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Losing Years Doing Time: Incarceration Exposure and Accelerated Biological Aging among African American Adults

Mark T. Berg[1], Ethan M. Rogers[1], Man-Kit Lei[2], and Ronald L. Simons[2]

Abstract

Research suggests that incarceration exposure increases the prevalence of morbidity and premature mortality. This work is only beginning to examine whether the stressors of the incarceration experience become biologically embedded in ways that affect physiological deterioration. Using data from a longitudinal sample of 410 African American adults in the Family and Community Health Study and an epigenetic index of aging, this study tests the extent to which incarceration accelerates epigenetic aging and whether experiences with violence moderate this association. Results from models that adjust for selection effects suggest that incarceration exposure predicted accelerated aging, leaving formerly incarcerated African American individuals biologically older than their calendar age. Direct experiences with violence also exacerbated the effects of incarceration. These findings suggest that incarceration possibly triggers a stress response that affects a biological signature of physiological deterioration.

Keywords

African Americans, epigenetic aging, health, incarceration

Mass incarceration has increasingly been recognized as a pressing public health challenge. Nearly 6.5 million adults—1 in 40—are under correctional supervision in the United States (Maruschak and Minton 2020). More than 2.1 million are currently serving sentences in prisons and jails. An additional 4.9 million individuals in the population were formerly imprisoned (Shannon et al. 2017). This decades-long expansion of the penal system has occurred without the structures in place to facilitate reintegration. Although the policies of mass incarceration have been far-reaching, their imprint has been concentrated among marginalized groups. Black adults account for 33% of individuals incarcerated in prisons—almost triple their share of the adult population—and they are incarcerated at rates that are three to five times higher than whites (Carson 2020; Zeng 2020). Noting these disparities, some scholars have argued that incarceration is so concentrated among disadvantaged groups that it "has become a normal stage of the life course" (Wildeman and Wang 2017:1465).

The deleterious effects of incarceration for individual well-being have been the subject of a growing body of interdisciplinary research at the intersection of medicine and the social sciences.

[1]University of Iowa, Iowa City, IA, USA
[2]University of Georgia, Athens, GA, USA

Corresponding Author:
Mark T. Berg, Department of Sociology and Criminology, University of Iowa, 400H North Hall, Iowa City, IA 52242, USA.
Email: mark-berg@uiowa.edu

Prisons and jails are known vectors for the transmission of chronic infectious diseases, such as tuberculosis and pneumonia (Hammett, Harmon, and Rhodes 2002). Another line of work developed in the psychoimmunology literatures maintains that incarceration triggers an upregulated, acute stress response that leads to physiological deterioration (Massoglia and Pridemore 2015). Empirical research indeed finds that formerly incarcerated persons have elevated levels of morbidity, especially for stress-related illnesses, and heightened risk for premature mortality (e.g., Boen 2020).

Despite important advances in recent years, this body of literature remains in a nascent stage owing to several substantive and methodological challenges (Massoglia and Remster 2019). First, the possibilities of selection bias and statistical confounding complicate efforts to isolate the causal effects of incarceration exposure on health outcomes. Second, the extent to which physical health is affected by witnessing or experiencing interpersonal violence while detained has received scant attention in the quantitative empirical research. This is an important omission because periods of confinement often involve unwanted, uncontrollable, and threatening experiences that conceivably govern the effects of incarceration on postrelease health (Porter 2019). As such, prior work has largely considered incarceration a homogeneous experience, thus ignoring the effects of varied experiences. It is therefore important to assess whether specific stress-inducing experiences during incarceration are differentially related to poor health.

Third, with few exceptions, most studies examine self-reported health outcomes, including perceived health, illnesses, and chronic conditions, rather than objective biomarkers obtained from independent assessments (Wildeman and Wang 2017:1468). Although useful, such indices of self-reported health provide limited information on the biological processes that precede the manifestations of disease. Asymptomatic conditions go unmeasured. Furthermore, subjective reports of health confound "physiological with psychological and emotional well-being" (Harris and Schorpp 2018:362). Because of the reliance on self-report instruments, the extent to which incarceration exposure becomes biologically embedded or "gets under the skin" is not well understood. Although objective biomarkers have been featured in some studies of incarceration and health, this work remains scarce (e.g., Boen 2020).

Accelerated aging is a "predominant risk factor for most diseases and conditions that limit health span" (Kennedy et al. 2014:709). The basis of healthy aging is a focus of modern medicine (Sinclair 2019). Newly developed epigenetic indices ("clocks") of biological aging have been shown to be powerful predictors of premature mortality and age-related diseases (Ryan et al. 2020). How social adversities affect biomarkers of accelerated aging has been the subject of burgeoning research on the global burden of age-related disease (Moffitt et al. 2017; Sinclair 2019). Despite its promise for understanding the biology of disease and illnesses, this work has not been integrated into the research on incarceration.

Using a longitudinal sample of African American adults from the Family and Community Health Study (FACHS), this study examines whether incarceration exposure and experiences with violence while incarcerated predict accelerated biological aging. Note that because the sample is limited to a sample of African American adults, the study does not assess whether incarceration contributes to racial disparities in aging. We posit that individuals exposed to incarceration will be biologically older than their numerical age and even more so among those who experienced violence while serving time. The study will inform efforts to understand the extent to which incarceration becomes biologically embedded to affect physiological changes implicated in the onset of chronic illness and disease.

BACKGROUND

Incarceration and Stressors

Although relative differences in mortality between black and white individuals is significantly lower in prison compared to the differences in the general population—a pattern largely attributable to deaths from firearms and motor vehicle accidents (Patterson 2010)—formerly incarcerated individuals are at greater risk of dying and suffering from disease following release. Incarceration leaves a negative imprint on the physical health of this population. The total health effects of incarceration are a product of the transition from arrest to confinement, the time spent in prison, and the period following release (Massoglia and Pridemore 2015). The initial transition from the community to prison is a disruptive event partly because individuals must learn to assimilate into the prison environment (Irwin 1980). Moreover, confinement removes individuals from their primary social networks, who are sources of critical emotional and psychological support. During spells of incarceration, personal relationships often become strained and difficult to nurture from a distance.

