HHS Public Access

Author manuscript

J Health Soc Behav. Author manuscript; available in PMC 2021 September 01.

Published in final edited form as:

J Health Soc Behav. 2020 September ; 61(3): 290–306. doi:10.1177/0022146520936208.

Criminal Justice Contacts and Psychophysiological Functioning in Early Adulthood: Health Inequality in the Carceral State

Courtney Boen

University of Pennsylvania

Abstract

Despite increased attention to the links between the criminal justice system and health, how criminal justice contacts shape health and contribute to racial health disparities remains to be better understood. Using data from the National Longitudinal Study of Adolescent to Adult Health (N=5,488) and several analytic techniques—including a quasi-treatment-control design, treatment weighting procedures, and mediation analyses—this study examines how criminal justice contacts shape inflammatory and depressive risk and contribute to Black-White health gaps. Findings revealed that incarceration is associated with increased C-reactive protein and depressive risk, particularly for individuals who experienced long durations of incarceration. Arrests are also associated with mental health, and mediation analyses showed that racial disparities in arrests and incarceration were drivers of Black-White gaps in depressive symptoms. Together, this study provides new evidence of the role of the criminal justice system in shaping health and patterning Black-White health gaps from adolescence through early adulthood.

INTRODUCTION

The rapid growth of the penal system over the past forty years is unprecedented (National Research Council 2014). Nearly 7 million individuals are currently under some form of correctional supervision in the United States (Kaeble and Glaze 2016), and millions more are stopped and arrested by police each year (U.S. Department of Justice 2013). While the increases in imprisonment in recent decades years have been historic (Pettit and Western 2004), data also reveals stark racial disparities in criminal justice contacts (New York Civil Liberties Union 2016; Pettit and Western 2004; Starr and Rehavi 2012). In particular, Black men and boys from socioeconomically disadvantaged backgrounds face the greatest risk. Estimates suggest that more than 60 percent of Black men without a high school degree can expect to be imprisoned by their mid-30s (Pettit and Western 2004; Western and Wildeman 2009).

Research increasingly links criminal justice contacts to increased health risk from a host of causes (Geller et al. 2014; Lee and Wildeman 2013; McFarland et al. 2018; Porter and Novisky 2017; Schnittker and John 2007; Sewell, Jefferson, and Lee 2016; Sugie and

Corresponding Author: Courtney Boen, 232 McNeil Building, 3718 Locust Walk, Philadelphia, PA 19104-6299, cboen@upenn.edu.

SUPPLEMENTAL MATERIAL

Additional supporting information may be found in the online version of this article.

Turney 2017; Wildeman and Muller 2012). In light of this emerging evidence, it is likely that the rises in aggressive policing and surveillance practices and mass incarceration in the United States over the past several decades—combined with the striking racial disparities in criminal justice contacts—have implications for individual health and population-level racial health inequities.

Still, several critical questions about the role of the criminal justice system in shaping individual health and population health disparities remain unanswered. In particular, existing research has been hindered by concerns about possible bias related to selection and confounding, casting uncertainty on the links between the criminal justice system and health. Further, due largely to data limitations, few studies examine how the life course timing, duration, or dosage of criminal justice encounters affect health; assess the psychophysiological mechanisms undergirding the associations between criminal justice contacts and health outcomes; or comprehensively consider how contacts along the criminal continuum shape individual health risk and contribute to population health disparities. Together, these gaps indicate that more research on the role of the criminal justice system in shaping health is needed.

This study advances knowledge on the health consequences of police surveillance and mass incarceration by examining whether contacts along the criminal justice continuum—including police stops, arrests, convictions, and incarceration—affect individual health risk and contribute to racial health inequality in early adulthood. Drawing on the stress process model and life course perspective, this study integrates critical markers of psychophysiological functioning, including indicators of inflammation and depressive risk, to provide new insights into the biological and psychological processes undergirding the associations between criminal justice contacts and health. Further, by testing life course theories of timing, duration, and dosage and explicitly assessing how Black-White disparities in criminal justice encounters contribute to racial health gaps, this study provides new evidence of the essential role of the criminal justice system in the early-life emergence and divergence of racial health inequality. Finally, this study utilizes a variety of analytic techniques—including prospective regression models, a unique treatment-control design, treatment weighting procedures, and mediation analyses—to offer a rigorous assessment of the linkages between the criminal justice system and health.

Given unprecedented and historic increases in aggressive policing practices and mass incarceration in the U.S. in recent decades, studies of population health inequality must consider the carceral context in which trajectories of health emerge, unfold, and diverge. Using nationally-representative data on young adults in the United States, this study interrogates the associations between criminal justice contacts and health and documents how contacts with criminal justice system are critical, early-life drivers of individual health and racial health inequities.

BACKGROUND

Mass Incarceration, Police Surveillance, and Racial Inequality

Since the 1960s, the number of incarcerated individuals in the U.S. has increased eightfold (Pettit and Western 2004), and more than 2.2 million people are in prisons and jails throughout the U.S. (The Sentencing Project 2017). The rapid rise of mass incarceration has been accompanied by increases in aggressive policing and surveillance practices (Alexander 2012). Annually, more than 12 million individuals are arrested (U.S. Department of Justice 2013), and an even larger number are stopped by police. Together, these processes have meant that an increasing number of Americans have been touched, directly and indirectly, by the criminal justice system.

Research also reveals striking racial disparities in criminal justice contact. Data from New York City's "Stop and Frisk" program revealed that, of the more than 685,000 police stops in 2011, nearly 90 percent of individuals stopped by police were Black or Hispanic (New York Civil Liberties Union 2016). Black Americans face higher arrest rates for various types of crimes than Whites, even after accounting for racial differences in socioeconomic status, criminal offending, and neighborhood-level police surveillance (Mitchell and Caudy 2015). Compared to Whites charged with similar offenses, Black Americans are more likely to be convicted and receive sentences that are approximately 10 percent longer (Starr and Rehavi 2012).

Racial disparities in police stops, arrest, and conviction combine to produce stark racial disparities in incarceration. For men born in the US since the late 1960s, more than 1 in 5 Black men could expect to be imprisoned at some point by their early 30s, compared to 1 in 30 White men (Western and Wildeman 2009). Taken together, research in this area provides compelling evidence that the policies and practices of criminal justice system are wrought with racial biases that systematically disadvantage Black Americans, in particular (Alexander 2012).

Criminal Justice Contacts and Health

A growing body of research assesses whether criminal justice contacts affect individual health and contribute to population health disparities, with much of the work in this area focused on the relationship between incarceration and health. Studies document that, while there may be short-term health improvements associated incarceration (Spaulding et al. 2011), these improvements—typically attributed to improved health care access within prisons and jails—do not translate into long-term health benefits (Wakefield and Uggen 2010). Formerly incarcerated individuals have higher rates of morbidity from an array of conditions, including hypertension (Massoglia 2008), sexually transmitted infections (Hammett, Harmon, and Rhodes 2002), and chronic conditions (Schnittker and John 2007), as well as higher rates of overall mortality (Spaulding et al. 2011).

