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EMPIRICAL REPORTS

CHILD DEVELOPMENT

Childhood poverty, immune cell aging, and African Americans' insulin resistance: A prospective study

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Abstract

The present study investigated developmental pathways that can contribute to chronic disease among rural African Americans. With a sample of 342 African American youth (59% female) from the southeastern United States followed for nearly two decades (2001–2019), we examined the prospective association between family poverty during adolescence (ages 11–18) and insulin resistance (IR) in young adulthood (ages 25–29) as well as underlying biological and psychosocial mechanisms. Results indicated family poverty during adolescence forecast higher levels of IR in young adulthood, with accelerated immune cell aging at age 20 partially mediating this association. Serial mediational models confirmed the hypothesized pathway linking family poverty, perceived life chances, cellular aging, and IR. Findings provide empirical support for theorized developmental precursors of chronic disease.

The present study focused on understanding the relation between childhood poverty and insulin resistance (IR) as well as biological (i.e., cellular aging) and psychological (i.e., perceived life chances) mechanisms that may underlie this association for African Americans in the rural Southeast of the United States. This demographic is among the most disadvantaged populations in the United States in terms of life expectancy (Geronimus et al., 2001; Singh & Siahpush, 2014) and is at heightened risk for various chronic diseases, including type 2 diabetes and cardiometabolic disease (Cherrington et al., 2012; Hartley, 2004). Existing research into these health disparities has tended to focus on contemporaneous social factors such as socioeconomic status and access to health care resources (Canedo et al., 2018; Williams et al., 2019). A growing body of research, however, suggests that these health disparities develop over the lifespan, with pathogenic processes starting in childhood but not manifesting clinically until middle and late adulthood (Miller et al., 2011).

In addition, although type 2 diabetes and other chronic diseases tend to affect adults in middle age or later, recent statistics suggest these illnesses are occurring at much younger ages, particularly for African Americans. Rates of type 2 diabetes and its preclinical state of insulin resistance have increased dramatically among African American young adults (Centers for Disease Control and Prevention, 2017; Wilmot & Idris, 2014), and African Americans, on average, experience diabetes 10 years earlier than their Caucasian peers (Thorpe et al., 2016). Moreover, individuals from low-resource backgrounds will also experience earlier onset of diabetes (Williams et al., 2016). Consequently, for low-income African Americans in the rural southern United States, the incubation period for chronic disease has been compressed (Jackson et al., 2010) and these trends, if sustained, will

further shorten the life expectancy for this population. Unfortunately, prospective research following individuals from childhood into adulthood is exceedingly scarce, particularly with African American samples, despite the need to establish an empirical basis for these proposed associations and their accompanying risk mechanisms.

Drawing from a prospective study of 342 African American youth spanning nearly two decades, the current study was designed to address this need by investigating the longitudinal association between family poverty during adolescence and insulin resistance (a precursor to type 2 diabetes) in young adulthood. To provide insight into mechanisms that underlie this association, the current study also investigated the role of low perceived life chances and immune cell aging as mediators in the developmental cascade linking poverty to insulin resistance among rural African Americans.

BIOLOGICAL WEATHERING AND HEALTH DISPARITIES AMONG AFRICAN AMERICANS

To help explain the developmental origins of racial health disparities, life-course perspectives on health emphasize the ways in which socioeconomic and other stressors experienced during childhood "get under the skin" to presage health disparities in adulthood (Pearlin et al., 1997; Umberson et al., 2010). As summarized in Geronimus's influential "weathering" hypothesis (2006), wear and tear from chronic stress, beginning in childhood and continuing throughout the life course, weathers multiple physiological systems. This weathering, in turn, initiates a cascade of physiological processes implicated in the deterioration of these systems, including the premature aging of cells, in a manner that eventually leads to disease and a shortened life expectancy overall.

