot & Wilkinson, 2001; Manuck, Phillips, Gianaros, Flory, & Muldoon, 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 & Cardarelli, 2007). Previous research has investigated the impact of low socioeconomic status at different points in the lifecourse (Smith, Hart, Blane, & Hole, 1998; Lynch et al., 1994; Kittleson et al., 2006; Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002), as well as the effects of change in income over time in longitudinal studies of health (McDonough, Duncan, Williams, & House, 1997; Poulton et al., 2002). In addition, studies have documented different effects of socioeconomic status across periods by racial groups (Maty, James, & Kaplan, 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; Osypuk, Joshi, Geronimo, & Acevedo-Garcia, 2014), or a worsening of individual health when economic conditions in a society worsen (Burgard, Ailshire, & Kalousova, 2013; Burgard, Seefeldt, & Zelner, 2012; Modrek & Cullen, 2013). However, these studies have typically relied on self-reported health outcomes (Burgard et al., 2013; Burgard et al., 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, Compton, Keeler, & Angold, 2003; Morris & 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.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The research reported in this article was supported by grants from the National Institute of Child Health and Human Development (R01 HD030588), National Heart, Lung, and Blood Institute (R01 HL108723, HL122328), and National Institute on Drug Abuse (P30 DA027827).

REFERENCES

- Adler NE, Rehkopf DH. U.S. disparities in health: descriptions, causes, and mechanisms. *Annu Rev Public Health*. 2008; 29:235–252. [PubMed: 18031225]
- Adler NE, Stewart J. Health disparities across the lifespan: meaning, methods, and mechanisms. *Ann N Y Acad Sci*. 2010; 1186:5–23. [PubMed: 20201865]
- Adler NE, Boyce WT, Chesney MA, Folkman S, Syme SL. Socioeconomic inequalities in health: No easy solution. *Journal of the American Medical Association*. 1993; 269:3140–3145. [PubMed: 8505817]
- Adler NE, Epel ES, Castellazzo G, Ickovics JR. Relationship of subjective and objective social status with psychological and physiological functioning: Preliminary data in health white women. *Health Psychology*. 2000; 19:586–592. [PubMed: 11129362]
- Bibbins-Domingo K, Pletcher MJ, Lin F, Vittinghoff E, Gardin JM, Arynchyn A, Hulley SB. Racial differences in incident heart failure among young adults. *N Engl J Med*. 2009; 360(12):1179–1190. [PubMed: 19297571]
- Blackburn EH, Epel ES. Telomeres and adversity: Too toxic to ignore. *Nature*. 2012; 490(7419):169–171. [PubMed: 23060172]
- Brody GH, Murry VM, Gerrard M, Gibbons FX, Molgaard V, McNair L, Neubaum-Carlan E. The Strong African American Families Program: translating research into prevention programming. *Child Dev*. 2004; 75(3):900–917. [PubMed: 15144493]
- Brody GH, Yu T, Chen YF, Kogan SM, Evans GW, Beach SR, Philibert RA. Cumulative socioeconomic status risk, allostatic load, and adjustment: a prospective latent profile analysis with contextual and genetic protective factors. *Dev Psychol*. 2013; 49(5):913–927. [PubMed: 22709130]
- Bureau of Labor Statistics. BLS Spotlight on Statistics: The recession of 2007–2009. 2012. www.bls.gov/spotlight
- Burgard SA, Ailshire JA, Kalousova L. The Great Recession and health: People, populations, and disparities. *Annals of the American Academy of Political and Social Science*. 2013; 650:194–213.
- Burgard SA, Seefeldt KS, Zelner S. Housing instability and health: findings from the Michigan Recession and Recovery Study. *Soc Sci Med*. 2012; 75(12):2215–2224. [PubMed: 22981839]
- Chase-Lansdale PL, Moffitt RA, Lohman BJ, Cherlin AJ, Coley RL, Pittman LD, Votruba-Drzal E. Mothers transitions from welfare to work and the well-being of preschoolers and adolescents. *Science*. 2003; 299(5612):1548–1552. [PubMed: 12624259]
- Chen YF, Brody GH. Family Economic Hardship, Corticotropin-Releasing Hormone Receptor Polymorphisms, and Depressive Symptoms in Rural African-American Youths. *J Adolesc Health*. 2015; 57(2):235–240. [PubMed: 26206446]
- Chen E, Matthews KA, Boyce WT. Socioeconomic differences in children's health: How and why do these relationships change with age? *Psychological Bulletin*. 2002; 128:295–329. [PubMed: 11931521]
- Cohen S, Doyle WJ, Baum A. Socioeconomic status is associated with stress hormones. *Psychosomatic Medicine*. 2006; 68(3):414–420. [PubMed: 16738073]
- Cohen S, Schwartz JE, Epel E, Kirschbaum C, Sidney S, Seeman T. Socioeconomic status, race, and diurnal cortisol decline in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. *Psychosomatic Medicine*. 2006; 68(1):41–50. [PubMed: 16449410]
- Conger RD, Ge X, Elder GH, Lorenz FO. Economic stress, coercive family process, and developmental problems of adolescents. *Child Development*. 1994; 65:541–561. [PubMed: 8013239]
- Conger, RD.; Elder, GH. Families in troubled times. New York, NY_: Aldine de Gruyter; 1994.