Adding to the stressors of adjustment, individuals face a variety of harsh and traumatic experiences during incarceration (Massoglia and Remster 2019). Direct experiences with violent events are not uncommon in jails and prisons (Labrecque, Scherer, and McCafferty 2018). For instance, prison surveys have found that upward of 21% of both men and women report attacks by other incarcerated individuals, and nearly 25% and 8%, respectively, report attacks by correctional staff (Wolff et al. 2007). National estimates indicate that approximately 13% of incarcerated individuals are injured in a violent encounter (Teasdale et al. 2016). The loss of personal security—one "pain of imprisonment"—is exacerbated by these distinct moments of danger (Maier and Ricciardelli 2019; Sykes 1958). Secondary, or witnessed, violence also increases fear, hypervigilance, and general unease with the social environment (Novisky and Peralta 2020). Concerns about safety and hostile interactions with other incarcerated individuals and correctional staff create an action readiness of being "on point" that contributes to an "overwhelming sense of anxiety" (Porter 2019:5). Altogether, encounters with violence may "contribute to the sustained experience of emotional distress…up to and beyond release from prison and jail" (Boxer, Middlemass, and Delorenzo 2009:802). Studies have reported associations between violent experiences and the worsening of psychological health during incarceration and after release (Wolff and Shi 2009; Zweig et al. 2015).

Several well-documented barriers to reintegration add to the burden of stressors associated with incarceration (Semenza and Link 2019). Many individuals reenter society with limited resources and narrow employment prospects because of exclusionary hiring practices and weak ties to conventional networks (Petersilia 2003). Safe and secure housing is also a "hurdle in the steeplechase of reentry" because some are barred from renting because of their criminal records (LeBel 2017:889). For instance, in a sample of formerly incarcerated individuals, only half were unemployed 6 months after release, and more than one-third stayed in marginal or temporary housing (Western et al. 2015). Harding and colleagues (2014:450) found more than one-quarter of formerly incarcerated persons shifted between "extreme desperation and survival when they were not in custody." These difficulties are compounded by substance abuse and mental health problems (Tyler and Brockmann 2017). Furthermore, even with the implementation of the Affordable Care Act, formerly incarcerated individuals are more likely to be uninsured and less likely to utilize preventive health care relative to the general population (Widdowson and Fisher 2020; Winkelman, Choi, and Davis 2017).

Incarceration, Stress Response, and Physical Health

According to the neuroendocrine and immunology literatures, the stressors resulting from social and environmental adversities, such as the incarceration experience, trigger the circuitry of the autonomic and parasympathetic nervous systems in addition to the hypothalamic–pituitary–adrenal axis (Miller, Chen, and Parker 2011; Snyder-Mackler et al. 2020). Activation of each system is necessary for survival and reproductive success. However, the prolonged and elevated stimulation of these systems has a gradual "wear and tear" effect on the body's adaptive capacities, producing impairments that are expressed in surrogate end-point biomarkers (Lu et al. 2019; McEwen and Gianaros 2011). Physiological adaptations in response to adversity may also contribute to the programming of the stress response via alternations in histone acetylation (Meaney and Szyf 2005). Moreover, phases of neuroinflammation disrupt the brain's capacity to monitor and control pathogen invasion, which is detrimental to cerebral functioning (Danese and Baldwin 2017). Geronimus and colleagues (2006) argue that the relatively extensive social adversities experienced by African Americans promotes a type of "biological weathering." Specifically, the weathering work contends that African Americans are burdened by material hardships and marginalization encountered in a race stratified society (Geronimus et al. 2006). The accumulation of these adverse conditions contributes to physiological deterioration, including accelerated aging (Simons et al. 2021).

Life course theories of the stress process (Pearlin et al. 2005), the psychoimmunology literatures (Miller et al. 2011), and the biological weathering model (Geronimus et al. 2006) all would contend that incarceration engenders a harmful stress response. Empirical research has indeed shown that incarceration exposure is associated with stress-related illnesses, such as hypertension, diabetes, obesity, heart problems, and functional limitations (Baquero et al. 2020; Houle 2011; Massoglia 2008; Schnittker and John 2007). However, a variety of substantive and methodological issues complicate efforts to fully understand the nature of the relationship between incarceration exposure and morbidity.

First, selection bias and confounding threaten the validity of causal estimates of the effects of incarceration exposure on health. Because adverse physical health conditions and criminal justice contact share many common correlates—social class, childhood adversities, and self-control—it is plausible that preexisting risk factors confound the effects of incarceration exposure on health outcomes (Schnittker, Massoglia, and Uggen 2012). Any unique effect attributable to incarceration might instead result from the endowments that individuals bring to prison. This possibility requires adjustments for preexisting confounds, including adverse childhood experiences and childhood disadvantage, which are reliable determinants of adult morbidity and mortality (Miller et al. 2011). Due to preexisting tendencies to have poor health and to engage in behaviors that result in confinement, it is also necessary for observational research to adjust for selection effects (Massoglia and Pridemore 2015). Prospective longitudinal data are a powerful tool in this regard because they allow for the implementation of pretreatment (i.e., incarceration) measures.

Second, there is a shortage of research on the extent to which violent and hostile social interactions during incarceration contribute to the health of released individuals. As noted earlier, these experiences are not uncommon based on survey and qualitative accounts. The psychological burden of these experiences could encumber the process of reintegration, engendering additional or secondary stressors that are expressed in the postrelease setting. There are good theoretical reasons to expect that exposure to violence, whether direct or indirect, might regulate the effects of incarceration on physical health outcomes. In this way, incarceration is not a homogeneous experience; rather, the extent to which incarceration affects the health of released individuals is determined by their differential experiences with social stressors during the period of confinement.

Third, most research on postrelease morbidity examines measures of general health, chronic conditions, illnesses, and diseases ascertained from survey self-reports. Self-report measures provide valuable information about one's perceived health burden, and some predict mortality (e.g., Jylhä 2009), but these tools are known to suffer from threats of validity and reliability, especially among marginalized sociodemographic groups (Assari, Lankarani, and Burgard 2016; Zajacova and Dowd 2011). Importantly, self-reports of various health conditions are unlikely to capture cases in which underlying illnesses have not yet manifested as obvious symptoms (Boen 2020; Morton and Ferraro 2020). For instance, respondents who report no evidence of illness but have undetected pathology would be considered healthy based on self-reports when they are not, thereby producing misclassification errors. The possibility of these types of errors is heightened among younger populations. Should formerly incarcerated individuals not report symptoms of illness or disease on self-reports, despite underlying pathology, this would thus bias estimates of the incarceration–health association. Incarcerated persons may also be better aware of their own health conditions than if they had not been incarcerated because of their prison medical assessments. If so, this could increase the association between incarceration and self-reported health conditions (Massoglia and Remster 2019). Objective biomarkers are not vulnerable to such subjective recall and reporting errors and, furthermore, they capture predisease pathology regardless of whether individuals exhibit symptoms.