The literature proposes several mechanisms underlying the incarceration-health link. First, incarceration may affect health outcomes through stress-related processes. Consistent with Pearlin's concept of stress proliferation (Pearlin, Aneshensel, and LeBlanc 1997), incarceration serves as a primary and secondary stressor in the lives of individuals. Primary

stressors associated with incarceration include the loss of autonomy, physical and emotional isolation, and fear for ones's safety (Massoglia 2008; Porter 2014). The stress of incarceration also extends beyond release, with secondary stressors including the many challenges associated with re-entry, such as difficulties securing food, employment, and housing and battling stigma (Kirk 2018; Porter 2014; Schnittker and John 2007; Testa and Jackson 2019). Studies linking incarceration to health often draw on evidence that shows that, in response to stressors, the hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS) respond by secreting hormones to up-regulate functioning across bodily systems, inducing a host of physiological and psychological changes. While this upregulation is necessary to protect health against acute threats, long-term activation of these systems in response to chronic stress exposure harms health by promoting systemic inflammation, suppressing immune function, and increasing distress (Cohen et al. 2012; Glaser and Kiecolt-Glaser 2005). In addition to direct stress-related pathways, incarceration also harms health by negatively impacting individuals' socioeconomic status and financial stability (Wakefield and Uggen 2010), restricting access to health resources such as food and housing (Kirk 2018; Testa and Jackson 2019), eroding individuals' social ties (Comfort 2009), and increasing exposure to communicable diseases and infections (Hammett, Harmon, and Rhodes 2002).

A growing body of research assesses the links between pre-incarceration criminal justice contacts—including police stops and arrests—and health. These studies indicate that highly surveilled individuals and communities face increased health risk, including worse mental health (Del Toro et al. 2019; Geller et al. 2014; Sewell, Jefferson, and Lee 2016; Sugie and Turney 2017), lower self-rated health (McFarland, Geller, and McFarland 2019), and accelerated cellular aging (McFarland et al. 2018). Studies in this area typically draw on the stress process model to understand how police contacts, in particular, can serve as stressors in the lives of individuals—inducing fear and distress, increasing financial burden through fines and fees, and increasing stigma—in ways that harm health (McFarland et al. 2018; McFarland, Geller, and McFarland 2019).

Gaps in the Literature

Despite increasing attention to the role of the criminal justice system in shaping individual and population health, four critical gaps in the literature remain. First, concerns about selection bias and unmeasured confounding plague this literature (Kirk and Wakefield 2018). Even models with extensive adjustment for factors endogenous to health and criminal justice contacts, such as criminality or drug use, may not provide convincing evidence of a relationship between arrest, imprisonment, release, and health (Wildeman 2011), as many of the same measured and unmeasured factors that increase individual risk for criminal justice system involvement—such early-life socioeconomic hardship—are also drivers of health. In this way, any observed relationship between criminal justice contacts and health may reflect underlying differences between those reporting no contacts and those with a history of criminal justice contact, rather than reflecting any health effects of the contacts themselves (Porter 2014). For these reasons, research using treatment weighting procedures or treatment-control designs can help in obtaining less biased estimates of the health consequences of criminal justice involvement.

Second, because most studies in this area use disease outcomes, concerns about biological plausibility and misclassification error remain. Of the studies that examine the health effects of incarceration, many focus on the spread of infectious diseases in jails and prisons or examine chronic disease outcomes. Less is known about how criminal justice contacts "get under the skin" to affect pre-disease markers of health and disease susceptibility. Though many studies of criminal justice contacts and health draw from the stress process model, few integrate biomarkers of psychophysiological stress response. Further, using measures of disease or diagnosis as outcomes raises concerns about misclassification error, whereby individuals who do not yet have a disease or have not yet been diagnosed with a disease are classified as "well" (Aneshensel et al. 1991). The misclassification of individuals with high levels of psychophysiological dysregulation as "healthy" could result in an underestimation of the role of criminal justice encounters in affecting health. Concerns misclassification error are particularly relevant for studies incarceration and health (Massoglia and Remster 2019), as well as studies of younger populations and racial disparities in health, given disparities in health care access and utilization and diagnosis by age and race. For these reasons, research using markers of psychophysiological functioning can elucidate how contacts with the criminal justice system contribute to disease emergence and progression in early adulthood and produce better estimates of the contribution of the criminal justice system to population health inequality.

Third, while life course researchers have highlighted the importance of the timing, duration, and repetition of exposures for health (Pavalko and Willson 2011), few studies of criminal justice contacts and health empirically test these concepts. Only a handful of studies have examined how duration of incarceration impacts health, and studies generally find that exposure to incarceration matters more for health than duration (Schnittker and John 2007). Some studies even suggest that long durations may result in health improvements (Zamble 1992). Similarly, despite evidence that the number of incarceration experiences relates to health risk (Porter and DeMarco 2019), prior research typically operationalizes incarceration history as a binary measure without consideration of incarceration "dosage." Further, few studies assess how age of incarcerated shapes health risk, despite evidence indicating that the life course timing of exposures has implications for health (Ben-Shlomo and Kuh 2002; Del Toro et al. 2019). Age-specific rates on criminal justice contacts indicate that encounters with the criminal justice system begin early in the life course, suggesting that these contacts have the potential to contribute to the early-life emergence and divergence of subpopulation trajectories of health inequality (London and Myers 2006). Research indicates that the transition to adulthood is a particularly sensitive period for both social well-being and health, when experiences such as incarceration have critical effects on future trajectories of health (Esposito et al. 2017), which highlights the need for research on the health consequences of criminal justice contacts among young adults (Kinner and Young 2018), in particular.

Finally, in studies of criminal justice contacts and health, few simultaneously consider how contacts along the criminal justice continuum jointly contribute to health risk, which raises a number of concerns. For one, it is possible that documented associations between incarceration and health are confounded by pre-incarceration criminal justice contacts. Given that studies link police stops and arrests to health (Del Toro et al. 2019; McFarland et

al. 2018; McFarland, Geller, and McFarland 2019; Sugie and Turney 2017), it is possible that the relationship between incarceration and health could result from high rates of police contact, arrest, and conviction among formerly incarcerated individuals, rather than reflecting any health consequences of incarceration. Second, while studies assess the unique associations between particular forms of criminal justice contacts and health, few comprehensively assess how contacts along the criminal justice continuum contribute to population health disparities, which suggests that the role of the criminal justice system in patterning population health may be underestimated. For these reasons, a more holistic accounting of the joint contributions of criminal justice contacts to population health is needed.