- Contreras LN, Hane S, Tyrrell JB. Urinary cortisol in the assessment of pituitary-adrenal function: Utility of 24-hour and spot determinations. *Journal of Clinical Endocrinology and Metabolism*. 1986; 62:965–969. [PubMed: 3958132]
- Costello EJ, Compton SN, Keeler G, Angold A. Relationships between poverty and psychopathology: A natural experiment. *Journal of the American Medical Association*. 2003; 290(15):2023–2029. [PubMed: 14559956]
- Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav*. 2012; 106(1):29–39. [PubMed: 21888923]
- Deverts DJ, Cohen S, Kalra P, Matthews KA. The prospective association of socioeconomic status with C-reactive protein levels in the CARDIA study. *Brain Behav Immun*. 2012; 26(7):1128–1135. [PubMed: 22884413]
- Epel ES, Blackburn EH, Lin J, Dhabhar FS, Adler NE, Morrow JD, Cawthon RM. Accelerated telomere shortening in response to life stress. *Proc Natl Acad Sci U S A*. 2004; 101(49):17312–17315. [PubMed: 15574496]
- Evans GW, Kim P. Childhood poverty and young adults' allostatic load: the mediating role of childhood cumulative risk exposure. *Psychol Sci*. 2012; 23(9):979–983. [PubMed: 22825357]
- Evans GW. A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology*. 2003; 39(5):924–933. [PubMed: 12952404]
- Evans GW. The environment of childhood poverty. *American Psychologist*. 2004; 59(2):77–92. [PubMed: 14992634]
- Fiscella K, Franks P. Poverty or income inequality as predictor of mortality: Longitudinal cohort study. *British Medical Journal*. 1997; 314:1724–1727. [PubMed: 9185498]
- GouldEllen, I.; Dastrup, S. Housing and the Great Recession. Stanford, CA: Stanford Center on Poverty and Inequality; 2012.
- Greenstone, M.; Looney, A. The lasting effects of the Great Recession: Six million missing workers and a new economic normal. Washington DC: The Hamilton Project; 2013.
- Hallqvist J, Lynch J, Bartley M, Lang T, Blane D. Can we disentangle life course processes of accumulation, critical period and social mobility? An analysis of disadvantaged socio-economic positions and myocardial infarction in the Stockholm Heart Epidemiology Program. *Social Science & Medicine*. 2004; 58(8):1555–1562. [PubMed: 14759698]
- Hannum G, Guinney J, Zhao L, Zhang L, Hughes G, Sadda S, Zhang K. Genome-wide methylation profiles reveal quantitative views of human aging rates. *Mol Cell*. 2013; 49(2):359–367. [PubMed: 23177740]
- Hays RD, Sherbourne CD, Mazel RM. The RAND 36-item Health Survey 1.0. *Health Economics*. 1993; 2:217–227. [PubMed: 8275167]
- Horvath S. DNA methylation age of human tissues and cell types. *Genome Biol*. 2013; 14(10):R115. [PubMed: 24138928]
- Horvath S, Erhart W, Brosch M, Ammerpohl O, von Schonfels W, Ahrens M, Hampe J. Obesity accelerates epigenetic aging of human liver. *Proc Natl Acad Sci U S A*. 2014; 111(43):15538–15543. [PubMed: 25313081]
- Howell, DC. Statistical methods for psychology. 6th. Belmont, CA: Thomson Wadsworth; 2007.
- Huston AC, Duncan GJ, Granger R, Bos J, McLoyd V, Mistry R, Ventura A. Work-based antipoverty programs for parents can enhance the school performance and social behavior of children. *Child Development*. 2001; 72:318–336. [PubMed: 11280487]
- Idler EL, Benyamini Y. Self-rated health and mortality: A review of twenty-seven community studies. *Journal of Health and Social Behavior*. 1997; 38:21–37. [PubMed: 9097506]
- Janicki-Deverts D, Cohen S, Adler NE, Schwartz JE, Matthews KA, Seeman TE. Socioeconomic status is related to urinary catecholamines in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Psychosom Med*. 2007; 69(6):514–520. [PubMed: 17636147]
- Jones MJ, Goodman SJ, Kobor MS. DNA methylation and healthy human aging. *Aging Cell*. 2015
- Kamarck TW, Jennings JR, Debski TT, Glickman-Weiss E, Johnson PS, Eddy MJ, Manuck SB. Reliable measures of behaviorally-evoked cardiovascular reactivity from a PC-based test battery: Results from student and community samples. *Psychophysiology*. 1992; 29:17–28. [PubMed: 1609024]