As such, there have been recent calls to integrate biomarkers into research on incarceration and health to understand how incarceration exposure becomes biologically embedded (see Massoglia and Remster 2019; Wildeman and Wang 2017). Notably, some researchers have modeled objective biomarkers, but there have been mixed results. Using longitudinal data from the Coronary Artery Risk Development in Young Adults study, Wang and colleagues (2009) found that prior incarceration was associated with hypertension and left ventricular hypertrophy among adults ages 23 to 35. More recently, scholars have leveraged the National Longitudinal Study of Adolescent to Adult Health (Add Health) study to investigate the association between incarceration and health biomarkers among young adults (Boen 2020; Esposito et al. 2017). For instance, Esposito and colleagues (2017) found no effect of incarceration on hypertension after adjusting for selection effects. Similarly, Boen (2020) found a relationship between incarceration and inflammation, but this effect was reduced after applying treatment weights to adjust for selection. Additionally, incarceration was only associated with inflammation in that study when the period of confinement was a year or longer. Other work with Add Health has found associations between incarceration and cardiovascular disease risk—an index derived from objective and subjective self-report items—using models that did not adjust for threats of selection bias (e.g., Semenza et al. 2021), which raises questions about the internal validity of the findings.

Incarceration and Epigenetic Aging

Research on the social determinants of health has increasingly focused on the speed of cellular aging. This work is consequential because aging is the "most important risk factor for many human diseases" (Ryan et al. 2020:481). Conditions including cognitive decline, heart disease, and cancers are documented products of biological aging (Sinclair 2019:67). Epigenetic aging involves the "accumulation of damage" responsible for cellular and organismal decline (Song and Johnson 2018:1). Researchers have studied mechanisms of aging with measures of telomere length and pubertal timing. Since the 1960s, researchers have been aware of the strong association between age and deoxyribonucleic acid (DNA) methylation (Koch and Wagner 2011). DNA methylation is a form of epigenetic modification—or chemical alteration to the genome that affects gene activity—whereby a methyl group attaches to a segment of DNA at a CpG site, which can inhibit gene expression. Through this process, the activity of a DNA segment can change without an adjustment to its base sequence. Many of these sites are positioned near genes that trigger age-related conditions, including cancer, oxidative stress, respiratory diseases, and Alzheimer's disease (Hannum et al. 2013; Yoon, Jin, and Sin 2019). Some have argued that cellular aging is caused by epigenetic signalers—which repair damaged DNA—becoming overtaxed from cellular insults, including those present in the external environment (Sinclair 2019:47–49). Site-specific patterns in DNA methylation are integral to the aging process among humans (Ryan et al. 2020).

Such findings have prompted the development of various epigenetic indices or "clocks" of biological aging (Murabito et al. 2018). These clocks assess the discrepancy between a person's chronological age and the age predicted by the epigenetic index, which indicates whether a person is aging at an accelerated or decelerated pace. A recently developed clock known as the GrimAge index examines methylation at 1,030 sites and has a superior ability to predict time to mortality and age-related illnesses (Lu et al. 2019) relative to the other available epigenetic clocks (see McCrory et al. 2021). Research indicates that accelerated GrimAge is associated with social adversities, including material hardship, discrimination (Simons et al. 2021), and trauma (Yang et al. 2020).

The extent to which incarceration exposure predicts accelerated biological aging has not been examined. As noted previously, the substantive literature implies that formerly incarcerated persons conceivably age at a faster pace than nonincarcerated persons. If so, this would provide an important clue about how incarceration might give rise to health disparities.

Current Study

Although there is consistent evidence that incarceration exposure undermines the physical health of individuals, important gaps remain in this research. Using data on a sample of African American adults, the present study addresses two objectives. First, using the newly developed GrimAge index, it assesses the extent to which incarceration exposure predicts accelerated biological aging. Second, the study examines whether experiences with violence, both direct and secondary, during confinement exacerbate the effect of incarceration on accelerated biological aging.

DATA AND METHODS

Sample

We tested hypotheses for the current study by employing data from the Family and Community Health Study (FACHS). FACHS is designed to investigate the factors associated with the health and well-being of African American adults (see Lei et al. 2018; Simons et al. 2019). The FACHS sampling strategy includes African American families with a child who was in fifth grade at the time of recruitment. Families were intentionally recruited from neighborhoods that varied on demographic and economic characteristics based on 1990 census data. For 259 census block groups (115 in Georgia and 144 in Iowa), households were randomly selected from rosters of fifth graders in the public school system. Households were then contacted until the required number of households were recruited (84% response rate).

The data for the first wave of FACHS were collected in 1997 and 1998 (for a description of FACHS waves, see Appendix A in the online version of the article). The sample consisted of 889 African American fifth-grade children who were an average age of 10.6 years and resided in Iowa (n = 467) or Georgia (n = 422). The second through sixth waves of data were collected between 1999 and 2012, capturing information when the target children were 12.5, 15.7, 18.8, 21.6, and 23.6 years. The seventh wave of data was completed between 2015 and 2016 when the target children had an average age of 28.8 years. By Wave 6, approximately 49% of respondents resided in Georgia, 40% in Iowa, and the remaining 10% were located in other states.