Research Questions

Using nationally-representative, longitudinal data, this study uses a multi-stage analytical design to advance understanding of the role of the criminal justice system in generating population health disparities. While I focus on the relationship between incarceration and health, the study also examines the role of pre-incarceration criminal justice contacts in shaping health risk and contributing to racial health disparities. Together, this study addresses four overarching research questions:

1. What is the association between incarceration and markers of physiological and psychological well-being in early adulthood?
2. Does the life course timing, duration, and dosage of incarceration matter for health?
3. Do pre-incarceration contacts with the criminal justice system shape health risk and help to account for the association between incarceration and health?
4. What is the contribution of Black-White disparities in criminal justice contacts to racial health inequities?

DATA AND METHODS

Data

Data for this study come from the National Longitudinal Study of Adolescent to Adult Health (Add Health) (1994–2009), a nationally representative, longitudinal study of U.S. adolescents (Harris et al. 2009). Using a school-based complex cluster sampling frame, Add Health began in 1994–95 with in-school questionnaires and in-home interviews at Wave I and followed up with in-home interviews in 1996 (Wave II), 2001–02 (Wave III), and 2007–08 (Wave IV). This study utilizes data from the in-home interviews at Waves I, III, and IV, the biomarker collection at Wave IV, and Census tract-level data linked to respondents' residences at Wave I and IV.

Measures

Outcomes—Outcomes include two measures of psychophysiological well-being that are critical markers of disease risk in young adulthood linked to social stress exposure. C-reactive protein (CRP) is an acute phase protein produced by the liver. A wide body of

research links stress exposure, particularly exposure to chronic stressors and strains, to increases in inflammation and declines in immune response, as indicated by circulating CRP (Everett et al. 2014; Steptoe et al. 2007). Chronically elevated levels of CRP have been linked to increased health risk, including higher rates of heart disease and mortality (Emerging Risk Factors Collaboration 2010). Add Health assayed CRP from dried blood spots at Wave IV. Because of a skewed distribution, the measure of CRP is log transformed. Supplementary analyses using categorical (<1 mg/L, 1–3 mg/L, >3mg/L) and binary measures (>3mg/L) of CRP produced substantively similar results. I also ran models using a composite indicator of immune function as the sum of dummy variables indicating high risk CRP (>3 mg/L) and Epstein-Barr virus (EBV) antibodies (>204 AU/ml, indicating top quartile of distribution), and these models produced substantively similar results to those presented here.

Second, this study uses a count of depressive symptoms based on the Center for Epidemiological Studies Depression Scale (CES-D) (Radloff 1977). At Wave IV, respondents were asked how often over the past seven days they felt: bothered by things that don't usually bother them; they could not shake off the blues, even with help from family or friends; they had trouble keeping their mind off what they were doing; they felt depressed; and they felt sad (for each measure: 0=never or rarely; 1=sometimes; 2=a lot of the time; 3=most or all of the time). To create the scale, I summed the values of the five individual questions, generating a measure ranging from 0–15, with higher values indicating greater depressive risk. Results were robust to alternative operationalizations of depressive risk. In addition to being predictive of future depression, research links depressive symptoms to declines in physical health, whereby depressive states upregulate physiological stress response and down-regulate immune function, increasing health risk from a variety of causes (Kiecolt-Glaser and Glaser 2002).

Previous research highlights the critical roles of inflammation (Everett et al. 2014) and depressive risk (Adkins et al. 2009) in linking social exposures to young adult health risk. Together, these outcomes provide unique yet complimentary insights into how criminal justice contacts shape both short and long-term health risk through a variety of psychophysiological mechanisms.

Criminal Justice Contacts—The key exposures in this study indicate contacts with the criminal justice system. First, I focus on the relationship between incarceration and health using a dummy variable for incarceration history (1=ever having been in prison or jail). I also use measures indicating age at first incarceration (0=never incarcerated; 1=incarcerated before age 18 years; 2=incarcerated after age 18 years), total duration of all incarceration experiences (0=never incarcerated; 1=incarcerated for less than one year; 2=incarcerated for more than one year), and incarceration dosage (0=never incarcerated; 1=incarcerated once; 2=incarcerated two or more times). Supplementary analyses with utilizing different age and duration cutoffs produced substantively similar results. Next, I conduct analyses that include contacts along the criminal justice continuum by constructing dummy variables indicating history of criminal justice contact: ever stopped by police, ever arrested, ever convicted, and formerly incarcerated.

Other Measures—I include a measure of respondent race to indicate racial disparities in the outcomes and the exposures (1=non-Hispanic Black; 0=non-Hispanic White). In addition to individual characteristics such as age and gender, I also include measures reflecting a host of background characteristics, including family socioeconomic status (SES) in adolescence, neighborhood economic disadvantage in adolescence, neighborhood racial composition in adolescence, mental health in adolescence, delinquency, whether the respondent is a repeat arrestee, whether the respondent was arrested for a violent crime, and whether the respondent ever had a parent incarcerated. Details on the coding of all measures used are in Appendix A. Supplementary analyses included county-level crime rates and substance use during adolescence as covariates, but these measures were not significantly associated with young adult health risk, made models less parsimonious, and were excluded from final models.

Analytic Strategy

Descriptive Analyses—I begin with descriptive analyses, paying particular attention to disparities between formerly incarcerated and never incarcerated individuals and racial disparities in the outcomes, exposures, and covariates to provide preliminary evidence of the role of criminal justice encounters in producing population-level racial health disparities.

Incarceration and Health—The first set of multivariable analyses focuses on the links between incarceration and the outcomes. First, I examine the associations between the incarceration-related measures (ever incarcerated, age of incarceration, duration of incarceration, and incarceration dosage) and the outcomes using generalized linear models (OLS for log CRP models and negative binomial models for depressive symptoms). This first set of models regresses the outcomes on incarceration history, age of incarceration, duration of incarceration, and incarceration dosage in a stepwise fashion while adjusting for the full set of covariates presented in Appendix A, providing a baseline association between incarceration and health.

In the second stage of the analysis, I use generalized linear models to examine the associations between incarceration and the outcomes, but limit my samples to strategic “treatment-control” groups. As discussed, isolating the health effects of incarceration can be difficult because of issues related to selection and unmeasured confounding. As such, the selection of a strategically appropriate comparison group is critical. In this second stage of the incarceration analysis, I use individuals who have been convicted, but not incarcerated, as the comparison group. The approach of using convicted but not incarcerated individuals as a comparison group in models attempting to isolate the effects of incarceration has been lauded (Massoglia and Warner 2011), and a growing number of studies use this approach (Apel and Sweeten 2010; Porter 2014). Because individuals who have been convicted but not incarcerated are “closest to the incarceration decision” (Apel and Sweeten 2010: 454), they are particularly useful as a control. In these analyses, I restrict the sample to only individuals who have been convicted of a crime and regress the outcomes on the incarceration-related measures, adjusting for the full set of covariates.