- Karlamangla AS, Singer BH, Seeman TE. Reduction in allostatic load in older adults is associated with lower all-cause mortality risk: MacArthur studies of successful aging. *Psychosom Med*. 2006; 68(3):500–507. [PubMed: 16738085]
- Kittleson MM, Meoni LA, Wang NY, Chu AY, Ford DE, Klag MJ. Association of childhood socioeconomic status with subsequent coronary heart disease in physicians. *Archives of Internal Medicine*. 2006; 166(21):2356–2361. [PubMed: 17130389]
- Kochhar, R.; Fry, R. Wealth inequality has widened along racial, ethnic lines since end of Great Recession. Washington DC: Pew Research Center; 2014.
- Kuh D, Hardy R, Langenberg C, Richards M, Wadsworth MEJ. Mortality in adults aged 26–54 years related to socioeconomic conditions in childhood and adulthood: Post war birth cohort study. *British Medical Journal*. 2002; 325:1076–1080. [PubMed: 12424168]
- Kurian AK, Cardarelli KM. Racial and ethnic differences in cardiovascular disease risk factors: a systematic review. *Ethn Dis*. 2007; 17(1):143–152. [PubMed: 17274224]
- Ludwig J, Sanbonmatsu K, Gennetian L, Adam E, Duncan GJ, Katz LF, McDade TW. Neighborhoods, obesity, and diabetes - A randomized social experiment. *New England Journal of Medicine*. 2011; 365:1509–1519. [PubMed: 22010917]
- Lynch JW, Kaplan GA, Cohen RD, Kauhanen J, Wilson TW, Smith NL, Salonen JT. Childhood and adult socioeconomic status as predictors of mortality in Finland. *Lancet*. 1994; 343:524–527. [PubMed: 7906766]
- Lynch JW, Kaplan GA, Shema SJ. Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. *New England Journal of Medicine*. 1997; 337:1889–1895. [PubMed: 9407157]
- Manuck SB, Phillips JE, Gianaros PJ, Flory JD, Muldoon MF. Subjective socioeconomic status and presence of the metabolic syndrome in midlife community volunteers. *Psychosom Med*. 2010; 72(1):35–45. [PubMed: 19933505]
- Marioni RE, Shah S, McRae AF, Chen BH, Colicino E, Harris SE, Deary IJ. DNA methylation age of blood predicts all-cause mortality in later life. *Genome Biol*. 2015; 16(1):25. [PubMed: 25633388]
- Marmot M, Wilkinson RG. Psychosocial and material pathways in the relation between income and health: a response to Lynch et al. *BMJ*. 2001; 322:1233–1236. [PubMed: 11358781]
- Maty SC, James SA, Kaplan GA. Life-course socioeconomic position and incidence of diabetes mellitus among blacks and whites: the Alameda County Study, 1965–1999. *Am J Public Health*. 2010; 100(1):137–145. [PubMed: 19197084]
- McDonough P, Duncan GJ, Williams D, House J. Income dynamics and adult mortality in the United States, 1972 through 1989. *American Journal of Public Health*. 1997; 87:1476–1483. [PubMed: 9314800]
- McDonough P, Sacker A, Wiggins RD. Time on my side? Life course trajectories of poverty and health. *Social Science & Medicine*. 2005; 61(8):1795–1808. [PubMed: 16029777]
- McLoyd VC. The impact of economic hardship on black families and children: Psychological distress, parenting, and socioemotional development. *Child Development*. 1990; 61:311–346. [PubMed: 2188806]
- Modrek S, Cullen MR. Health consequences of the ‘Great Recession’ on the employed: evidence from an industrial cohort in aluminum manufacturing. *Soc Sci Med*. 2013; 92:105–113. [PubMed: 23849284]
- Morris PA, Gennetian LA. Identifying the effects of income on children’s development using experimental data. *Journal of Marriage and Family*. 2003; 65:716–729.
- Osyuk TL, Joshi P, Geronimo K, Acevedo-Garcia D. Do social and economic policies influence health? A review. *Current Epidemiology Reports*. 2014; 1:149–164. [PubMed: 25984439]
- Pollitt RA, Kaufman JS, Rose KM, Diez-Roux AV, Zeng D, Heiss G. Cumulative life course and adult socioeconomic status and markers of inflammation in adulthood. *J Epidemiol Community Health*. 2008; 62(6):484–491. [PubMed: 18477746]
- Poulton R, Caspi A, Milne BJ, Thomson WM, Taylor A, Sears MR, Moffitt TE. Association between children’s experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet*. 2002; 360(9346):1640–1645. [PubMed: 12457787]

