Brain, Behavior, and Immunity 53 (2016) 234–241

Contents lists available at ScienceDirect

Brain, Behavior, and Immunity

journal homepage: www.elsevier.com/locate/ybrbi

Full-length Article

The Great Recession and health risks in African American youth

Edith Chen [a,*], Gregory E. Miller [a], Tianyi Yu [b], Gene H. Brody [b]

[a] Institute for Policy Research and Department of Psychology, Northwestern University, USA
[b] Center for Family Research, University of Georgia, USA

ARTICLE INFO

Article history:
Received 25 September 2015
Received in revised form 19 December 2015
Accepted 19 December 2015
Available online 21 December 2015

Keywords:
Socioeconomic status
Epigenetic aging
Adolescent health

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.

1. 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

* Corresponding author at: Northwestern University, 2029 Sheridan Road, Evanston, IL 60208, USA.
E-mail address: edith.chen@northwestern.edu (E. Chen).

http://dx.doi.org/10.1016/j.bbi.2015.12.015
0889-1591/

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 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 1
Sample characteristics in 2007 by family economic hardship groups.

	Low hardship	Downward mobility	High hardship
Characteristics	% or M (SD)	% or M (SD)	% or M (SD)
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%

(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 ($\geqslant 3$ 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 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 Agilant 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 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 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,

Table 2
Family economic hardship index variables in 2007 and 2010 by family economic hardship groups.

	2007			2010		
	Low hardship	Downward mobility	High hardship	Low hardship	Downward mobility	High hardship
Variables	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)
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)
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)
Unemployment	0.09 (0.28)	0.23 (0.42)	0.53 (0.50)	0.09 (0.28)	0.48 (0.50)	0.63 (0.48)
Adequacy of income	3.52 (1.10)	3.18 (0.97)	1.93 (0.83)	2.72 (0.78)	1.75 (0.76)	1.70 (1.06)
Unmet material needs	7.68 (2.50)	8.64 (2.57)	11.51 (2.40)	8.52 (2.10)	11.20 (2.44)	12.57 (2.09)
Cannot make ends meet	3.91 (1.78)	4.91 (1.86)	7.40 (1.97)	4.87 (1.64)	7.25 (1.99)	7.51 (1.78)

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.

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.

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). 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.

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.

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 Fig. 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 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 (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.

	Polynomial contrasts Linear		
Variables	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.

Table 4
Adolescents' biological and health measures at age 19 by family economic hardship groups.

	Family economic hardship groups							
	Low hardship (n = 131)		Downward mobility (n = 105)		High hardship (n = 82)			
Variables	M	SE	M	SE	M	SE	F (2,310)	p
Allostatic load	−0.74[a]	0.32	0.57[b]	0.35	0.89[b]	0.40	5.778	.003
Epigenetic aging (Hannum)	−0.73[a]	0.48	0.12	0.52	1.29[b]	0.60	3.286	.039
Epigenetic aging (Horvath)	−0.73[a]	0.40	−0.26[a]	0.43	1.28[b]	0.50	4.881	.008
Overall health status	23.33[a]	1.54	22.87[a]	1.67	30.34[b]	1.93	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).

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.

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 multisystemic 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 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 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 et al., 2006a,b; Deverts et al., 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 et al., 1994; McLoyd, 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; Osypuk 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