Only members of the sample residing in Georgia, Iowa, or a contiguous state were identified as eligible for the blood draws needed for the current research due to the logistical challenges of scheduling home visits by phlebotomists. After also excluding persons who were deceased (n = 12), incarcerated at the time (n = 15), hard refusals (n = 19), unidentifiable phone number (n = 46), or otherwise unreachable (n = 99), 556 respondents agreed to be interviewed, 470 of whom successfully provided blood. Of these respondents, successful assaying for methylation was achieved for 449 individuals. In the current study, analyses were based on the 410 African American respondents who were successfully assayed and for whom data were available on all study measures—13 respondents were missing data on incarceration history (2.9%), and an additional 26 respondents were missing data on the covariates in the model (5.8%).[1]

Procedures

Questions were administered to families in their homes using audio-enhanced, computer-assisted, and self-administered interviews. Respondents answered questions on a computer as they were presented visually on the screen. In Wave 7, participants were also asked to provide a blood sample. The phlebotomist drew four tubes of blood (30 mL) from each participant; these were shipped on the same day to a laboratory at a public university for preparation. Blood samples were assessed for genome-wide methylation status using standard protocols. The Illumina Infinium HumanMethylationEPIC 850 BeadChip was used to assay genome-wide DNA methylation. Participants were randomly assigned to 16 sample "slides/chips" with groups of eight slides being bisulfite converted in a single plate, resulting in two "batches/plates." A replicated sample of DNA was included in each plate to aid in assessment of batch variation. Methylation data were filtered according to the following guidelines: (a) Samples containing 1% of CpG sites with detection $p < .05$ were removed, (b) sites were removed if a bead count of <3 was present in 5% of samples, and (c) sites with a detection $p < .05$ in 1% of samples were removed.

Measures

Accelerated aging. To calculate the speed of aging, we used an epigenetic clock known as the GrimAge index (Horvath and Raj 2018). The index is called *GrimAge*—after the Grim Reaper—because an accelerated score is grim news. This measure of aging has been shown to be a more robust predictor of morbidity and mortality among different racial groups than other epigenetic clocks currently available (Lu et al. 2019; McCrory et al. 2021). The index was developed by identifying a set of plasma protein predictors of mortality and then using these protein predictors to identify CpG sites that could predict time to death. As expected, the GrimAge index has a weak to moderate correlation with chronological age in the FACHS sample ($r = .22$), indicating a discrepancy between biological and calendar ages. Following standard procedures, we regressed GrimAge on chronological age to create a speed of aging score or "accelerated aging." The resulting speed of Grim aging measure was thus adjusted to correlate with chronological age at 0. A positive value on this variable indicates, in years, accelerated epigenetic aging, whereas a negative value indicates, in years, decelerated aging.

Incarceration exposure. To assess the prevalence of *incarceration*, we used reports from Waves 5, 6, and 7. If respondents reported being arrested (since age 18 in Wave 7), respondents were asked to report their frequency of being to jail and their frequency of being to prison (since age 18 in Wave 7). If respondents reported being in jail or prison at least once, the measure was coded as 1 = formerly incarcerated. If the respondent never reported being in jail or prison at Waves 5, 6, or 7, the measure was coded as 0 = never incarcerated. "Don't know" responses were treated as missing. Although most studies assess incarceration using a lifetime prevalence measure, it may be that the experience in itself does not fully capture how incarceration exposure is associated with individual health. For this reason, it is important to conduct comprehensive tests of the varieties of exposure. To probe the nature of exposure effects, we also measured the timing and frequency of incarceration. *Past 4 years* was coded as 1 if the respondent reported being in jail or prison in the past 4 years at Wave 7 (average age at Wave 7 was 28.8 years). This 4-year measure was included in the Wave 7 survey to capture spells of incarceration since Wave 6, which was administered approximately 4 years earlier. The measure of incarceration in the past 4 years is intended to assess whether the effect of incarceration exposure on accelerated aging extends beyond lifetime prevalence to recent spells of incarceration. *More than once* was coded as 1 if the respondent reported being in jail or prison two or more times. A frequency measure of incarceration is intended to assess the effect of exposure to multiple spells of incarceration on accelerated aging.

Violence during incarceration. We created two measures to examine experiences with violence during incarceration. To measure *direct violence*, respondents were asked to report during Waves 6 and 7 how often the following experiences happened during jail or prison: property was taken from them using force or intimidation, they were threatened with violence, they were assaulted with an object used as a weapon, or they were involved in physical fights with others incarcerated. If respondents reported experiencing any of these violent events, direct violence was coded as 1 (0 otherwise). To measure *secondary violence*, respondents reported how often they witnessed other incarcerated individuals being assaulted with a weapon or involved in a physical fight (Waves 6 and 7). If respondents reported experiencing either of these events, secondary violence was coded as 1 (0 otherwise).

Confounds. We controlled for the following demographic and socioeconomic characteristics at Wave 7: *male*, *high school education*, *income*, and *married or cohabiting* (for description of the measurement of the confounds, see Appendix B in the online version of the article). These measures were concurrent to the outcome. Income was assessed by asking respondents to report their income in the past year and was log-transformed to reduce skew. The average income in the analytical sample was $21,474.26. We also adjusted for past-year *health insurance* at Wave 7. To account for preincarceration health and health risk behaviors, we included measures of smoking, drinking, diet, exercise, and BMI. This class of measures was included either on or before the wave of the incarceration measure based on their availability in the data. *Smoking* was measured as the prevalence of cigarette smoking in the past year, and *binge drinking* was measured as the frequency of consuming three or more drinks of alcohol during the past year (Waves 1 to 4). Two items indicating the frequency of eating fruits and vegetables were used to measure a *healthy diet* (Waves 4 to 5), and two items indicating the frequency of physical activity for at least 30 minutes were used to measure *exercise* (Waves 3 to 5). *BMI* was calculated using the respondents' recorded height and weight (Waves 1 to 2, 4).

Additional preincarceration conditions were included as potential confounds (see Appendix B in the online version of the article). To measure *community disorder* (Waves 1 to 4), participants were asked about the frequency of criminal acts in their neighborhood. Mental health is an important confound because it is related to both criminal behavior and physical health (Beyers and Loeber 2003). A battery from the Diagnostic Interview Schedule for Children, Version 4 (DISC-IV), was used to measure *depression* (Waves 1 to 4; Shaffer et al. 1993). We summed all responses to create a count variable scored from 0 (no symptoms) to 22 (all symptoms). A measure of *self-control* (Waves 1 to 2, 4) was measured using an 11-item scale from the Kendall and Wilcox (1979) inventory of self-constraint (e.g., "you usually think before you act"). We also adjusted for a measure of *delinquency* (Waves 1 to 4) using the conduct disorder section of the DISC-IV. We summed responses to past-year involvement in 15 acts to create a count variable scored from 0 (no acts) to 15 (all acts).