While restricting the analytic sample to respondents to those who have been convicted of a crime minimizes unmeasured heterogeneity, there may still be systematic differences between those who were never incarcerated and those who have spent time in a prison or jail. In order to further account for potential differences between the "treated" and "untreated" groups, the third stage of the incarceration analysis utilizes the restricted "treatment-control" groups and also includes inverse probability of treatment weighting (IPTW) with regression adjustment. IPTW involves calculating a propensity score for respondents, which is the probability of incarceration (including the timing, duration, and dosage of incarceration) conditional on a set of covariates (Rosenbaum and Rubin 1983). IPTW then uses the calculated propensity scores to weight observations to create a new pseudo-population in which treatment is no longer confounded by the covariates. IPTW balances the treatment assignment across the covariates by giving more or less weight to respondents with covariate histories that are under- or over-represented in the exposure groups (Robins 1999; Robins, Hernan, and Brumback 2000). As a result, in this study, exposure to incarceration—as well as timing, duration, and dosage of incarceration—behave as if they were randomized with respect to the observed covariates. In addition to IPTW, I also use a regression adjustment estimator. This strategy combines IPTW with regression modeling of the relationship between the covariates and outcomes. These models have the property of being doubly robust, so as long as either the model for the treatment or the outcome is correctly specified, the estimate of the effect of incarceration on health will be unbiased.

Using the IPTWs with regression adjustment, I model the "treatments" using logistic (for the binary incarceration history indicator) and multinomial (for the categorical age at first incarceration, duration of incarceration, and incarceration dosage measures) regression. I use OLS (for CRP) and negative binomial (for CESD) regression techniques to model the outcomes. The models for the treatments and outcomes include the same set of measures: respondent race, gender, and age; measures of neighborhood racial composition and socioeconomic conditions; family SES, mental health, and delinquency in adolescence; and indicators for whether the respondent ever had a parent incarcerated, was a repeat arrestee, or violent arrestee. Covariate balance statistics for the treatment-control groups before and after inverse probability of treatment weighting are in Appendix B. According to Rubin (2001), the standardized mean differences between two groups should generally be less than 0.25, and the variance ratios of the propensity scores in the two groups should be close to 1 and generally between 0.5 and 2. Results in Appendix B show that the standardized differences are all less than 0.25, and all variance ratios fall within acceptable ranges. Though the imbalances fall within acceptable ranges, there is still some imbalance in the sample after using the IPTWs, including in the gender and arrest variables. However, the covariance balance statistics presented in Appendix B only include the IPTW component of the estimators, and do not account for the additional regression adjustment implemented in the study. The IPTW estimators model the treatment to account for the nonrandom treatment assignment, while the regression adjustment models the outcome to further account for nonrandom treatment assignment. Together, these estimators more fully account for the nonrandom treatment assignment in the treatment-control samples than either strategy alone.

Pre-Incarceration Criminal Justice Contacts and Health—Next, I assess whether police stops, arrests, and convictions are associated with the psychophysiological outcomes and account for the associations between incarceration and health using generalized linear models (OLS models for log CRP; and negative binomial models for CESD). First, I regress the outcomes on the binary measures indicating contacts with the criminal justice system simultaneously, with basic adjustments for age, race, and gender. The second set of models build on these basic adjusted models to include the full set of covariates in Appendix A.

Criminal Justice Contacts and Racial Health Disparities: Mediation Analysis—Finally, I assess whether disparities in criminal justice contacts mediate racial disparities in the outcomes using parametric regression models for causal mediation analysis (Emsley and Liu 2013; Valeri and VanderWeele 2013). This technique involves estimating two models: a model for the mediator conditional on treatment and covariates and a model for the outcome conditional on treatment and covariates. The mediation analyses determine the proportion of the Black-White gaps in the outcomes mediated by contacts with the criminal justice system —including police stops, arrests, convictions, and incarceration—and offer evidence of the role of criminal justice contacts in producing population-level racial health disparities.

Analytic Samples—Analytic samples include respondents with complete data on the variables used in the analyses, as well as valid sampling weights. Sample sizes vary by outcome: log CRP (N=4,976) and depressive symptoms (N=5,488). For models using restricted “treatment-control” groups, I restrict the sample to respondents who have been convicted of a crime (CRP: N=585; depressive symptoms: N=634).

RESULTS

Descriptive Statistics

Table 1 presents descriptive statistics for the outcomes and key explanatory variables for the full sample as well as by incarceration history and race. Thirteen percent of individuals have been incarcerated, with greater numbers of individuals reporting having been stopped by police (19.8%) or arrested (26.5%). Of those who were previously incarcerated, most were first incarcerated when they were older than 18 years, and a majority reported only being incarcerated once and spending less than one year incarcerated. Formerly incarcerated individuals reported more depressive symptoms in both adolescence and young adulthood ($p<0.001$). Supplementary analyses revealed that the bivariate association between incarceration history and CRP was suppressed by gender; women have higher CRP than men on average but are less likely than men to be incarcerated. Supplementary analyses showed that after adjusting for gender differences in CRP, the association between incarceration history and inflammation was highly significant. Table 1 also reveals that, compared to Whites, Black young adults had higher CRP ($p=0.017$) and more depressive symptoms ($p<0.001$). Black individuals were also more likely than Whites to have been arrested ($p=0.032$) and incarcerated ($p=0.046$).

Incarceration and Health

For both outcomes, examination of the association between incarceration and health includes three stages: multivariable regression models with full analytic samples; multivariable regression models with “convicted only” treatment-control samples; and IPTW with regression adjustment with “convicted only” samples. For each stage of the analysis, I regress the outcomes on incarceration history, age at first incarceration, duration of incarceration, and incarceration dosage in a stepwise fashion.

Inflammation

A summary of the results from the multi-stage analyses examining the associations between incarceration and log CRP are in Models 1–12 of Table 2. In Table 2, results from all three stages of analyses reveal a consistent relationship between exposure to incarceration and inflammation, where formerly incarcerated individuals had higher levels of CRP than individuals who were never incarcerated. Model 1 includes results from the OLS regression models and indicates that formerly incarcerated individuals had higher CPR than those who have never been incarcerated, adjusting for the full set of covariates. In Model 5, I restricted the sample to only those who have been convicted, and results indicate that, compared to those who have been convicted of a crime but not incarcerated, those who were both convicted and incarcerated had higher CRP (β=0.230, p=0.035). Model 9, which uses the “convicted only” sample and utilizes the IPTW and regression adjustment strategy, provides similar though slightly attenuated results, with individuals who were both convicted and incarcerated have higher levels of inflammation than individuals who were convicted but not incarcerated.