In light of this hypothesis, developmental and health psychologists have sought to develop metrics for quantifying biological weathering. Previously employed indices include allostatic load (Repetti et al., 2011), telomere length (Brown et al., 2017), inflammatory activity (Simons et al., 2016), and, of central importance to this study, cellular aging (Grant et al., 2017; Quach et al., 2017; Toperoff et al., 2015). Derived from the DNA methylation of cells, cellular (or epigenetic) aging reflects the disparity between an individual's biological age and that individual's chronological age (Horvath, 2013; Horvath et al., 2014). This biomarker has been validated in cells from more than a dozen tissues and forecasts heightened risk for diabetes (Grant et al., 2017) and all-cause mortality (Horvath & Raj, 2018). Research has also highlighted the relevance of accelerated cellular aging during adolescence for later health outcomes (Huang et al., 2019).

With respect to insulin resistance, recent research has shown the relevance of childhood adversity in accounting for racial disparities. Fuller-Rowell et al. (2019), for instance, found a composite risk index of childhood adversity was positively associated with insulin resistance in middle-aged adults and, when accounted for, attenuated 18% of the racial differences in insulin resistance. This study also found support for inflammation and cortisol as mediating variables, suggesting the potential for stress-related biological mechanisms that underlie this association.

CONTEXTUAL PRECURSORS AND PSYCHOSOCIAL MECHANISMS OF CELLULAR AGING

Contextual precursors

Additional research has begun to identify contextual factors that influence rates of cellular aging. Early findings suggest that social environments in childhood and adolescence characterized by threat and disadvantage (e.g., exposure to violence, psychosocial adversity, traumatic stress) are associated with accelerated cellular aging (Jovanovic et al., 2017; Lawn et al., 2018; Wolf et al., 2018). Prior studies involving the sample in the current study have documented associations of racial discrimination and parental depression with accelerated aging of immune cells (Brody, 2016; Brody, Yu, Chen, et al., 2016). Although informative, prospective research assessing the childhood environment, biological weathering in late adolescence, and clinical (or pre-clinical) markers of disease in emerging and young adulthood remained limited (and, to our knowledge, non-existent for studies with low-resource African American samples).

The present study tested the hypothesis that accelerated aging of immune cells would mediate the association between childhood poverty and insulin resistance. Our specific consideration of immune cells as a mechanism of interest for insulin resistance is logical given prior evidence that low-grade inflammation interferes with insulin signaling and glucose regulation more generally (and vice versa; see Lackey & Olefsky, 2016; Odegaard & Chawla, 2013).

Psychosocial mechanisms

Prior research highlights the ways in which exposure to persistent and repetitive poverty-related stressors affect youths' cognitive orientations in areas that have implications for mental and behavioral health (McDade et al., 2011; McLoyd et al., 2009; Steinberg et al., 2009). However, corresponding attention to the association between these cognitive orientations and biological processes involved in disease is rare, despite some evidence that beliefs, particularly about the future, forecast health outcomes. For instance, diminished purpose in life, fatalistic tendencies, and a negative future orientation all have been associated

with higher allostatic load (Bourdon et al., 2020; Zilioli et al., 2015). Informed by this literature, the current study considered the possibility that the perception of fewer life chances—a cognitive orientation more common among disadvantaged children (Jessor et al., 1990)—would operate as a mediator linking family poverty to cellular aging.

METHOD

Participants

Data for the study were drawn from the Strong African American Families Healthy Adult Project (SHAPE; Brody et al., 2013). Starting in 2001, SHAPE enrolled 667 Black children in fifth grade (M_{age} = 11.2 years, SD = 0.3) along with their primary caregivers. Families resided in rural counties in Georgia, where poverty rates are among the highest in the United States. Economically, these households can be characterized as working poor. When the youth had reached ages 19 to 20, a subgroup of 500 was randomly selected for a substudy of DNA methylation; 399 (79.8%) provided a blood sample. When participants were 25, 27, and 29 years old, blood was drawn from 391 (age 25), 388 (age 27), and 327 (age 29) participants, respectively, from which insulin resistance (IR) was quantified. A total of 419 participants provided blood samples for at least 1 year from ages 25 to 29. The sample for the present study was composed of 342 participants (140 men and 202 women) from whom blood was drawn at both age 20 and at least one time between ages 25 and 29. Compared with the original study cohort, the analytic sample had a higher percentage of female participants (59.1% vs. 52.8%) and experienced more years living in poverty (Ms = 2.46 vs. 2.16); the samples were similar to the other study variables (for more sample selection information, see Supplemental Material and Brody et al., 2020).