Two measures accounted for childhood characteristics implicated in criminal justice contact and poor health (see Appendix B in the online version of the article). To assess *childhood adversities*, respondents were asked at Wave 1 to report whether they experienced various negative events during the past year (see Berg et al. 2020). Each item was summed to create a total childhood adversity score ranging from 0 to 14. *Childhood socioeconomic status* was assessed by summing caregivers' reports of education and income and averaging the reports across Waves 1 and 2.

Analytical Strategy

We first conducted descriptive analyses by examining the means and percentages of all variables for the total sample and by incarceration exposure. We then used a series of ordinary least squares regression models to test our hypothesis regarding the association between incarceration and our continuous speed of aging variable, GrimAge, adjusting for a series of key confounds. Given that respondents were not randomly assigned to incarceration, we then estimate the analysis using an inverse probability of treatment weighting (IPTW) scheme to adjust for possible threats of selection bias. For the IPTW procedures, we first created propensity scores by calculating the probabilities of incarceration conditional on our confounds (Rosenbaum and Rubin 1983). We then reran our regression analysis weighted by the propensity scores. By doing so, the IPTW method balances the distribution of confounds across the treated and untreated groups and allows for less biased estimates of the potential causal effect of incarceration (Robins, Hernán, and Brumback 2000). For both the treatment and outcome models, we used the same set of measures. For the raw and weighted mean scores and standardized

differences between the formerly and never incarcerated samples, see Appendix C in the online version of the article. We also conducted sensitivity analyses often recommended for the proper use of IPTW: restricting to common support and trimming (Cole and Hernán 2008). It is worth noting that the quality of estimates—the extent to which they minimize selection bias—derived from propensity score-based methods depends on the extent to which the observed variables not only exhaust sources of possible selection but also if the variables have good validity and reliability.

Finally, we applied an internal moderator approach to assess if the timing, frequency, and exposure to violence determine the health effects of incarceration on GrimAge. Timing, frequency, and exposure to violence during incarceration are conditionally relevant measures—they were only asked of individuals who have been incarcerated. The internal moderator approach explicitly considers that these hypothesized moderators only apply to the respondents who experienced incarceration, providing a "contingent" interaction effect (Mirowsky 2013). In other words, this approach allowed us to compare those who were formerly incarcerated with those who were not while simultaneously assessing if the qualities of the situation—the incarceration spell—determine the effects of being in that situation (Ross and Mirowsky 1992).

RESULTS

Means and percentages for the total sample and by incarceration exposure are displayed in Table 1. Over half of this African American sample reported being formerly incarcerated (52.2%), over one-third were incarcerated in the past 4 years (35.0%), and two-thirds were incarcerated more than once (65.9%). These descriptive results are consistent with literature indicating the mass incarceration of young low-income black adults (see Wildeman and Wang 2017).[2] Among the formerly incarcerated individuals, 12.0% experienced direct violence during incarceration, and 23.4% experienced secondary violence. GrimAge scores indicate that, on average, formerly incarcerated individuals experience accelerated aging ($x = 1.179$), whereas those who were never incarcerated experience decelerated aging ($x = –1.349$, $p < .001$). Table 1 also reveals differences by incarceration exposure across demographic characteristics, preincarceration health behaviors, and childhood adversity.

Incarceration Exposure and Accelerated Biological Aging

Regression models predicting the speed of Grim aging are shown in Table 2. Model 1 presents the estimates of the association between incarceration exposure and the speed of aging net of the full array of confounds. The results show that incarceration is associated with accelerated epigenetic aging in this African American sample, with exposure predicting a greater accelerated aging score by about 2 years ($b = 1.970$; $p \leq .001$). After applying IPTW (Model 2) to the model, the association between incarceration exposure and the speed of aging is slightly reduced but remains statistically significant. Exposure to incarceration is associated with a 1.719 difference (approximately 1 year and 9 months) in accelerated aging ($p \leq .001$). This finding is illustrated in Figure 1—formerly incarcerated individuals are roughly 11 months ($12 \times .902$) older biologically than expected based on their chronological (or calendar) age, whereas those who have never been incarcerated are 10 months younger biologically ($12 \times –.816$) than expected based on their chronological age. In Model 3, we restricted the sample to respondents with propensity scores that fall within the range of propensities calculated for both the incarcerated and never incarcerated respondents. Similarly, in Model 4, we exclude all respondents with atypical propensities—propensity scores within the fifth percentiles. Both models reveal a stronger association between incarceration exposure and the speed of Grim aging, indicating a conservative estimate in Model 2.[3] Together, the evidence from Table 2 indicates that among the African American respondents in our sample, incarceration exposure is associated with an acceleration in biological aging by 21 to 28 months.

To probe the effects of incarceration exposure, we examine whether the timing and frequency of incarceration operate as internal moderators of the association between incarceration exposure and the speed of aging (see Models 1 and 2 of Table 3). Results suggest that the timing of incarceration may not moderate the association—formerly incarcerated individuals did not experience greater accelerated aging if they were incarcerated in the past 4 years (Model 1; see also, Boen 2020). Results also show that the effects of incarceration exposure did not vary by the frequency of incarceration spells (Model 2).[4] These findings align with past studies in which incarceration exposure or prevalence mattered more for health outcomes than dosage (e.g., Schnittker and John 2007; but see Boen 2020).

Table 1. Means (Standard Deviations) and Proportions of Study Variables Stratified by Incarceration Exposure.