Models 2, 6, and 10 of Table 2 assess whether and how the life course timing of incarceration is associated with CRP, and results offer no compelling evidence that the life course timing of incarceration shapes health risk. These findings were robust to alternative age cut points. Models 3, 7, and 11 of Table 2 assess the extent to which duration of incarceration is associated with CRP. Across all of these models, results reveal that individuals who were incarcerated for one year or more have the greatest inflammatory risk. In Model 11, after using the IPTW with regression adjustment with the convicted only sample, individuals who have been incarcerated for longer than one year have the highest levels of inflammation (β=0.300, p=0.013). In this way, duration of exposure to incarceration plays an essential role in producing divergent health risks within the population of formerly incarcerated individuals. Finally, Models 4, 8, and 12 include the measure of incarceration dosage, with results consistently showing that individuals who have been incarcerated once have the highest levels of inflammation.

Depressive Symptoms

Results of the incarceration-depressive risk models are in Models 13–24 of Table 2. Consistent with the CRP analyses, within each stage of analysis, I regress depressive symptoms on incarceration history (Models 13, 17, and 21), age at first incarceration (Models 14, 18, and 22), duration of incarceration (Models 15, 19, and 23), and incarceration dosage (16, 20, and 24). I find a significant association between incarceration history and depressive risk in Models 13 that is partially attenuated when the sample is

restricted to only those who have been incarcerated (Model 17). Results from Model 21, which combines the restricted treatment-control sample with the IPTW strategy, indicate no significant relationship between dichotomous exposure to incarceration and depressive risk.

Results from Models 14 and 18 offer some evidence incarceration at earlier ages is associated with heightened depressive risk. In Model 22, which includes the restricted sample of convicted respondents and implements the treatment weighting procedures, results offer little evidence that age of first incarceration shapes health risk, with incarceration before and after 18 both being associated with greater depressive risk.

Results from Model 15 indicate that incarceration is associated with worse mental health, regardless of duration. However, consistent with the CRP and immune function models, results from models with the restricted sample (Models 19 and 23) indicate that durations over one year are associated with more depressive symptoms.

Finally, results offer little consistent evidence that incarceration dosage differentially shapes depressive risk. In Model 20, greater numbers of incarceration experiences are associated with higher numbers of depressive symptoms (β=0.260, p=0.032), but this association is attenuated after using the IPTW strategy in Model 24.

Pre-Incarceration Criminal Justice Contacts and Health

In the next stage of the analysis, I examine how pre-incarceration contacts with the criminal justice system are associated with the markers of psychophysiological functioning. Results of these analyses are in Table 3. Models with the subscript “a” regress the outcomes on the measures of criminal justice contact simultaneously with basic adjustment for race, age, and gender, while models with the subscript “b” adjust for the full set of covariates.

Consistent with results from the incarceration models in Table 2, results show strong associations between incarceration history and health, where formerly incarcerated individuals have higher CRP and more depressive symptoms than never incarcerated individuals, net of other pre-incarceration contacts with the criminal justice system. For both outcomes, adjusting for the full set of covariates only slightly attenuates these associations. I find no associations between police stops or convictions and the outcomes. Models 1a and 1b reveal a negative association between arrest and inflammation when simultaneously adjusting for all measures of criminal justice contacts. Models 2a and 2b show that arrests are associated with more depressive symptoms, net of the other criminal justice contacts.

Mediation Analyses: Criminal Justice Contacts and Racial Health Disparities

Finally, I assessed the extent to which racial disparities in arrest and incarceration contribute to Black-White disparities in the outcomes using parametric regression models for causal mediation analysis. Given documented racial disparities in arrest and incarceration in Table 1, in these analyses I focused on estimating the contribution of disparities in arrest and incarceration to racial disparities in the outcomes. For depressive symptoms, the models indicated that Black individuals had more depressive symptoms than Whites; that arrests and incarceration were associated with increases in depressive symptoms; and that Blacks had greater risk of arrest and incarceration than Whites. Estimates of the natural indirect or

mediating effects indicated the average depressive score among Whites would increase by 1.01 symptoms if White young adults had similar levels of arrest as Black young adults (p=0.042). Similarly, results showed that the average depressive score among White young adults would increase by 1.01 symptoms if individuals had similar incarceration histories as Black young adults (p=0.084). These results indicate that Black-White disparities in arrest and incarceration are particularly salient sources of racial disparities in depressive risk. For CRP, results of mediation analyses failed to achieve statistical significance, as the Black-White disparities in CRP were no longer statistically significant after adjusting for the full set of covariates.

DISCUSSION

A growing body of research assesses the role of the criminal justice system in population health inequities. Still, questions about the psychophysiological mechanisms underlying the links between criminal justice contacts and health remain. Further, little attention has been given to how the life course timing, duration, or dosage of incarceration matter for health, and few studies provide rigorous tests for potential causality or consider how pre-incarceration contacts with the criminal justice system could confound incarceration-health links. By integrating life course concepts such as age, duration, and dosage of exposure, utilizing pre-disease markers of biological and psychological well-being, and employing treatment control designs, treatment weighting procedures, and causal mediation techniques, this study provides a nuanced and thorough assessment of the role of the criminal justice system in shaping health inequality. Together, findings from this study offer four key contributions to our understanding of the role of the criminal justice system in population health.

First, the results indicate that incarceration, in particular, is an essential driver of individual health and population health disparities. Across the several stages of analyses—which included multivariable regression models, a unique treatment-control sample, and treatment weighting procedures—findings revealed associations between incarceration and physiological inflammation, specifically, with formerly incarcerated individual having higher levels of CRP than those who were never incarcerated. Concerns about selection, causality, and confounding are abundant in studies of incarceration and health. However, findings from this study showed that using robust methods to better account for these biases produced consistent positive associations between incarceration and physiological inflammation, which speaks to the powerful influence that incarceration has on the lives and well-being of formerly incarcerated individuals. While the relationship between incarceration and inflammation were robust to a variety of outcome operationalizations and model specifications, the association between incarceration and depressive symptoms was slightly weaker. Results from Models 13 and 17 of Table 2 indicated that formerly incarcerated individuals had more depressive symptoms than never incarcerated individuals. In Model 21, which included inverse probability of treatment weighting with regression adjustment on the “convicted-only” sample, the association between incarceration and depressive symptoms faded. Still, as I describe below, results from Models 22 and 23 showed a link between duration of incarceration and depressive risk in the IPTW models with the treatment-control sample, indicating that consideration of the duration of

incarceration, in particular, is essential to understanding whether and how incarceration shapes health risk.

The robustness of the CRP models indicate that physiological inflammation may be an especially important physiological pathway linking incarceration to health among young adults. Research links chronic exposure to stressful events and environments to systemic inflammation and poor immune function (Everett et al. 2014). The physical and emotional environments of jails and prisons—which are characterized by solitude, isolation, and fear—may chronically activate physiological stress response systems—including inflammatory responses—in ways that irreparably alter the body's ability to maintain optimal health (Massoglia 2008; Porter 2014). The stigma and damage of social relationships that extends beyond one's sentence may serve to further exacerbate psychophysiological dysregulation (Schnittker and John 2007). Incarceration also exposes individuals to heightened infection risk (Hammett, Harmon, and Rhodes 2002), which may further contribute to the robust association between incarceration and inflammation. Taken together, findings from this study indicate that incarceration is a critical risk factor shaping young adult health, with the health consequences of incarceration continuing to shape well-being even after release.