Procedures

All data were collected in participants' homes using a standardized protocol. African American field researchers visited families' homes to administer computer-based interviews, allowing respondents to answer sensitive questions privately. An African American field researcher who was also a certified phlebotomist went to each participant's home in the morning to draw a fasting blood sample from which cellular aging at age 20 and IR at ages 25, 27, and 29 was quantified. Each family was paid $100 for each wave of data collection across adolescence. Young adult participants were paid $160 for blood draws and psychosocial assessments starting at age 19. Informed consent forms were completed at all data collection points. The University of Georgia's Institutional Review Board reviewed and approved all study procedures.

Measures

Years living in poverty

When participants were 11, 12, 13, 16, 17, and 18 years of age, caregivers provided data on their families' income-to-needs ratios based on family size; these data were used to compute household poverty. Poverty statuses at the six assessment waves were summed to determine the number of years participants lived below federal poverty standards.

Perceived life chances

Perceived life chances at ages 16–18 were assessed with the 10-item Perceived Life Chances scale (Jessor et al., 1990). Each item was rated on a Likert-type scale ranging from 1 (*very low*) to 5 (*very high*). Example items include, "what are the chances that you will go to college?" and "what are the chances that you will have a job that pays well?" Alphas across waves ranged from .88 to .91. Youths' ratings across the three waves of assessments were averaged.

Accelerated immune cell aging

When participants were age 20 years, a certified phlebotomist went to each of their homes to draw blood. Peripheral blood mononuclear cells (PBMC) were isolated through density-gradient centrifugation, and DNA methylation was subsequently assessed with the Illumina Infinium (Sequenom, Inc.) HumanMethylation450 Beadchip. The beta value at each CpG locus was calculated as the ratio of the intensity of the methylated probe to the sum of intensities of the methylated and unmethylated probes. We then assessed accelerated cellular aging using Horvath and Raj's (2018) skin and blood clock. To transform Horvath's skin and blood clock into a measure of accelerated cellular aging, we regressed the clock on chronological age, resulting in a measure of accelerated cellular aging that is adjusted to correlate with chronological age at 0 (see Supplemental Material for additional details). A positive value on this variable indicates accelerated cellular aging in years.

Insulin resistance

At the ages 25, 27, and 29 assessments, a phlebotomist visited each participant's home in the morning to draw an overnight fasting blood sample. Blood was drawn into serum separator tubes (Becton, Dickinson and Company). Specimens were centrifuged on site at 1500×g for 20 min. Aliquoted serum glucose was measured photometrically using a UV test on a Roche/Hitachi Cobas c502 instrument,

and serum insulin was assayed in duplicate using a multiplex, electrochemiluminescent, immunoassay (Human Leptin/Insulin Kit K15164C; MesoScale Discovery) on a SECTOR Imager 2400A (MesoScale Discovery). The mean (standard deviation) for glucose and insulin were 91.00 (20.67) mg/dl and 114.02 (97.03) pmol/L, respectively.

IR was estimated according to the updated homeostasis assessment (HOMA2) model (Wallace et al., 2004). IR values were skewed and/or kurtotic, so we normalized their distributions with log-10 transformations. The logged values at three waves were then averaged (see Supplemental Material for additional details). Thus, in the current study, we treat IR as a continuous variable to examine its variations among participants and do not classify individuals on the basis of having elevated insulin resistance as defined by clinical criteria.

Self-report of diabetes status was not assessed and thus could not be considered (see Supplemental Materials for information on diabetes status based on fasting glucose levels).

Covariates

Covariates included sex, body mass index (BMI), and smoking at age 20 (given potential contributions to cellular aging and IR). As the SHAPE cohort was initially recruited for a randomized, controlled trial of a family-oriented prevention program, a dichotomous covariate reflecting intervention condition (treatment vs. control) was included in all models.

Plan of analysis

Pearson's and Spearman's correlation coefficients were calculated to examine the associations of IR with family poverty and cellular aging. Multiple linear regression models were executed to test the study hypotheses. The first model was designed to determine whether number of years living in poverty at ages 11–18 was associated with IR at ages 25–29. The second regression model was designed to determine whether accelerated cellular aging at age 20 would mediate the association between number of years living in poverty and IR. Finally, the third regression model was executed to determine whether perceived life chances at ages 16–18 would serve as a second mediator connecting number of years living in poverty at ages 11–18 with accelerated cellular aging at age 20, or with IR at ages 25–29.