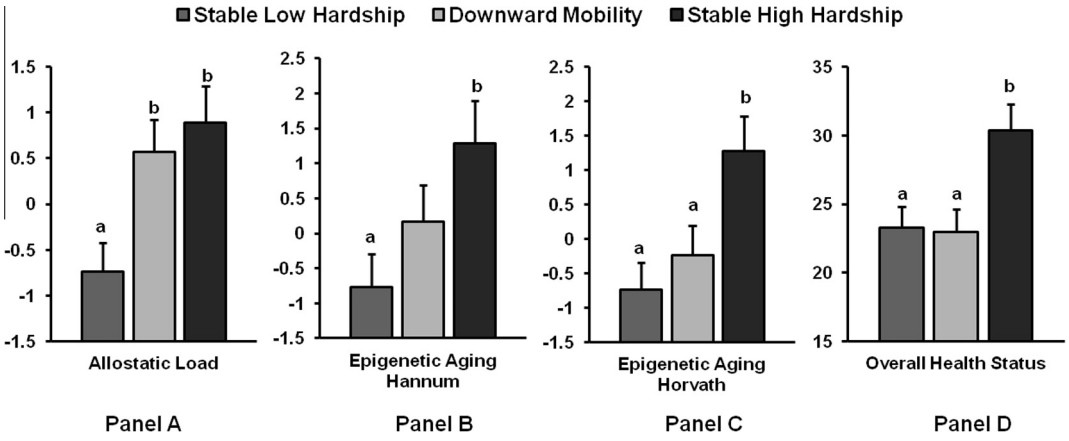

Fig. 1. Differences in adolescent outcomes by economic trajectory across the Great Recession. Means of (a) allostatic load, (b) epigenetic aging using the Hannum method, (c) epigenetic aging using the Horvath method, and (d) overall health status (higher numbers indicating worse health) by family economic 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.

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.

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). All authors declare no conflicts of interest.

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

Adler, N.E., Rehkopf, D.H., 2008. U.S. disparities in health: descriptions, causes, and mechanisms. Annu. Rev. Public Health 29, 235–252.
Adler, N.E., Stewart, J., 2010. Health disparities across the lifespan: meaning, methods, and mechanisms. Ann. N. Y. Acad. Sci. 1186, 5–23.
Adler, N.E., Boyce, W.T., Chesney, M.A., Folkman, S., Syme, S.L., 1993. Socioeconomic inequalities in health: no easy solution. J. Am. Med. Assoc. 269, 3140–3145.
Adler, N.E., Epel, E.S., Castellazzo, G., Ickovics, J.R., 2000. Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in health white women. Health Psychol. 19, 586–592.
Bibbins-Domingo, K., Pletcher, M.J., Lin, F., Vittinghoff, E., Gardin, J.M., Arynchyn, A., Hulley, S.B., 2009. Racial differences in incident heart failure among young adults. New Engl. J. Med. 360 (12), 1179–1190.
Blackburn, E.H., Epel, E.S., 2012. Telomeres and adversity: too toxic to ignore. Nature 490 (7419), 169–171.
Brody, G.H., Murry, V.M., Gerrard, M., Gibbons, F.X., Molgaard, V., McNair, L., Neubaum-Carlan, E., 2004. The strong African American families program: translating research into prevention programming. Child Dev. 75 (3), 900–917.

Brody, G.H., Yu, T., Chen, Y.F., Kogan, S.M., Evans, G.W., Beach, S.R., Philibert, R.A., 2013. Cumulative socioeconomic status risk, allostatic load, and adjustment: a prospective latent profile analysis with contextual and genetic protective factors. Dev. Psychol. 49 (5), 913–927.

Bureau of Labor Statistics, 2012. BLS Spotlight on Statistics: the recession of 2007–2009. <www.bls.gov/spotlight>.

Burgard, S.A., Seefeldt, K.S., Zelner, S., 2012. Housing instability and health: findings from the Michigan Recession and recovery study. Soc. Sci. Med. 75 (12), 2215–2224.

Burgard, S.A., Ailshire, J.A., Kalousova, L., 2013. The Great Recession and health: people, populations, and disparities. Ann. Am. Acad. Political Soc. Sci. 650, 194–213.

Chase-Lansdale, P.L., Moffitt, R.A., Lohman, B.J., Cherlin, A.J., Coley, R.L., Pittman, L.D., Votruba-Drzal, E., 2003. Mothers transitions from welfare to work and the well-being of preschoolers and adolescents. Science 299 (5612), 1548–1552.

Chen, Y.F., Brody, G.H., 2015. Family economic hardship, corticotropin-releasing hormone receptor polymorphisms, and depressive symptoms in rural African-American youths. J. Adolesc. Health 57 (2), 235–240.

Chen, E., Matthews, K.A., Boyce, W.T., 2002. Socioeconomic differences in children's health: How and why do these relationships change with age? Psychol. Bull. 128, 295–329.

Cohen, S., Doyle, W.J., Baum, A., 2006a. Socioeconomic status is associated with stress hormones. Psychosom. Med. 68 (3), 414–420.