Variable	Total Sample (n = 410)	Never Incarcerated (n = 196)	Formerly Incarcerated (n = 212)	t Test
Accelerated GrimAge	–.030 (4.202)	–1.349 (3.708)	1.179 (4.272)	–6.373***
Incarceration exposure	.522	—	—	—
Past 4 years	.183	—	.350	—
More than once	.344	—	.659	—
Direct violence	.063	—	.120	—
Secondary violence	.122	—	.234	—
Male	.376	.260	.481	–4.731***
High school education	.915	.959	.874	3.119**
Income	7.908 (4.195)	8.446 (3.780)	7.415 (4.494)	2.502*
Married or cohabiting	.300	.306	.294	.258
Health insurance	.834	.867	.804	1.732
Smoking	.067 (.149)	.039 (.115)	.093 (.171)	–3.716***
Binge drinking	1.235 (.394)	1.162 (.306)	1.301 (.451)	–3.632***
Healthy diet	2.912 (.931)	2.906 (.908)	2.917 (.954)	–.124
Exercise	2.653 (.790)	2.615 (.782)	2.688 (.796)	–.939
BMI	24.452 (5.239)	25.047 (5.814)	23.906 (5.239)	2.090*
Depression	.309 (.730)	.343 (.748)	.278 (.714)	.896
Community disorder	1.264 (.247)	1.240 (.237)	1.287 (.255)	–1.951
Self-control	1.660 (.249)	1.662 (.266)	1.658 (.233)	.124
Delinquency	.255 (.510)	.184 (.387)	.320 (.595)	–2.725**
Childhood adversity	2.844 (2.735)	2.546 (2.632)	3.117 (2.805)	–2.120*
Childhood socioeconomic status	–.032 (1.449)	.138 (1.438)	–.189 (1.444)	2.293*

Note: Data from Family and Community Health Study, Waves 1997–2016.
*p < .05, **p < .01, ***p < .001, two-tailed.

Violence During Incarceration

Next, in Models 3 and 4 of Table 3, we examine whether violence moderates the association between incarceration exposure and the speed of Grim aging. As shown in Model 3, direct violence intensifies the association between incarceration exposure and accelerated aging in this African American sample (b = 1.388; p < .05). Figure 2 illustrates these patterns. As shown, formerly incarcerated individuals who did not report direct violence are roughly 9 months (12 × .776) older biologically than expected based on their chronological age. Those who did report direct violence during incarceration are approximately 2 years and 2 months (12 × 2.164) older biologically than expected based on their chronological age. These results indicate that the direct experiences with violence during incarceration moderate the association between incarceration exposure and the speed of aging. Secondary violence, however, does not moderate the association (see Model 4), suggesting that witnessing violence may not exacerbate the already stressful incarceration experience for African American adults.

Supplementary Analyses

The literature remains mixed regarding gender differences in the physical health impacts of incarceration. Some studies find that the association between incarceration and mortality is stronger for women than men (e.g., Massoglia et al. 2014; Patterson 2010), whereas studies of incarceration and morbidity tend to find little variation (e.g., Baquero et al. 2020; Schnittker and John 2007; Wang et al. 2009). Few studies, however, have examined gender differences among a sample of black individuals (cf. Semenza et al. 2021). In supplementary analyses, we explore whether the gender of respondents moderates the association between incarceration exposure and the speed of Grim aging (not shown in tabular form). Approximately 66.9% of males and 43.4% of females were formerly incarcerated.

Table 2. Ordinary Least Squares Regression Models Predicting Accelerated GrimAge.

	Model 1[a]		Model 2[b]		Model 3[c]		Model 4[d]	
Variable	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Incarceration exposure	1.970***	(.408)	1.719***	(.415)	1.846***	(.429)	2.339***	(.446)
Male	2.041***	(.458)	2.186***	(.501)	2.142***	(.515)	1.981***	(.577)
High school education	–.666	(.841)	–1.016	(.772)	–.976	(.786)	.069	(1.215)
Income	–.086	(.057)	–.063	(.058)	-.058	(.057)	–.103	(.065)
Married or cohabiting	–.627	(.417)	–.592	(.443)	–.500	(.434)	–.402	(.471)
Health insurance	.399	(.578)	.219	(.593)	.186	(.594)	.310	(.639)
Smoking	2.967*	(1.450)	4.856*	(1.906)	5.827**	(2.097)	3.503	(1.885)
Binge drinking	.179	(.576)	1.029	(.555)	.684	(.695)	.527	(.764)
Healthy diet	–.197	(.204)	.003	(.215)	–.068	(.216)	–.154	(.248)
Exercise	–.105	(.266)	–.028	(.276)	–.029	(.277)	–.295	(.331)
BMI	.086*	(.034)	.074*	(.036)	.083*	(.037)	.070*	(.039)
Depression	.148	(.306)	–.234	(.256)	–.060	(.268)	.101	(.346)
Community disorder	–.405	(.883)	–.922	(.902)	–.807	(.929)	–.092	(1.012)
Self-control	–.380	(.838)	–.356	(.838)	–.206	(.831)	–.211	(.920)
Delinquency	–.334	(.482)	–.426	(.475)	–.433	(.495)	–.571	(.502)
Childhood adversities	–.064	(.076)	–.010	(.081)	–.031	(.088)	–.043	(.087)
Childhood socioeconomic status	–.040	(.161)	–.008	(.152)	.097	(.151)	.094	(.183)
Constant	–.989	(2.282)	–1.641	(2.217)	–1.767	(2.286)	–2.235	(2.619)

Note: Data from Family and Community Health Study, Waves 1997–2016. Unstandardized coefficients (*b*) and robust standard errors (*SE*) are presented.
[a]Ordinary least squares regression ($n = 410$).
[b]Ordinary least squares regression with inverse probability of treatment weighting ($n = 410$).
[c]Ordinary least squares regression with inverse probability of treatment weighting restricted to common support ($n = 402$).
[d]Ordinary least squares regression with inverse probability of treatment weighting trimmed at the fifth percentiles ($n = 313$).
*$p < .05$, **$p < .01$, ***$p < .001$, two-tailed.

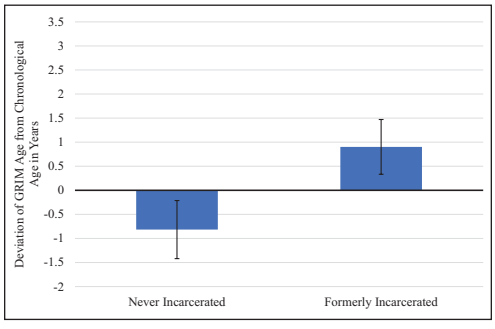

Figure 1. Speed of GrimAge by Incarceration Exposure.
Note: Data from Family and Community Health Study, Waves 1997–2016. Marginal effects estimates derived from regression Model 2 of Table 2. Vertical bars indicate the 95% confidence interval; $n = 410$.

