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Full-length Article

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.

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

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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%