Cohen, S., Schwartz, J.E., Epel, E., Kirschbaum, C., Sidney, S., Seeman, T., 2006b. Socioeconomic status, race, and diurnal cortisol decline in the coronary artery risk development in young adults (CARDIA) study. Psychosom. Med. 68 (1), 41–50.

Conger, R.D., Elder, G.H., 1994. Families in Troubled Times. Aldine de Gruyter, New York, NY.

Conger, R.D., Ge, X., Elder, G.H., Lorenz, F.O., 1994. Economic stress, coercive family process, and developmental problems of adolescents. Child Dev. 65, 541–561.

Contreras, L.N., Hane, S., Tyrrell, J.B., 1986. Urinary cortisol in the assessment of pituitary–adrenal function: utility of 24-hour and spot determinations. J. Clin. Endocrinol. Metab. 62, 965–969.

Costello, E.J., Compton, S.N., Keeler, G., Angold, A., 2003. Relationships between poverty and psychopathology: a natural experiment. J. Am. Med. Assoc. 290 (15), 2023–2029.

Danese, A., McEwen, B.S., 2012. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. Physiol. Behav. 106 (1), 29–39.

Deverts, D.J., Cohen, S., Kalra, P., Matthews, K.A., 2012. The prospective association of socioeconomic status with C-reactive protein levels in the CARDIA study. Brain Behav. Immun. 26 (7), 1128–1135.

Epel, E.S., Blackburn, E.H., Lin, J., Dhabhar, F.S., Adler, N.E., Morrow, J.D., Cawthon, R. M., 2004. Accelerated telomere shortening in response to life stress. Proc. Natl. Acad. Sci. USA 101 (49), 17312–17315.

Evans, G.W., 2003. A multimethodological analysis of cumulative risk and allostatic load among rural children. Dev. Psychol. 39 (5), 924–933.

Evans, G.W., 2004. The environment of childhood poverty. Am. Psychol. 59 (2), 77–92.

Evans, G.W., Kim, P., 2012. Childhood poverty and young adults' allostatic load: the mediating role of childhood cumulative risk exposure. Psychol. Sci. 23 (9), 979–983.

Fiscella, K., Franks, P., 1997. Poverty or income inequality as predictor of mortality: longitudinal cohort study. Br. Med. J. 314, 1724–1727.

Gould Ellen, I., Dastrup, S., 2012. Housing and the Great Recession. Stanford Center on Poverty and Inequality, Stanford, CA.

Greenstone, M., Looney, A., 2013. The Lasting Effects of the Great Recession: Six Million Missing Workers and a New Economic Normal. The Hamilton Project, Washington DC.

Hallqvist, J., Lynch, J., Bartley, M., Lang, T., Blane, D., 2004. 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. Soc. Sci. Med. 58 (8), 1555–1562.

Hannum, G., Guinney, J., Zhao, L., Zhang, L., Hughes, G., Sadda, S., Zhang, K., 2013. Genome-wide methylation profiles reveal quantitative views of human aging rates. Mol. Cell 49 (2), 359–367.

Hays, R.D., Sherbourne, C.D., Mazel, R.M., 1993. The RAND 36-item health survey 1.0. Health Econ. 2, 217–227.

Horvath, S., 2013. DNA methylation age of human tissues and cell types. Genome Biol. 14 (10), R115.

Horvath, S., Erhart, W., Brosch, M., Ammerpohl, O., von Schonfels, W., Ahrens, M., Hampe, J., 2014. Obesity accelerates epigenetic aging of human liver. Proc. Natl. Acad. Sci. USA 111 (43), 15538–15543.

Howell, D.C., 2007. Statistical Methods for Psychology, sixth ed. Thomson Wadsworth, Belmont, CA.

Huston, A.C., Duncan, G.J., Granger, R., Bos, J., McLoyd, V., Mistry, R., Ventura, A., 2001. Work-based antipoverty programs for parents can enhance the school performance and social behavior of children. Child Dev. 72, 318–336.

Idler, E.L., Benyamini, Y., 1997. Self-rated health and mortality: a review of twenty-seven community studies. J. Health Soc. Behav. 38, 21–37.