Supplementary models—available on request—revealed that gender does not significantly moderate the association between incarceration exposure and accelerated aging in this African American sample ($b = .303$, $SE = .900$; $p = .736$). Exposure to incarceration is associated with a 1.904 difference (approximately 1 year and 11 months) in accelerated aging for African American men ($p \leq .001$) and a 1.602 difference (approximately 1 year and 7 months) in accelerated aging for African American women ($p \leq .001$).

DISCUSSION

A growing literature traces the consequences of mass incarceration for dimensions of individual-level health and well-being. Some have argued that the

Table 3. Ordinary Least Squares Regression Models Predicting Accelerated GrimAge from Incarceration Timing, Frequency and Exposure to Violence[a]

	Model 1		Model 2		Model 3		Model 4	
Variable	*b*	*SE*	*b*	*SE*	*b*	*SE*	*b*	*SE*
Incarceration exposure	1.504***	(.452)	1.194*	(.546)	1.593***	(.424)	1.616***	(.441)
Internal moderators:								
× past 4 years	.693	(.613)	—		—		—	
× more than once	—		.854	(.624)	—		—	
× direct violence	—		—		1.388*	(.701)	—	
× secondary violence	—		—		—		.488	(.625)

Note: Data from Family and Community Health Study, Waves 1997–2016. Unstandardized coefficients (*b*) and robust standard errors (*SE*) are presented.

[a]Ordinary least squares regression with inverse probability of treatment weighting ($n = 410$). Confound estimates not shown in the table: male, high school education, income, married or cohabiting, health insurance, smoking, binge drinking healthy diet, exercise, BMI, depression, community disorder, self-control, delinquency, childhood adversities, childhood socioeconomic status.

*$p < .05$, *** $p < .001$, two-tailed.

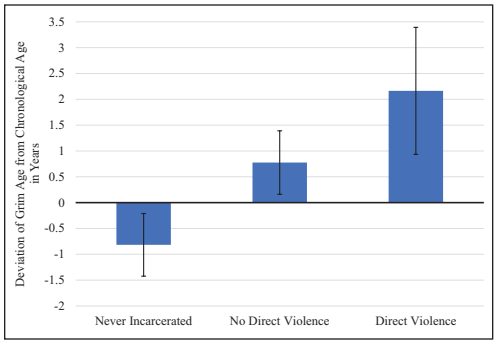

Figure 2. Speed of GrimAge by Incarceration Exposure and Direct Violence during Incarceration.

Note: Data from Family and Community Health Study, Waves 1997–2016. Marginal effects estimates derived from multivariate regression model in Model 3 of Table 3. Vertical bars indicate the 95% confidence interval; $n = 410$.

pervasiveness and concentration of incarceration among disadvantaged groups contributes to widening health inequalities (Wildeman and Muller 2012:12). Although research has reported associations between incarceration exposure and various negative health outcomes, important substantive and methodological gaps remain in this body of work. These gaps reflect special challenges in determining whether and why formerly incarcerated individuals report an elevated burden of chronic illnesses and diseases. The goal of the current study was to extend this area of research by examining whether incarceration exposure is related to the pace of biological aging and if experiences with violence during incarceration intensify this association. To do so, the study employed data on a sample of African American adults along with measures of several relevant confounds from different life course domains, and it implemented adjustments for selection.

The results of the analysis support two key substantive conclusions. First, incarceration exposure significantly predicts accelerated biological aging among African American adults, as assessed in the GrimAge measure. Study participants who reported a history of incarceration were, on average, biologically older than expected given their numerical age. Notably, the estimates were robust to a host of potential confounding factors obtained earlier in the life course, including adverse childhood experiences, social class, depression, and delinquency. Even after implementing statistical adjustments, the findings remained stable. This is noteworthy because the proposition that any observed effects of incarceration on physical health are due to selection, confounding, or both has a strong logical basis (Schnittker et al. 2012). The results therefore provide reasonable evidence that the stressors of incarceration exposure appear to contribute to the pace of epigenetic aging.

This evidence is important for understanding the physical health toll of incarceration among African Americans because accelerated aging is a fundamental predisease process. This process reliably predicts mortality risk and the onset of chronic and acute morbidities (Sinclair 2019). Research has

shown that accelerated aging is among the most important drivers of adverse health conditions already linked to incarceration, including hypertension and cancer (Massoglia 2008; Wang et al. 2009), all of which diminish total health span (Murabito et al. 2018; Yoon et al. 2019). Epigenetic aging may be a biological pathway whereby the hardships of incarceration exposure affect the onset of acute illnesses and chronic conditions. Furthermore, the current findings add to the burgeoning body of work focused on how incarceration may "get under the skin" to leave an imprint on bodily systems (e.g., Boen 2020; Esposito et al. 2017). That we uncovered evidence of accelerated aging in a sample of African American adults who were approximately 30 years of age is worth noting—this is a period in the life course when age-related chronic diseases and emerging pathology typically do not yet manifest and register clinically. As Moffitt and colleagues (2017) note, accelerated aging is not simply an affliction of the elderly but a lifelong biological process that begins early in the life span.

Second, exposure to violence during incarceration exacerbates the degree to which incarceration exposure hastens the pace of Grim aging among African American adults. Those who experienced violence were more than 2 years older than their calendar age. This effect, however, was exclusive to direct incidents of violent threats, assaults with and without weapons, and robbery. Interpersonal violence likely engenders a stress response that chronically activates immunological and nervous system capacities. This evidence aligns with the literature on prison violence and mental health (Zweig et al. 2015). Moreover, it accords with research indicating that threatening social environments trigger adverse physiological responses (Miller et al. 2011; Simons et al. 2021). Perhaps violent victimization is especially stressful in the confined spaces of the prison context where hostile interpersonal relations are particularly difficult to circumvent. As one formerly incarcerated individual explained: "The whole mental thing of me… trying to think of who I could have a problem with or who might have a problem with me all the time… is extremely stressful" (Porter 2019:9). Other potentially traumatic prison and jail conditions are deserving of additional research on the biological consequences of incarceration, including discriminatory treatment and placement in solitary confinement, the latter of which has been linked to mental health (Strong et al. 2020). Similarly, researchers should study conditions during postrelease that might buffer the link between incarceration and adverse physiological deterioration, including relational and community supports.