- Proctor, BD.; Dalaker, J. Poverty in the United States: 2002 (Current Population Reports). Washington DC: US Census Bureau; 2003.
- Ranjit N, Diez-Roux AV, Shea S, Cushman M, Ni H, Seeman T. Socioeconomic position, race/ethnicity, and inflammation in the multi-ethnic study of atherosclerosis. *Circulation*. 2007; 116(21):2383–2390. [PubMed: 18025402]
- Repetti RL, Robles TF, Reynolds B. Allostatic processes in the family. *Dev Psychopathol*. 2011; 23(3): 921–938. [PubMed: 21756442]
- Riggin RM, Kissinger PT. Determination of catecholamines in urine by reverse-phase liquid chromatography with electrochemical detection. *Analytical Chemistry*. 1977; 49:2109–2111. [PubMed: 907164]
- Shalev I, Entringer S, Wadhwa PD, Wolkowitz OM, Puterman E, Lin J, Epel ES. Stress and telomere biology: a lifespan perspective. *Psychoneuroendocrinology*. 2013; 38(9):1835–1842. [PubMed: 23639252]
- Simpkin AJ, Gemani G, Suderman M, Gaunt TR, Lyttleton O, McArdle WL, Davey Smith G. Prenatal and early life influences on epigenetic age in children: A study of mother-offspring pairs from two cohort studies. *Human Molecular Genetics*. in press.
- Singh-Manoux A, Adler NE, Marmot MG. Subjective social status: its determinants and its association with measures of ill-health in the Whitehall II study. *Social Science & Medicine*. 2003; 56(6): 1321–1333. [PubMed: 12600368]
- Smedley, BD.; Stith, AY.; Nelson, AR. Unequal treatment: Confronting racial and ethnic disparities in health care. Washington DC: The National Academies Press; 2003.
- Smith GD, Hart C, Blane D, Hole D. Adverse socioeconomic conditions in childhood and cause specific adult mortality: Prospective observational study. *British Medical Journal*. 1998; 316:1631–1635. [PubMed: 9603744]
- Sobolewski JM, Amato PR. Economic hardship in the family of origin and children's psychological well-being in adulthood. *Journal of Marriage and Family*. 2005; 67:141–156.
- Tabachnick, BG.; Fidell, LS. Using multivariate statistics. 5th. Boston, MA: Allyn and Bacon; 2007.
- Tabassum F, Kumari M, Rumley A, Lowe G, Power C, Strachan DP. Effects of socioeconomic position on inflammatory and hemostatic markers: a life-course analysis in the 1958 British birth cohort. *Am J Epidemiol*. 2008; 167(11):1332–1341. [PubMed: 18367468]
- Taylor, P.; Morin, R.; Kochhar, R.; Parker, K.; Cohn, D.; Lopez, MH.; Espinoza, S. How the Great Recession has changed life in America. Washington DC: Pew Research Center; 2010.
- Tietz, NW. Fundamentals of clinical chemistry. 2nd. Philadelphia, PA: Saunders; 1976.
- Williams DR, Mohammed SA, Leavell J, Collins C. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Ann N Y Acad Sci*. 2010; 1186:69–101. [PubMed: 20201869]

Research Highlights

- More time in economic hardship, greater epigenetic aging in African American youth.
- More time in economic hardship, higher allostatic load in African American youth.
- More time in economic hardship, poorer self-reported health in African American youth.