Janicki-Deverts, D., Cohen, S., Adler, N.E., Schwartz, J.E., Matthews, K.A., Seeman, T. E., 2007. Socioeconomic status is related to urinary catecholamines in the coronary artery risk development in young adults (CARDIA) study. Psychosom. Med. 69 (6), 514–520.

Jones, M.J., Goodman, S.J., Kobor, M.S., 2015. DNA methylation and healthy human aging. Aging Cell.

Kamarck, T.W., Jennings, J.R., Debski, T.T., Glickman-Weiss, E., Johnson, P.S., Eddy, M. J., Manuck, S.B., 1992. Reliable measures of behaviorally-evoked cardiovascular reactivity from a PC-based test battery: results from student and community samples. Psychophysiology 29, 17–28.

Karlamangla, A.S., Singer, B.H., Seeman, T.E., 2006. Reduction in allostatic load in older adults is associated with lower all-cause mortality risk: MacArthur studies of successful aging. Psychosom. Med. 68 (3), 500–507.

Kittleson, M.M., Meoni, L.A., Wang, N.Y., Chu, A.Y., Ford, D.E., Klag, M.J., 2006. Association of childhood socioeconomic status with subsequent coronary heart disease in physicians. Arch. Intern. Med. 166 (21), 2356–2361.

Kochhar, R., Fry, R., 2014. Wealth Inequality has Widened Along Racial, Ethnic Lines Since End of Great Recession. Pew Research Center, Washington DC.

Kuh, D., Hardy, R., Langenberg, C., Richards, M., Wadsworth, M.E.J., 2002. Mortality in adults aged 26–54 years related to socioeconomic conditions in childhood and adulthood: post war birth cohort study. Br. Med. J. 325, 1076–1080.

Kurian, A.K., Cardarelli, K.M., 2007. Racial and ethnic differences in cardiovascular disease risk factors: a systematic review. Ethn. Dis. 17 (1), 143–152.

Ludwig, J., Sanbonmatsu, K., Gennetian, L., Adam, E., Duncan, G.J., Katz, L.F., McDade, T.W., 2011. Neighborhoods, obesity, and diabetes – a randomized social experiment. New Engl. J. Med. 365, 1509–1519.

Lynch, J.W., Kaplan, G.A., Cohen, R.D., Kauhanen, J., Wilson, T.W., Smith, N.L., Salonen, J.T., 1994. Childhood and adult socioeconomic status as predictors of mortality in Finland. Lancet 343, 524–527.

Lynch, J.W., Kaplan, G.A., Shema, S.J., 1997. Cumulative impact of sustained economic hardship on physical, cognitive, psychological, and social functioning. New Engl. J. Med. 337, 1889–1895.

Manuck, S.B., Phillips, J.E., Gianaros, P.J., Flory, J.D., Muldoon, M.F., 2010. Subjective socioeconomic status and presence of the metabolic syndrome in midlife community volunteers. Psychosom. Med. 72 (1), 35–45.

Marioni, R.E., Shah, S., McRae, A.F., Chen, B.H., Colicino, E., Harris, S.E., Deary, I.J., 2015. DNA methylation age of blood predicts all-cause mortality in later life. Genome Biol. 16 (1), 25.

Marmot, M., Wilkinson, R.G., 2001. Psychosocial and material pathways in the relation between income and health: a response to Lynch et al. BMJ 322, 1233–1236.

Maty, S.C., James, S.A., Kaplan, G.A., 2010. Life-course socioeconomic position and incidence of diabetes mellitus among blacks and whites: the Alameda County Study, 1965–1999. Am. J. Public Health 100 (1), 137–145.

McDonough, P., Duncan, G.J., Williams, D., House, J., 1997. Income dynamics and adult mortality in the United States, 1972 through 1989. Am. J. Public Health 87, 1476–1483.

McDonough, P., Sacker, A., Wiggins, R.D., 2005. Time on my side? Life course trajectories of poverty and health. Soc. Sci. Med. 61 (8), 1795–1808.

McLoyd, V.C., 1990. The impact of economic hardship on black families and children: Psychological distress, parenting, and socioemotional development. Child Dev. 61, 311–346.