Second, consistent with previous research (e.g., Del Toro et al. 2019; McFarland et al. 2019), findings show that the mental health impacts of the criminal justice system extend beyond incarceration to include pre-incarceration contacts, with results showing particularly strong associations between arrests and depressive risk. Though recent work by Sugie and Turney (2017) finds that arrest accounts for nearly half of the association between incarceration and mental health risk, findings from this study suggest that incarceration is a relatively independent driver of health, net of other contacts with the criminal justice system. Still, results from the depressive risk models in Table 3 provide evidence that arrests are also implicated in population health inequality. In Model 2b of Table 3, the magnitude of the association between arrest and depressive risk is similar to the coefficient estimate of parental incarceration, a well-documented driver of mental health risk that fundamentally alters the well-being and life chances of young people (Turney and Goodsell 2018). In this way, the primary and secondary stressors associated with police contacts—which can include trauma, stigma, job and financial losses, and anticipatory stress (Geller et al. 2014; Smith, Allen, and Danley 2007; Sugie and Turney 2017)—are important contributors to mental health risk. By contrast, results provide little evidence that pre-incarceration criminal justice contacts are associated with increased risk of inflammation, at least not in the long-term. It is possible that while pre-incarceration criminal justice encounters may not irreparably damage inflammatory function, these encounters could still have lasting effects on mental health. Together, findings from this study indicate that incarceration shapes health risk net of other criminal justice contacts, but that arrests, in particular, are also critical early-life drivers of mental health risk.

Third, while studies rarely consider the timing, duration, or dosage of incarceration, this study extends the literature by integrating these important life course concepts. Findings revealed little evidence that the life course timing of incarceration differentially affects health risk, though results from the depressive symptoms models provided somewhat mixed results. Insights from life course epidemiology indicate that early-life periods, including

adolescence and young adulthood, are particularly sensitive to social exposures, when stress-inducing experiences have particularly strong or long-lasting impacts on health (Ben-Shlomo and Kuh 2004). However, results from this study show that incarceration harms health, regardless of timing.

Results also revealed that duration of incarceration matters for health. Across the CRP and CESD models in Table 2, results indicated that individuals who were incarcerated for longer than one year had particularly high levels of inflammatory and depressive risk. In Model 12, I used the "treatment-control" sample with IPTWs and a regression adjustment estimator to more fully account for the measured and unmeasured factors that may confound incarceration-health links. Results still revealed strong links between incarceration duration and CRP. To put results from Model 12 in perspective, those who were convicted and incarcerated for more than one year had CRP levels that were nearly 1 mg/L higher than individuals who were convicted but not incarcerated and who were similar across a host of background characteristics. An increase of 1 mg/L increase is not negligible or insignificant. A CRP level of less than 1 mg/L is considered low risk, but a level of between 1 and 3 mg/L indicates moderate or intermediate risk, suggesting that a 1 mg/L increase in CRP can move individuals across clinical thresholds and greatly increase overall health risk. These results suggest that individuals' physiological and psychological well-being may be able to recuperate following shorter jail or prison stays, but longer exposures may irreparably harm one's psychophysiological functioning. Indeed, research indicates that repeated, chronic exposure to social stress diminishes the ability of psychophysiological systems to down-regulate (Cohen et al. 2012; Miller et al. 2002), which contributes to the malfunctioning of psychophysiological systems and increases disease and mortality risk from a host of causes (Friedman and Herd 2010). Findings from this study suggest that long-term exposure to the chaotic, isolating conditions in prisons and jails may serve to chronically up-regulate bodily stress response systems in ways that erode health over time. Individuals incarcerated for longer durations may also have greater difficulty repairing social ties or finding work upon release, which may be some of the secondary stressors linking longer prison and jail stays to health.

Incarceration dosage, or number of incarceration experiences, also matters for health. Results from the CRP models reveal that individuals who have been incarcerated once have greater health risk than those incarcerated multiple times, whereas results from the depressive risk models offer mixed evidence. It is possible that the first incarceration is a particularly salient stressor in the lives of individuals with especially grave consequences for health. After multiple incarcerations, individuals may "adapt" and be better equipped to handle the stress associated with incarceration (Porter and DeMarco 2017). It is also possible that those reporting higher incarceration dosage have experienced several jail—rather than prison—stays, which may relate to health in different ways. Because Add Health does not differentiate jail from prison stays or provide detailed incarceration histories, a more nuanced understanding of how incarceration spells shape health is not possible, though this is an important area for future research inquiry. Taken together, the results presented here are consistent with the notion that incarceration history is not a "dichotomous" experience (Porter and DeMarco 2017), with individuals having varying durations and dosages of incarceration in ways that matter for health and warrant consideration.

Finally, the results presented here indicate that criminal justice contacts are drivers of racial health disparities. Descriptive analyses in Table 1 showed that Black individualss were more likely than Whites to be arrested and incarcerated. Causal mediation analyses using parametric regression techniques further revealed that these criminal justice contacts were drivers of Black-White gaps in depressive risk, with estimates indicating that White young adults would have significantly higher levels of depressive risk if they had similar levels of criminal justice contacts as Black young adults. Together, these findings indicate that the criminal justice system is not only a driver of social and racial inequality in a general sense, but that it is also an essential determinant of racial health disparities.

Given their persistence across time and space, a wide body of literature seeks to understand the social determinants of racial health gaps. Racial disparities and biases across mutually reinforcing systems of housing, education, media, health care, earnings, and criminal justice compromise an integrated system of structural racism that generates racial gaps across a variety of outcomes, including health (Reskin 2012). Findings from this study indeed indicate that the criminal justice system, with its large and increasing role in determining the life chances of Black Americans, plays a particularly salient role in producing population health gaps. Racism is a fundamental determinant of racial health gaps (Phelan and Link 2015), and the present study shows that racial disparities in criminal justice contacts—including disproportionate contacts with police, jails, and prisons—provide a critical link between macro-level racial inequality and individual health and offers new insights into the mechanisms through which racism generates population health gaps.