Figure 1. Differences in Adolescent Outcomes by Economic Trajectory Across the Great Recession

Means of (a) allostatic load, (b) epigenetic aging using the Hannum method, (c) epigenetic aging using the Horvath method, and (d) overall health status (higher numbers indicating worse health) by family economic hardship groups. 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). $n = 131$ for stable low hardship group, 105 for downward mobility group, and 82 for stable high hardship group. Significant differences are indicated by different letters ($p < .05$). Error bars = +1 standard error.

Table 1

Sample Characteristics in 2007 by Family Economic Hardship Groups

	Low Hardship	Downward Mobility	High Hardship
Characteristics	% or <i>M</i> (<i>SD</i>)	% or <i>M</i> (<i>SD</i>)	% or <i>M</i> (<i>SD</i>)
Adolescent age (in years)	16.54 (0.54)	16.59 (0.51)	16.65 (0.50)
Parent age (in years)	42.14 (6.06)	43.56 (8.59)	43.61 (7.48)
Adolescent gender			
Male	46.7%	45.5%	46.9%
Female	53.3%	54.5%	53.1%
Parent education			
< high school	13.0%	19.8%	38.8%
High school degree or GED	22.3%	36.6%	36.7%
Some college	53.3%	40.5%	21.4%
college graduate	11.4%	3.1%	3.1%
Parent marital status			
Married or partnered	44.8%	32.6%	18.4%
Single	55.2%	67.4%	81.6%

Table 2
Family Economic Hardship Index Variables in 2007 and 2010 by Family Economic Hardship Groups

Variables	2007				2010			
	Low Hardship		Downward Mobility		High Hardship		Low Hardship	
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
Income-to-needs ratio	2.17 (2.94)	1.86 (3.65)	0.78 (0.80)	1.76 (1.28)	0.77 (0.71)	0.50 (0.48)	0.46 (0.50)	0.63 (0.48)
Receipt of TANF	0.02 (0.15)	0.02 (0.12)	0.13 (0.34)	0.11 (0.31)	0.39 (0.49)	0.46 (0.50)	0.63 (0.48)	1.70 (1.06)
Unemployment	0.09 (0.28)	0.23 (0.42)	0.53 (0.50)	0.09 (0.28)	0.48 (0.50)	12.57 (2.09)	7.51 (1.78)	
Adequacy of income	3.52 (1.10)	3.18 (0.97)	1.93 (0.83)	2.72 (0.78)	1.75 (0.76)			
Unmet material needs	7.68 (2.50)	8.64 (2.57)	11.51 (2.40)	8.52 (2.10)	11.20 (2.44)			
Cannot make ends meet	3.91 (1.78)	4.91 (1.86)	7.40 (1.97)	4.87 (1.64)	7.25 (1.99)			

Note. Range of income-to-needs ratio=0–30. Range of receipt of TANF variable=0–1. Range of unemployment=0–1. Range of adequacy of income variable=1–5. Range of unmet material needs score=4–16. Range of cannot make ends meet score=2–10. 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.

Table 3

Polynomial Contrasts for Means Differences in Adolescents' Health Outcomes by Family Economic Hardship Groups

Variables	Polynomial Contrasts		
	Linear		
	Estimate	se	p
Allostatic load	1.151	0.375	.002
Epigenetic aging (Hannum)	1.431	0.558	.011
Epigenetic aging (Horvath)	1.421	0.466	.002
Overall health status	4.957	1.800	.006

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.

Adolescents' Biological and Health Measures at Age 19 by Family Economic Hardship Groups

Table 4

Variables	Family Economic Hardship Groups					
	Low Hardship (n = 131)		Downward Mobility (n = 105)		High Hardship (n = 82)	
	M	SE	M	SE	M	SE
Allostatic load	−0.74 ^a	0.32	0.57 ^b	0.35	0.89 ^b	0.40
Epigenetic aging (Hannum)	−0.73 ^a	0.48	0.12	0.52	1.29 ^b	0.60
Epigenetic aging (Horvath)	−0.73 ^a	0.40	−0.26 ^a	0.43	1.28 ^b	0.50
Overall health status	23.33 ^a	1.54	22.87 ^a	1.67	30.34 ^b	1.93
					5.155	5.155
						.006

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).