All analyses were conducted using IBM SPSS 27 and the statistical macro package PROCESS (Hayes, 2012). For indirect effects, nonparametric bootstrapping was used to obtain the bias-corrected and accelerated confidence intervals (BCA) of parameter estimates for significance testing (Preacher et al., 2007). The parameter estimate was calculated 5000 times using random sampling with replacement to build a sampling distribution.

TABLE 1 Correlations and descriptive statistics among study variables

Variable	*n* (%) or *M* (*SD*)	Correlations						
		1	2	3	4	5	6	7
1. Sex, male	140 (40.9%)	—						
2. Intervention, SAAF	209 (61.1%)	−.031	—					
3. Years in poverty (ages 11–18)	2.459 (1.926)	−.037	.018	—				
4. Perceived life chances (ages 16–18)	46.136 (4.039)	−.228***	.027	−.201***	—			
5. Immune cell aging (age 20)	−0.018 (3.205)	.139*	−.059	.152**	−.171**	—		
6. Smoking (age 20)	0.102 (0.209)	.336***	−.036	.081	−.261**	.039	—	
7. BMI (age 20)	28.922 (8.630)	−.182**	.046	.090	.077	−.006	−.097	—
8. IR (ages 25–29)	0.513 (0.317)	−.326***	.063	.137*	.086	.110*	−.233***	.585***

Note: $N = 342$. *$p < .05$. **$p < .01$. ***$p < .001$.
Abbreviations: BMI, body mass index; IR, insulin resistance.

At ages 25 and 27, individuals self-reported medications currently being taken. Nine individuals (2.6% of sample) reported use of diabetes medication. The results of models excluding these nine individuals were nearly identical to those obtained using the full sample (see Supplemental Materials). Although containing elements of both, the present study was more confirmatory than exploratory as it sought to test directional hypotheses informed by the existing literature.

RESULTS

Bivariate correlations and descriptive statistics for the study variables are presented in Table 1.

Years living in poverty at ages 11–18 and IR at ages 25–29

Our initial analysis was designed to determine whether years living in poverty during adolescence was associated with IR during young adulthood. The results of the regression model revealed that years living in poverty at ages 11–18 was associated with IR at ages 25–29: $b = .016$, 95% CI [.002, .029], $p = .025$. These values indicate that for each additional year of adolescent poverty, HOMA values were 1.038 higher in young adulthood.

Immune cell aging at age 20 mediated the association between years living in poverty at ages 11–18 and IR at ages 25–29

The second regression model was executed to determine whether immune cell aging at age 20 would mediate the association between number of years during adolescence spent living in poverty and adult IR. The results (see Figure 1) revealed that accelerated cellular aging at age 20 mediated the association between years living in poverty at ages 11–18 and IR at ages 25–29. A greater number of years living in poverty at ages 11–18 was associated with faster immune cell aging at age 20 ($b = .259$, 95% CI [.082, .436], $p = .004$), which in turn was associated with higher IR levels at ages 25–29 ($b = .013$, 95% CI [.004, .021], $p = .003$). Thus, for each additional year of poverty, immune cell aging increased by 0.26 years. Multiplying these coefficients yielded an indirect mediated effect of .003 (95% bootstrapped CI of [.0004, .009]). The presence of the mediator, immune cell aging, reduced the direct effect of years living in poverty on IR, from $b = .016$, 95% CI [.002, .029], $\Delta R^2 = .009$, $p = .025$ to $b = .012$, 95% CI [−.001, .026], $\Delta R^2 = .005$, $p = .077$. Thus, after controlling for immune cell aging, the variance of IR that was explained by poverty was reduced by 44.4%.