This study is not without limitations. First, while the incarceration analyses include a variety of techniques aimed at improving causal inference, the examinations of pre-incarceration contacts and health use multivariable regression models, which are more vulnerable to issues of selection, endogeneity, and confounding. This study was among the first to use nationally-representative, individual-level data to simultaneously examine how police stops, arrests, and convictions impact pre-disease markers of physiological and psychological functioning, so future research should build on the evidence provided here by including more robust tests for causality. Second, because of data limitations, I was not able to distinguish jail from prison stays or provide more detailed assessments of how various incarceration spells differentially shape health risk. Similarly, I was not able to assess how the life course timing or frequency of pre-incarceration contacts might be implicated in health risk. Together, findings from this study point to the need for social surveys to include more detailed data on criminal justice contacts, including information on the life course timing, duration, and repetition of these contacts. Third, analytic samples include only Black and White survey respondents because of concerns about sample size. However, the processes described in this study extend to other racial and ethnic groups, which should be examined in future research. Similarly, future research should assess how the links between criminal justice contacts and health may vary by gender. Supplementary analyses revealed that men experience more contacts with the criminal justice system than women, and it is possible that the impacts of these contacts on health risk could vary by gender. Fourth, as longitudinal biomarker and criminal justice contact data becomes available, the ability to make causal claims about how criminal justice contacts affect health will only be improved. Finally, this study focuses on the links between individual-level criminal justice contacts and health, but research should

continue to interrogate how vicarious and indirect exposures to the criminal justice system—including familial contacts and neighborhood policing and incarceration contexts—contribute to population health inequality.

Recent scholarship has highlighted the role of police violence in shaping population health patterns and generating particularly stark racial disparities in mortality risk (Edwards, Lee, and Esposito 2019). Findings from this study show that even those contacts that do not result in death or other forms of acute physical injury have lasting impacts on health. In many ways, contacts with the criminal justice system serve as repeated, chronic stressors in the lives of individuals, increasing levels of ongoing stress, anxiety, stigma, and fear, in ways that upregulate psychological and physiological dysregulation and erode health over time. Importantly, the most structurally vulnerable and disadvantaged young people—including Black adolescents and young adults and young people from poor neighborhoods and families—are also the most likely to experience criminal justice contacts, which further exacerbates health inequities early in the adult life course. Given the expansive size and scope of the penal system in the U.S., continued efforts to understand the role of criminal justice system in shaping the health and well-being of individuals is essential to improving knowledge of the determinants of population health generally, and racial health gaps, in particular.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

ACKNOWLEDGEMENTS

I thank Y. Claire Yang, Karolyn Tyson, Robert Hummer, Kathleen Mullan Harris, and Anthony Perez for comments on earlier drafts of this paper. I am grateful to the Population Studies Center at the University of Pennsylvania for general support (National Institutes of Health's (NIH)'s Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH grant number: R24 HD044964). This research uses data from Add Health, a program project directed by Kathleen Mullan Harris and designed by J. Richard Udry, Peter S. Bearman, and Kathleen Mullan Harris at the University of North Carolina at Chapel Hill, and funded by grant P01-HD31921 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, with cooperative funding from 23 other federal agencies and foundations. Information on how to obtain the Add Health data files is available on the Add Health website (http://www.cpc.unc.edu/addhealth). No direct support was received from grant P01-HD31921 for this analysis.

AUTHOR BIOGRAPHY

Courtney E. Boen is an assistant professor in the Department of Sociology and research associate in the Population Studies Center and Population Aging Research Center at the University of Pennsylvania. Dr. Boen's research focuses on the social determinants of population health inequality, with a particular focus on the role of racism in shaping health disparities across the life course.

References

Adkins Daniel E., Wang Victor, Dupre Matthew E., Van den Oord Edwin JCG, and Elder Glen H. Jr. 2009. "Structure and stress: Trajectories of depressive symptoms across adolescence and young adulthood." Social Forces 88(1): 31–60.

Alexander Michelle. 2012. The New Jim Crow: Mass Incarceration in the Age of Colorblindness.The New Press.

Aneshensel Carol S., Rutter Carolyn M., and Lachenbruch Peter A.. 1991. “Social Structure, Stress, and Mental Health: Competing Conceptual and Analytic Models.” American Sociological Review 56(2):166–178.

Apel Robert and Sweeten Gary. 2010. “The Impact of Incarceration on Employment during the Transition to Adulthood.” Social Problems 57(3):448–479.

Ben-Shlomo Yoav, and Kuh Diana. 2002. “A Life Course Approach to Chronic Disease Epidemiology: Conceptual Models, Empirical Challenges and Interdisciplinary Perspectives.” International Journal of Epidemiology 31(2):285–293. [PubMed: 11980781]

Cohen Sheldon, Denise Janicki-Deverts, Doyle William J., Miller Gregory E., Frank Ellen, Rabin Bruce S., and Turner Ronald B.. 2012. “Chronic Stress, Glucocorticoid Receptor Resistance, Inflammation, and Disease Risk.” Proceedings of the National Academy of Sciences of the United States of America 109(16):5995–5999. doi: 10.1073/pnas.1118355109 [doi]. [PubMed: 22474371]

Comfort Megan. 2009. Doing time together: Love and family in the shadow of the prison. University of Chicago Press.

Del Toro Juan, Tracey Lloyd, Buchanan Kim S., Robins Summer Joi, Bencharit Lucy Zhang, Meredith Gamson Smiedt, Reddy Kavita S., Pouget Enrique Rodriguez, Kerrison Erin M., and Goff Phillip Atiba 2019. “The criminogenic and psychological effects of police stops on adolescent black and Latino boys.” Proceedings of the National Academy of Sciences 116(17): 8261–8268.

Edwards Frank, Lee Hedwig, and Esposito Michael. 2019. “Risk of being killed by police use of force in the United States by age, race–ethnicity, and sex.” Proceedings of the National Academy of Sciences 116(34): 16793–16798.

Emerging Risk Factors Collaboration. 2010. “C-Reactive Protein Concentration and Risk of Coronary Heart Disease, Stroke, and Mortality: An Individual Participant Meta-Analysis.” The Lancet 375(9709):132–140.

Emsley Richard, and Liu Hanhua. 2013. PARAMED: Stata module to perform causal mediation analysis using parametric regression models.

Esposito Michael H.; Lee Hedwig; Hicken Margaret T.; Porter Lauren C.; & Herting Jerald R. 2017. “The consequences of contact with the criminal justice system for health in the transition to adulthood.” Longitudinal and Life Course Studies 8(1): 57–74. [PubMed: 28781613]

Everett Bethany G., Rosario Margaret, McLaughlin Katie A., and Austin S. Bryn. 2014. “Sexual orientation and gender differences in markers of inflammation and immune functioning.” Annals of Behavioral Medicine 47(1): 57–70. [PubMed: 24347405]

Geller Amanda, Fagan Jeffrey, Tyler Tom and Link Bruce G.. 2014. “Aggressive Policing and the Mental Health of Young Urban Men.” American Journal of Public Health 104(12):2321–2327. [PubMed: 25322310]

Glaser Ronald and Kiecolt-Glaser Janice K.. 2005. “Stress-Induced Immune Dysfunction: Implications for Health.” Nature Reviews Immunology 5(3):243–251.

Hammett Theodore M., Harmon Mary Patricia, and Rhodes William. 2002. “The Burden of Infectious Disease among Inmates of and Releasees from US Correctional Facilities, 1997.” American Journal of Public Health 92(11):1789–1794. [PubMed: 12406810]

Harris KM, Halpern CT, Whitsel E, Hussey J, Tabor J, Entzel P and and Udry JR. 2009. “The National Longitudinal Study of Adolescent to Adult Health: Research Design.”, Retrieved 08/11, 2017. (http://www.cpc.unc.edu/projects/addhealth/design).

