Self-control forecasts better psychosocial outcomes but faster epigenetic aging in low-SES youth

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There are persistent socioeconomic disparities in many aspects of child development in America. Relative to their affluent peers, children of low socioeconomic status (SES) complete fewer years of education, have a higher prevalence of health problems, and are convicted of more criminal offenses. Based on research indicating that low self-control underlies some of these disparities, policymakers have begun incorporating character-skills training into school curricula and social services. However, emerging data suggest that for low-SES youth, self-control may act as a “double-edged sword,” facilitating academic success and psychosocial adjustment, while at the same time undermining physical health. Here, we examine this hypothesis in a five-wave study of 292 African American teenagers from rural Georgia. From ages 17 to 20 y, we assessed SES and self-control annually, along with depressive symptoms, substance use, aggressive behavior, and internalizing problems. At age 22 y, we obtained DNA methylation profiles of subjects’ peripheral mononuclear cells. These data were used to measure epigenetic aging, a methylation-derived biomarker reflecting the disparity between biological and chronological aging. Among high-SES youth, better mid-adolescent self-control presaged favorable psychological and methylation outcomes. However, among low-SES youth, self-control had divergent associations with these outcomes. Self-control forecasted lower rates of depressive symptoms, substance use, aggressive behavior, and internalizing problems but faster epigenetic aging. These patterns suggest that for low-SES youth, resilience is a “skin-deep” phenomenon, wherein outward indicators of success can mask emerging problems with health. These findings have conceptual implications for models of resilience, and practical implications for interventions aimed at ameliorating social and racial disparities.

Significance

Most childhood outcomes pattern by socioeconomic status (SES). Children from low-SES families complete less education, have worse health, and are convicted of more crimes. To ameliorate these disparities, policymakers are incorporating character-skills training into school curricula and social services. Among other goals, these programs attempt to improve self-control, or the ability to resist temptations that interfere with long-term aspirations. However, data suggest that self-control has unforeseen consequences for the health of low-SES youth. Here, we follow 292 African American teenagers as they transition into adulthood. Among low-SES youth, self-control forecasted better psychosocial outcomes, including less depression, substance use, and aggression. However, it also forecasted more rapid cell aging, highlighting the potential health costs of successful adjustment for disadvantaged youth.


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individuals had achieved sustained academic success—they had graduated from high school and were now attending college—despite living in challenging neighborhoods with concentrated poverty. Compared with other participants, this cohort had lower rates of cigarette, alcohol, and marijuana use at age 20 y. However, this resilience was only “skin deep.” Despite academic success and healthy lifestyles, these youth showed relatively poor cardiometabolic health at age 20 y, as reflected in obesity, blood pressure, and stress hormones (19).

These findings suggest that self-control may act as a “double-edged sword” in low-SES youth, facilitating academic success and psychosocial adjustment, while at the same time undermining cardiometabolic health. What could explain these divergent outcomes? Research shows that for low-SES youth, particularly those of African American descent, achieving normatively favorable outcomes poses intense self-regulatory demands (20–22). Because such demands result in sustained activation of stress hormone systems (18, 19, 23–25), we reasoned they would prematurely age bodily tissue through a process known as weathering (26). Here, we test this hypothesis in a new sample of rural African American youth, who were followed across the transition from adolescence to adulthood. To clarify the mechanisms by which skin-deep resilience develops, we focus on aging of immune cells, using an epigenetic biomarker derived from DNA methylation. This epigenetic clock has been validated in cells from over a dozen tissues and reflects the disparity between biological and chronological age. Using this metric, faster aging rates have been documented in tumor-derived cells from over 20 cancers, as well as liver biopsies from obese patients (27–29). Faster epigenetic aging also presages higher risks for all-cause mortality (30).