Perceived life chances at ages 16–18 mediated the association between years living in poverty at ages 11–18 and immune cell aging at age 20

We further examined the hypothesized mediation effect of perceived life chances by including the pathways in which years living in poverty at ages 11–18 predicted perceived life chances at ages 16–18 (Path A1), which, in turn, predicted accelerated cellular aging at age 20 (Path B1). This mediation model is presented in Figure 2. As predicted, the negative coefficient for Path A1 indicates that a greater number of years living in poverty at ages 11–18 was associated with the perception of fewer life chances at ages 16–18 ($b = -.435$, 95% CI [−.646, −.224], $p < .001$). The negative coefficient for Path B1 indicates that the perception of fewer life chances at ages 16–18 was associated with faster immune cell aging at age 20 ($b = -.106$, 95% CI [−.194, −.018], $p = .018$). Furthermore, the indirect pathway from years living in poverty to IR through perceived life chances and immune cell aging was statistically significant (indirect effect = .0006, 95% CI [.0001, .0012]). Nevertheless, years living in poverty remained associated with cellular aging even after accounting for perceived life chances, as the significant Path A2 coefficient indicates. Thus, perceived life chances partially mediated the association between years living in poverty and immune cell aging.

DISCUSSION

Poverty and other contextual stressors endemic to the rural southern United States have long been theorized

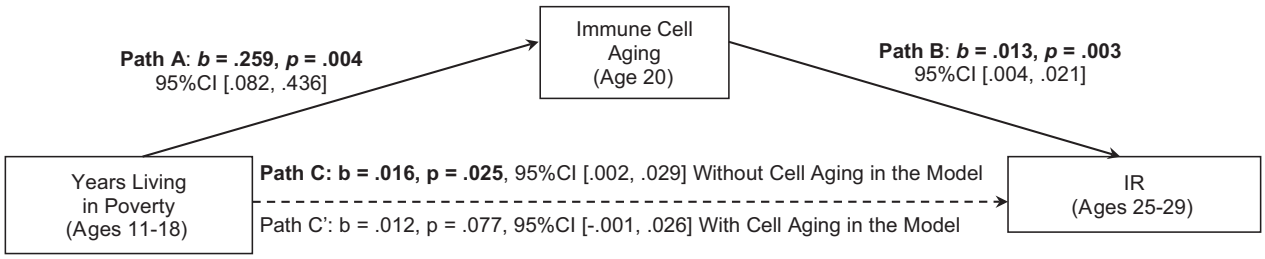

FIGURE 1 Immune cell aging as a mediator of the relation between years living in poverty and IR. Control variables not shown. Unstandardized coefficients with 95% confidence intervals (CI) are presented. $N = 342$

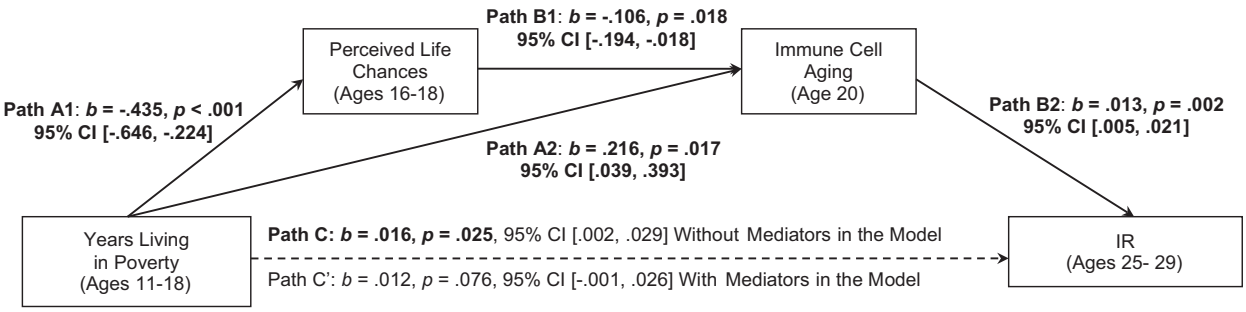

FIGURE 2 Perceived life chances and immune cell aging as mediators of the relation between years living in poverty and IR. Control variables not shown. Unstandardized coefficients with 95% confidence intervals (CI) are presented. $N = 342$

to be factors contributing to racial health disparities (Brody, Yu, & Beach, 2016). Prospective research investigating this proposed association is exceedingly sparse, particularly with disadvantaged populations. With a sample of rural African American youth and parents followed over nearly two decades, the current study addressed this gap by providing some of the first empirical evidence to document (a) the prospective association between childhood poverty and insulin resistance in young adulthood, and (b) psychosocial and biological mechanisms that contribute to this association. Findings provide support for life-course perspectives on health and the developmental origins of chronic disease (Berens et al., 2017; Miller et al., 2011).