The present study did not examine how incarceration contributes to racial disparities in aging given that the sample was limited to African American individuals. But this work is needed. Contact with the criminal justice system—stops, arrests, and jail and prison stays—is disproportionately felt by African Americans owing to a legacy of discrimination and marginalization in a racialized society. Whether the linkage between incarceration and adverse health outcomes differs by race is the focus of ongoing research (Boen 2020; Semenza et al. 2021). The findings from this study suggest ways forward with research on health disparities, particularly biological markers. As noted, epigenetic aging indices are thought to be direct measures of the biological weathering conceptualized in Geronimus and colleagues' (2006) model of health inequalities (see Simons et al. 2021). Perhaps, then, epigenetic aging serves as a biological mechanism through which disparities in incarceration contribute to racial differences in chronic morbidity and excess mortality. Note, however, that we are unable to test this possibility given our reliance on an African American sample. Future research should examine the validity of this assumption given that it might hold important clues about how mass incarceration and other race-related social adversities combine to affect health inequalities. Modern medicine often considers differences in health risk behaviors (e.g., diet) as the major cause of class and race disparities in chronic illness (Milani and Lavie 2015). Some argue that this view perhaps exaggerates the importance of health risk behaviors as a cause of health inequalities. Our findings show that regardless of diet, exercise, and smoking, incarceration exposure is a significant social determinant of accelerated aging.

The current study is not without limitations. Despite utilizing a series of preincarceration measures of likely confounds, the analysis did not assess the GrimAge of respondents prior to incarceration. Rather, GrimAge was obtained at a single period—Wave 7. Assessing a biomarker of aging at multiple time points would allow for a more compelling assessment of how incarceration affects changes across the life course in the pace of biological aging. This question should be a priority for future research. Readers should also use caution when generalizing our findings to other health biomarkers, including objective markers of inflammation and cardiovascular disease risk. Furthermore, the IPTW

procedure is intended to minimize threats of selection based on differences in observables, which provides some confidence in our estimates. However, as noted earlier, the quality of these estimates is dependent on the reliability and validity and scope of the observed measures. Furthermore, the estimates remain vulnerable to unobserved sources of selection that are not controlled in the current IPTW methods but may be addressed through fixed effects estimators in longitudinal data.

Additionally, as noted, our sample consists of African American adults under age 30, which limits the generalizability of our findings to other groups. Although an exclusive focus on an African American sample is a strength given their high level of exposure to incarceration and worse health profile, future research should explore the extent to which incarceration accelerates aging among other racial and ethnic groups. Similarly, researchers should consider the possibility that our findings could vary in a sample of older adults with a more substantial health burden. A sample that included older adults could also allow for a detailed assessment of how the timing of incarceration may affect the speed of aging. In addition, because the majority of FACHS respondents reside in two states, this limits the generalizability of the findings to individuals located in other states. The characteristics of prisons and jails and correctional policies, including reentry programming, in each of these states might uniquely affect the biological health of the formerly incarcerated in such a way that similar findings may not be observed among those in other states.

The consequences of mass incarceration for health and well-being have become a topic of interest among policymakers and academics from multiple scientific disciplines for good reason: With so many Americans cycling through prisons and jails each year, the public health toll of the carceral state is likely to mount, creating challenges for communities already beleaguered by accumulated hardships. The findings would seem to have important intervention and policy implications. At the very least, they provide further support for decreasing the scale of the carceral state and providing sound alternatives to punitive penal policies for the sake of reversing health inequalities.

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ORCID IDS

Mark T. Berg https://orcid.org/0000-0003-2709-0950
Ronald L. Simons https://orcid.org/0000-0002-4902-0462

SUPPPLEMENTAL MATERIAL

Appendices A through C are available in the online version of the article.

NOTES

1. We reestimated our analyses using a multiple imputation strategy to recover missing values from the confounding measures—this step increased the sample size to 436 respondents. The results did not substantively depart from the main findings and conclusions.
2. Furthermore, Iowa—where many in the Family and Community Health Study (FACHS) sample reside—incarcerates black residents at a rate that exceeds most other states (Beck and Blumstein 2018). Compared to other states, Georgia's rate of incarceration (1,110 per 100,000 adult residents) is higher than the average rate (750 per 100,000 adult residents) in the United States (Maruschak and Minton 2020). Iowa's rate of incarceration (580 per 100,000 adult residents) is lower than the average in the United States.
3. In an additional sensitivity analysis, we use a regression adjustment estimator in addition to inverse probability of treatment weighting (see Boen 2020). When using a regression adjustment, we find a similar treatment effect ($b = 1.836$, $SE = .386$, $p = .000$).
4. All formerly incarcerated individuals in the sample spent time in jail; 25 (6.1%) spent time in prison in addition to jail. There were no cases where individuals spent time in prison but not jail. Supplemental analyses revealed that exposure to prison did not have a unique effect on the speed of aging among those exposed to incarceration ($b = -.348$, $SE = .906$, $p = .701$).

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AUTHOR BIOGRAPHIES

Mark T. Berg is a professor of sociology and criminology and director of the Crime and Justice Policy Research Program at the University of Iowa, where he is also a Collegiate Scholar. His current research examines conflict

escalation and the life course determinants of biological aging and cardiovascular disease risk.

Ethan Rogers is an assistant research scientist in the Crime and Justice Policy Research Program at the University of Iowa Public Policy Center. His research utilizes quantitative analyses to explore interpersonal conflict and violence, the spatial distribution of crime across communities, trends in violence, and the health consequences of incarceration.

Man-Kit Lei is an assistant professor of sociology and affiliated with the Center for Family Research at the University of Georgia. His research utilizes quantitative methods to examine health-related outcomes such as chronic inflammation and rate of biological aging. Recent articles have investigated the impact, for better or worse, of the social environment on biomarkers of health, especially among minority populations.

Ronald L. Simons is Regent's professor in the Department of Sociology and codirector of the Center on the Biological Embedding of Social Events and Relationship at the University of Georgia. He has been funded continuously by National Institutes of Health for over 25 years. His research utilizes longitudinal data to investigate avenues whereby social events and circumstances such as racial discrimination, economic hardship, neighborhood disorder, and relationship conflict become biologically embedded and influence speed of aging, health, and mortality.