Results

The subjects were part of a larger study, Adults in the Making (AIM), which included five waves of assessment (31). As Table 1 shows, the sample consisted of adolescents from predominately working-poor families; 65% of the subjects lived in single-parent households, and 45% had incomes below the federal poverty threshold. Fewer than 10% of subjects were from households where a caregiver had a bachelor’s degree. Annually from ages 17 to 19 y, subjects completed validated measures of self-control, which were supplemented by caregiver reports. From these data, we generated a composite indicator of “Self-Control” by aggregating standardized values across assessments, respondents, and instruments (see details in Methods). At each wave, we also

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Fig. 1. Self-control’s association with epigenetic age acceleration varies according to SES. (A) Depiction of estimated Hannum values at lower (−1.5 SD) and higher (+1.5 SD) levels of self-control and socioeconomic disadvantage. (B) Depiction of individual data points and regression slopes for subjects who are more (≥1.5 SD above sample mean) (Left), medium (−1.49 to +1.49 SD) (Center), and less (less than or equal to −1.5 SD) (Right) disadvantaged relative to the sample distribution.
gathered socioeconomic data from caregivers and formed a composite that assigned one point for each of six indicators of disadvantage (see details in Methods). Psychosocial outcomes were assessed annually from ages 17 to 20 y, via youth reports of depressive symptoms and substance use, as reflected in cigarette, alcohol, and marijuana consumption and parent reports of aggressive behavior and internalizing symptoms. Approximately 2 y later, when subjects were an average of 22 y old, we collected blood to measure epigenetic aging. DNA was extracted from peripheral blood mononuclear cells (PBMCs) and hybridized to HumanMethylation450 BeadChips following the manufacturer’s protocol; β values were used to generate two metrics of epigenetic aging, based on formulas provided by Horvath (27) and Hannum (28). All data are contained in Dataset S1.

Initially, we tested hypotheses in linear regression equations, where outcomes were predicted from three blocks of variables: covariates, main effects of Self-Control and “Disadvantage” at ages 17–19 y, and the interaction of these variables. In all equations, sex was modeled as a covariate, as was receipt of the AIM intervention (which did not affect behavioral or epigenetic outcomes reported here; P > 0.37). Consistent with previous research, there were main effects of adolescent self-control on all age 20 y psychosocial outcomes, even after accounting for age 17 y values (SI Appendix, Table S1; P = 0.0005–0.02; ΔR² = 0.01–0.04). To the extent that they had better self-control in mid-adolescence, subjects experienced declines in depressive symptoms, internalizing problems, substance use, and aggressive behavior as they transitioned into adulthood. These patterns were consistent across strata of disadvantage, except in the case of substance use, where there was a significant interaction (P = 0.05). We used standard methods to clarify the nature of this interaction (32), plotting estimated age 20 y substance use by lower (−1.5 SD) and higher (+1.5 SD) levels of Self-Control and Disadvantage, after partialing out covariates. SI Appendix, Fig. S1 shows that as disadvantage increased, self-control’s association with substance use became more negative.

We repeated these analyses with indicators of epigenetic age acceleration. SI Appendix, Table S2 shows the significant Self-Control × Disadvantage interactions for both metrics (P = 0.003–0.004; ΔR² = 0.03–0.04). We plotted these interactions in Figs. 1 and 2 and computed simple slopes, again using standard methods (32). For subjects who were less disadvantaged, the patterns mirrored behavioral outcomes. In other words, better mid-adolescent self-control presaged less epigenetic aging of PBMCs by young adulthood. However, as in previous reports of skin-deep resilience, psychosocial and biomedical outcomes diverged among the sample’s more disadvantaged youth. For them, better mid-adolescent self-control presaged more epigenetic aging of PBMCs by young adulthood. At the sample’s typical level of disadvantage, self-control and epigenetic age were unrelated.

Although these patterns are suggestive of skin-deep resilience, they do not address convergence of outcomes at the individual level. In other words, they do not answer the following question: Are the same disadvantaged youth, with high self-control, having relatively good psychosocial and relatively poor epigenetic outcomes? To address this question, we performed latent class growth analyses (33), using data from ages 17 to 19 y to sort youth into categories based on Disadvantage and Self-Control. Model-fit indices suggested a parsimonious four-group solution (SI Appendix, Tables S3 and S4). The solution first stratified the sample into groups who

![Fig. 2.](image-url)
were more or less disadvantaged and then into groups in whom self-control was either stably low or high increasing (i.e., grew steadily across adolescence). The percentage of subjects in each group was 14% (more disadvantaged/low self control), 27% (more disadvantaged/high self control), 26% (less disadvantaged/low self control), and 33% (less disadvantaged/high self control).