Modrek, S., Cullen, M.R., 2013. Health consequences of the 'Great Recession' on the employed: evidence from an industrial cohort in aluminum manufacturing. Soc. Sci. Med. 92, 105–113.

Morris, P.A., Gennetian, L.A., 2003. Identifying the effects of income on children's development using experimental data. J. Marriage Family 65, 716–729.

Osypuk, T.L., Joshi, P., Geronimo, K., Acevedo-Garcia, D., 2014. Do social and economic policies influence health? A review. Curr. Epidemiol. Rep. 1, 149–164.

Pollitt, R.A., Kaufman, J.S., Rose, K.M., Diez-Roux, A.V., Zeng, D., Heiss, G., 2008. Cumulative life course and adult socioeconomic status and markers of inflammation in adulthood. J. Epidemiol. Community Health 62 (6), 484–491.

Poulton, R., Caspi, A., Milne, B.J., Thomson, W.M., Taylor, A., Sears, M.R., Moffitt, T.E., 2002. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. Lancet 360 (9346), 1640–1645.

Proctor, B.D., Dalaker, J., 2003. Poverty in the United States: 2002 (Current Population Reports). US Census Bureau, Washington DC.

Ranjit, N., Diez-Roux, A.V., Shea, S., Cushman, M., Ni, H., Seeman, T., 2007. Socioeconomic position, race/ethnicity, and inflammation in the multi-ethnic study of atherosclerosis. Circulation 116 (21), 2383–2390.

Repetti, R.L., Robles, T.F., Reynolds, B., 2011. Allostatic processes in the family. Dev. Psychopathol. 23 (3), 921–938.

Riggin, R.M., Kissinger, P.T., 1977. Determination of catecholamines in urine by reverse-phase liquid chromatography with electrochemical detection. Anal. Chem. 49, 2109–2111.

Shalev, I., Entringer, S., Wadhwa, P.D., Wolkowitz, O.M., Puterman, E., Lin, J., Epel, E. S., 2013. Stress and telomere biology: a lifespan perspective. Psychoneuroendocrinology 38 (9), 1835–1842.

Simpkin, A.J., Gemani, G., Suderman, M., Gaunt, T.R., Lyttleton, O., McArdle, W.L., Smith, G. Davey, 2016. Prenatal and early life influences on epigenetic age in children: a study of mother-offspring pairs from two cohort studies. Hum. Mol. Genet. 25 (1), 191–201.

Singh-Manoux, A., Adler, N.E., Marmot, M.G., 2003. Subjective social status: its determinants and its association with measures of ill-health in the Whitehall II study. Soc. Sci. Med. 56 (6), 1321–1333.

Smedley, B.D., Stith, A.Y., Nelson, A.R., 2003. Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care. The National Academies Press, Washington DC.

Smith, G.D., Hart, C., Blane, D., Hole, D., 1998. Adverse socioeconomic conditions in childhood and cause specific adult mortality: prospective observational study. Br. Med. J. 316, 1631–1635.
Sobolewski, J.M., Amato, P.R., 2005. Economic hardship in the family of origin and children's psychological well-being in adulthood. J. Marriage Family 67, 141–156.
Tabachnick, B.G., Fidell, L.S., 2007. Using Multivariate Statistics, fifth ed. Allyn and Bacon, Boston, MA.
Tabassum, F., Kumari, M., Rumley, A., Lowe, G., Power, C., Strachan, D.P., 2008. Effects of socioeconomic position on inflammatory and hemostatic markers: a life-course analysis in the 1958 British birth cohort. Am. J. Epidemiol. 167 (11), 1332–1341.
Taylor, P., Morin, R., Kochhar, R., Parker, K., Cohn, D., Lopez, M.H., Espinoza, S., 2010. How the Great Recession has changed life in America. Pew Research Center, Washington DC.
Tietz, N.W., 1976. Fundamentals of Clinical Chemistry, second ed. Saunders, Philadelphia, PA.
Williams, D.R., Mohammed, S.A., Leavell, J., Collins, C., 2010. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. Ann. N. Y. Acad. Sci. 1186, 69–101.