The results were consistent with Geronimus's weathering hypothesis (2006) with a scenario wherein immune cell aging partially mediated the association between adolescent poverty and IR in adulthood. Poverty-related variations in immune cell aging were evident at age 20, suggesting this biomarker may be a sensitive indicator of weathering at younger ages when variability in clinical endpoints (e.g., metabolic syndrome, cardiovascular disease) is limited (also see Sumner et al., 2019). Additional studies, however, are needed that have multiple waves of cellular aging data to evaluate this possibility. With a single measure of immune cell aging at age 20, we cannot be certain whether variation in this biomarker preceded or followed exposure to childhood poverty, or whether there is a causal association between these variables. Also, although the current results highlight the relevance of biological weathering, unexplained variability suggests that future research should examine other factors, such as adult socioeconomic position and adult stress exposure, which could contribute to the association between childhood adversity and poor health in adulthood (see Turner et al., 2016).

This study also adds to the literature on psychosocial precursors to cellular aging, highlighting a potential intermediating role for perceived life chances. Having well-established associations with behavioral and mental health outcomes (Barton et al., 2015; Griffin et al., 2004; Hawkins et al., 1998), the current study is one of the first to examine the association between perceived life chances and accelerated biological aging. Although novel, this finding is consistent with previous research on the association between less hopeful and more fatalistic cognitive orientations and physical health (Bourdon et al., 2020; Zilioli et al., 2015).

Future research is needed that can identify moderators of this cascading pathway. Supportive parenting has emerged as a promising moderator, in human and animal research (Hostinar et al., 2014) as well as family-based interventions (Brody, Miller, Yu, et al., 2016; Miller et al., 2014).

Several limitations of this study should be noted. First, our study employed a measure of biological weathering consistent with the Horvath approach to cellular aging. Although there is strong empirical support for this method, various other methylation and nonmethylation-based approaches have been identified as means of assessing biological weathering (Ferrucci et al., 2020). Second, because families began the study when youth were age 11, we were unable to examine the extent to which stressors at even earlier developmental stages may contribute to the developmental origins of chronic disease. Such prospective work is important and, given the findings on the long-term influence of prenatal environmental factors (Gillman, 2005), studies beginning at the earliest stages of development are warranted. Related to this, there were no measures of biological health in childhood, so a scenario of reverse causality cannot be ruled out. In addition, for African Americans, the long-term effects of early life racial discrimination (ELRD) on physical and mental health remains a key area of investigation (see Thomas Tobin & Moody, 2021). Third, homeostasis assessment provides an indirect measure of IR and is not a current standard diagnostic technique (i.e., oral glucose tolerance test or continuous glucose monitoring). Fourth, the direct and indirect effects observed in the present study were small in magnitude, suggesting that additional mechanisms and processes contribute to the association between poverty and IR. As such, cellular aging should be interpreted as representing one of the multiple potential mechanisms linking poverty to IR. Future research exploring this area could consider behavioral

risk factors (e.g., diet, sleep) as possible additional mechanisms (McDade et al., 2011). Fifth, since most of sample was in the normal range of IR, analyses comparing clinically relevant versus non-clinically relevant levels of elevated IR were not possible. Finally, our study focused on youth from rural African American families, many of whom were living with considerable socioeconomic disadvantage and chronic stress. Given the unique characteristics of this sample, we are hesitant to speculate about the generalizability of these findings, but we encourage researchers with access to ongoing longitudinal studies to consider these questions in their research.

In summary, our findings suggest that youths' exposure to family poverty across adolescence is associated with higher levels of IR in young adulthood, with perceived life chances and cellular aging mediating this association. The study incorporated multiple strengths, including having nearly two decades of data from a high risk, understudied population, as well as data collection that occurred across multiple informants and multiple levels (e.g., self-report, intravenous blood collection). The current findings underscore the value of a developmental perspective to understand the onset of chronic disease.

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