When the groups were compared, results mirrored regression analyses. Specifically, better mid-adolescent self-control pre-saged favorable young adult psychosocial outcomes across the sample (SI Appendix, Table S5). On all four of psychosocial outcomes—depressive symptoms, internalizing problems, substance use, and aggressive behavior—the groups high in self-control fared better (Fig. 3), as reflected by smaller intercept values and/or more favorable trajectories. However, for epigenetic aging, self-control’s effects depended on the level of disadvantage ($F = 3.48–3.89$; $P = 0.01–0.02$; SI Appendix, Table S6 and Fig. 4). Among more disadvantaged subjects, better self-control was associated with a 1.46- to 2.27-y acceleration of epigenetic aging (by Hanum and Horvath methods, respectively). By contrast, among less-disadvantaged subjects, better self-control was associated with a 0.27- to 2.14-y deceleration (by Horvath and Hanum methods, respectively).

**Discussion**

Previously, we reported a pattern of skin-deep resilience in low-SES African American youth, wherein self-control portends favorable psychosocial outcomes but worse cardiometabolic health status (18, 19). Here, we extend these findings to a new cohort of adolescents making the transition into adulthood and highlight premature aging of PBMCs as a mechanism that could underlie this phenomenon. How this process unfolds remains unclear. We considered the role of obesity, because youth who exhibit skin-deep resilience have greater body mass (18, 19), which in turn relates to faster epigenetic aging (29). However, mediation analyses did not yield support for this scenario nor one wherein these youth have more general life stress (SI Appendix, Table S7). Nonetheless, the patterns here converge with evidence that relentlessly pursuing goals can undermine health (34), particularly when structural forces like discrimination impede progress toward those goals (20, 23). As disadvantaged youth strive for favorable life outcomes, they have substantial barriers to overcome and competing demands to balance, including resource-deprived schools, family obligations, and managing social identity threats (35). These challenges are particularly salient for African Americans.

Navigating these challenges requires intense and persistent self-control (36), which is metabolically and behaviorally demanding to sustain. Acutely, exerting self-control triggers the release of stress hormones (24, 25) and erodes the ability to resist tempting stimuli, like high-fat food (37). These effects subside when people can suspend willpower and indulge in restorative activities. However, for lower-SES youth, opportunities for respite are likely to be infrequent. To achieve upward mobility, these youth must overcome multiple obstacles and often do so with limited support from their schools, peers, and families (12). Even if they succeed, these youth may go on to experience alienation in university and workplace settings and discrimination if they are African American. Collectively, these experiences seem likely to cause persistent activation of stress-response systems, in particular the sympathetic nervous system and hypothalamic–pituitary–adrenocortical axis. The hormonal products of these systems, glucocorticoids and catecholamines, are elevated in youth who exhibit skin-deep resilience (18, 19). These hormones also can modify the chromatin architecture of leukocytes by altering the enzymatic activity of DNA methyltransferases, as well as histone deacetylases and acetyltransferases (38).

Future research should explicitly test these mechanistic hypotheses. We are unable to do so here because AIM did not assess stress hormone output or obtain methylation profiles during adolescence. A follow-up study with multiple waves of psychosocial, hormonal, and epigenetic data would be ideally

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**Fig. 3.** Trajectories of psychosocial adjustment from ages 17 to 20 y as a function of latent class grouping on self-control and socioeconomic disadvantage. Outcomes are substance use (A), depressive symptoms (B), aggressive behavior (C), and internalizing problems (D).

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suited to identifying mechanisms. In future research, it also will be important to establish the demographic circumstances in which skin-deep resilience develops. To date, this work has focused exclusively on low-SES, African American youth, living in rural areas of the South. As a consequence, it remains unclear whether vulnerability to skin-deep resilience accrues by virtue of race, class, geography, or interaction(s) of these factors. Further research is also needed to clarify the nature and meaning of self-control in the population of interest here. Self-control is a multidimensional construct, and the questionnaires we used capture elements of industriousness, seriousness, impulsivity, and perseverance. To understand the origins of skin-deep resilience, research must determine which of these characteristics presage health problems. Laboratory observations would be especially valuable in this regard, providing researchers with an opportunity to catalog specific behaviors and their relationship with health outcomes. These observations also would clarify whether our questionnaires are capturing the same phenomenon across the sample. It is possible these scales capture distinct behaviors and/or competencies in disadvantaged youth who go on to have better vs. worse health outcomes. Finally, additional research is needed to understand the causes and effects of epigenetic age acceleration, particularly in youth. Although epigenetic clocks are widely believed to reflect premature aging (27–30), they also might conceivably tap cellular maturation in younger populations. If so, our findings would suggest that self-control forecasts precocious development in at-risk youth, rather than weathering. Viewed in light of our previous findings on cardiometabolic risk (18, 19), we see this as an unlikely possibility but one that should be considered in future research.

Since 2000, the prevalence of childhood poverty in America has increased and so have the magnitude of socioeconomic disparities in many aspects of youth development (39). These trends are fuelling concerns about public health, human capital, and economic security in the coming decades (15, 17). As one way to counteract these effects, policymakers are drawing on self-control research and incorporating character-building interventions into school curricula and government programs. Although these interventions will likely improve the educational and psychosocial outcomes of low-SES youth, the accumulating data on skin-deep resilience suggest the potential for unintended health consequences. Ironically, the children most vulnerable to such consequences—who from disadvantaged families—already have disproportionately more health problems. Thus, to maximize return on human-capital investments, policymakers should broaden character-building programs to include health education and, where possible, monitoring and treatment of emerging medical problems. This approach could mitigate health problems that prevent upwardly mobile youth from realizing their full potential. More broadly, these findings challenge our understanding of what it means to be resilient. Current thinking suggests that if low-SES youth do well in school and stay out of trouble, they have overcome disadvantage. As we show, that is only partially accurate.

**Methods**

**Sample.** AIM was a randomized trial focused on alcohol- and substance-use prevention in African American teenagers who were making the transition to adulthood (31). It recruited 496 youth from public schools in six rural counties in Georgia. Subjects were eligible if they were assigned to AIM or control condition. The intervention consisted of six weekly group meetings held at community facilities, with separate parent and youth skill-building sessions and a family curriculum. The University of Georgia’s Institutional Review Board approved AIM’s protocol. At each wave, parents gave written consent and youths gave written assent or consent.

The intervention did not influence any of the psychosocial or epigenetic outcomes (P > 0.37; SI Appendix, Tables S1 and S2), but receipt of AIM is nonetheless controlled in all analyses. Of the 496 Wave 1 participants, 424 provided self-report data at Wave 4 (age 20 y), a retention rate of 85.5%. Of these youth, 292 (88.9%) agreed to the blood draw at age 22 y and constitute the analytic sample. Using independent t tests and χ² analyses, we compared these youth with the broader sample and found no differences on major study variables listed in Table 1. There was one exception: missing methylation data were more common in male versus female subjects, χ² (1) = 5.76, P = 0.02. Thus, sex was controlled in all analyses.

**Psychosocial Assessments.** Self-control and socioeconomic disadvantage were assessed annually from ages 17 to 19 y. At each wave, subjects completed the 11-item Self-Control Inventory (40) and 23-item Self-Regulation Questionnaire (41). To supplement self-reports, we had a caregiver describe each subject’s disposition on the Self-Control Inventory. Both of these scales have been extensively validated and showed high internal consistency (α = 0.87–0.96), cross-wave stability (r = 0.54–0.63), and parent-child concordance here (r = 0.30). These scales also were strongly intercorrelated (average r = 0.76). Accordingly, we formed a form of a Self-Control composite by aggregating standardized scores across assessments, respondents and waves. At each wave, we also gathered SES data from caregivers and formed a disadvantage composite that assigned one point for each of six risk indicators: household income below the federal poverty line, receipt of Temporary Assistance for Needy Families, caregiver report of income as insufficient to meet all needs, and primary caregiver without high school education or current employment.

Psychosocial outcomes were assessed annually from age 17 to 20 y. At each wave, we obtained youth self-reports of depressive symptoms (42) and substance use (31). For the latter, subjects reported their past-month cigarette, alcohol, and marijuana use and the number of times they drank alcohol to excess. Responses were made on seven-point scales with the categories 0, 1–3, 5–6, 7–9, 10–19, 20–39, and 40+. Responses to these four items were summed to form a substance use composite. Simultaneously, we obtained caregiver reports of youth aggressive behavior and internalizing symptoms (43). Cronbach’s α values on these instruments ranged from 0.82–0.85.

**DNA Methylation.** When youth were age 22 y, phlebotomists went to their homes and collected antecubital blood. PBMCs were isolated through density-gradient centrifugation (Ficoll-Paque Media PM 400; GE Healthcare). Genomic DNA was extracted with Qiagen DNA Mini Kits, and quality was verified on an Agilent 2100 Bioanalyzer. Methylation profiling was then conducted by the University of Minnesota’s Genome Center, following the manufacturer’s protocol for the Illumina HumanMethylation 450 BeadChip. The resulting data were inspected for complete bisulfite conversion, and average β values for each
targeted CpG residue were determined using the Illumina Genome Studio Methylation Module. Version 3.2; β values were calculated as the ratio of methylated to unmethylated probes, ranging from 0 (entirely unmethylated) to 1 (fully methylated). The resulting data were then cleaned using a Perl-based algorithm (44) to remove those β values with detection P values, an index of the likelihood that the observed sequence represents random noise, that were greater than 0.05. Nearly all probes (99.76%) yielded reliable data by this criterion.

Epigenetic Age Acceleration. Two epigenetic aging metrics have been proposed, which use distinct targets, covariates, and formulas. Horvath's clock was estimated with a publicly available R script, which aggregates methylation values from 335 CpG sites (27). Hannum's clock was estimated by summing weighted methylation values from 71 CpG sites, using coefficients he validated for PBMCs (28).

Latent Class Growth Analyses. To evaluate the convergence of psychosocial and epigenetic outcomes, we performed latent class growth analyses (33). First, to characterize trajectories of self-control and socioeconomic disadvantage from ages 17 to 19 y, we estimated a three-wave latent growth curve model with parallel outcomes. Linear models were fit with four individual growth parameters: two intercept parameters representing self-control and disadvantage at age 17 y and two linear slope parameters representing changes in these outcomes through age 19 y. Next, we used latent class growth analysis to estimate person-specific intercepts and trajectories and then clustered subjects into groups exhibiting similar patterns.

Mediation Models. To examine whether body mass or life stress might operate as pathways underlying skin-deep resilience, we estimated a series of mediation models (45). Body mass was measured during a home assessment and calculated as weight in kilograms divided by the square of height in meters. Life stress was assessed annually from age 17 to 19 y with an event checklist. Youth reported whether each of 12 events (e.g., death of a friend, parental divorce, serious injury) had occurred during the past 6 mo. The average count across waves was used to represent life stress.

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Analyses were conducted using Mplus Version 7.2. Fit indices were obtained for models with two to six classes (SI Appendix, Table S3). Lower Akaike Information Criterion and Bayesian Information Criterion scores represent better-fitting models, whereas higher entropy scores reflect greater classification accuracy. To select a final solution, we considered these fit indices, as well as theoretical parsimony and the size of resulting subgroups. In view of these criteria, a four-group model was selected, as detailed in Results. Next, we estimated a series of multigroup latent growth models, comparing the four groups’ trajectories of psychosocial outcomes from ages 17 to 20 y (SI Appendix, Table S5). Because epigenetic age acceleration was only measured at age 22 y, we used univariate ANOVAs to compare the groups with respect to this measurement (SI Appendix, Table S6).