Kaeble Danielle and Glaze Lauren. 2016. “Correctional Population in the United States, 2015.” Washington D.C.: Bureau of Justice Statistics.

Kiecolt-Glaser Janice K. and Glaser Ronald. 2002. “Depression and Immune Function: Central Pathways to Morbidity and Mortality.” Journal of Psychosomatic Research 53(4):873–876. [PubMed: 12377296]

Kinner Stuart A., and Young Jesse T.. 2018. “Understanding and improving the health of people who experience incarceration: An overview and synthesis.” Epidemiologic *reviews* 40(1): 4–11.

Kirk David S., and Wakefield Sara. 2018. “Collateral consequences of punishment: a critical review and path forward.” Annual Review of Criminology.

Kirk David S. 2018. “The Collateral Consequences of Incarceration for Housing.” In Handbook on the Consequences of Sentencing and Punishment Decisions (pp. 53–68). Routledge.

Lee Hedwig and Wildeman Christopher. 2013. “Things fall apart: Health consequences of mass imprisonment for African American women.” The Review of Black Political Economy 40(1): 39–52.

London Andrew S. and Myers Nancy A.. 2006. “Race, Incarceration, and Health A Life-Course Approach.” Research on Aging 28(3):409–422.

Massoglia Michael and Remster Brianna. 2019. “Linkages between incarceration and health.” Public Health Reports 134(1-S):: 8S–14S. [PubMed: 31059410]

Massoglia Michael and Warner Cody. 2011. “The Consequences of Incarceration.” Criminology & Public Policy 10(3):851–863.

Massoglia Michael. 2008. “Incarceration as Exposure: The Prison, Infectious Disease, and Other Stress-Related Illnesses.” Journal of Health and Social Behavior 49(1):56–71. [PubMed: 18418985]

McFarland Michael J., Taylor John, McFarland Cheryl AS, and Friedman Katherine L.. 2018. “Perceived Unfair Treatment by Police, Race, and Telomere Length: A Nashville Community-based Sample of Black and White Men.” Journal of Health and Social Behavior 59(4): 585–600. [PubMed: 30417689]

McFarland Michael J., Geller Amanda, and McFarland Cheryl. 2019. “Police contact and health among urban adolescents: The role of perceived injustice.” Social Science & Medicine 238.

Mitchell Ojmarrh and Caudy Michael S.. 2015. “Examining Racial Disparities in Drug Arrests.” Justice Quarterly 32(2):288–313.

National Research Council. 2014. The Growth of Incarceration in the United States: Exploring Causes and Consequences. Washington, DC: The National Academies Press.

New York Civil Liberties Union. 2016. “NYPD Stop and Frisk Data.”, Retrieved January 22, 2016. (http://www.nyclu.org/content/stop-and-frisk-data).

Pavalko Eliza K. and Willson Andrea E.. 2011. “Life course approaches to health, illness and healing.” Pp. 449–464 in Handbook of the sociology of health, illness, and healing.”Life course approaches to health, illness and healing.” Springer.

Perreira Krista M., Deeb-Sossa Natalia, Harris Kathleen Mullan, and Bollen Kenneth. 2005. “What are we measuring? An evaluation of the CES-D across race/ethnicity and immigrant generation.” Social Forces 83(4): 1567–1601.

Pettit Becky and Western Bruce. 2004. “Mass Imprisonment and the Life Course: Race and Class Inequality in US Incarceration.” American Sociological Review 69(2):151–169.

Phelan Jo C., & Link Bruce G. 2015. “Is racism a fundamental cause of inequalities in health?” Annual Review of Sociology 41:311–30.

Porter Lauren C. 2014. “Incarceration and Post-Release Health Behavior.” Journal of Health and Social Behavior 55(2):234–249. doi: 55/2/234 [pii]. [PubMed: 24872468]

Porter Lauren C., and DeMarco Laura M.. 2019. “Beyond the dichotomy: Incarceration dosage and mental health.” Criminology 57(1): 136–156.

Porter Lauren C., and Novisky Meghan A.. 2017. “Pathways to depressive symptoms among former inmates.” Justice Quarterly 34(5): 847–872.

Radloff Lenore S. 1977. “The CES-D Scale: A Self-Report Depression Scale for Research in the General Population.” Applied Psychological Measurement 1(3):385–401.

Reskin Barbara. 2012. “The Race Discrimination System.” Annual Review of Sociology 38:17–35.

Robins James M. 1999. “Association, Causation, and Marginal Structural Models.” Synthese 121(1):151–179.

Robins James M., Hernan Miguel A. and Brumback Babette. 2000. “Marginal Structural Models and Causal Inference in Epidemiology.” Epidemiology:550–560. [PubMed: 10955408]

Rosenbaum Paul R. and Rubin Donald B.. 1983. “The Central Role of the Propensity Score in Observational Studies for Causal Effects.” Biometrika 70(1):41–55.

Rubin Donald B 2001. “Using propensity scores to help design observational studies: Application to the tobacco litigation. Health Services & Outcomes Research Methodology.” 2:169–188.

Schnittker Jason and John Andrea. 2007. “Enduring Stigma: The Long-Term Effects of Incarceration on Health.” Journal of Health and Social Behavior 48(2):115–130. [PubMed: 17583269]

Sewell Abigail A., Jefferson Kevin A. and Lee Hedwig. 2016. “Living Under Surveillance: Gender, Psychological Distress, and Stop-Question-and-Frisk Policing in New York City.” Social Science & Medicine 159:1–13. [PubMed: 27155224]

Smith William A., Allen Walter R. and Danley Lynette L.. 2007. ““Assume the Position … You Fit the Description” Psychosocial Experiences and Racial Battle Fatigue among African American Male College Students.” American Behavioral Scientist 51(4):551–578.

Spaulding Anne C., Seals Ryan M., McCallum Victoria A., Perez Sebastian D., Brzozowski Amanda K., and Steenland N. Kyle. 2011. “Prisoner Survival Inside and Outside of the Institution: Implications for Health-Care Planning.” American Journal of Epidemiology 173(5):479–487. doi: 10.1093/aje/kwq422 [doi]. [PubMed: 21239522]

Marit Rehavi, M., and Starr Sonja B.. 2012. “Racial Disparity in Federal Criminal Charging and its Sentencing Consequences.” University of Michigan Law School, Program in Law and Economics Working Paper Series, Working Paper No. 12–002.

Steptoe Andrew, Hamer Mark, and Chida Yoichi. 2007. “The effects of acute psychological stress on circulating inflammatory factors in humans: a review and meta-analysis.” Brain, Behavior, and Immunity 21(7): 901–912.

Sugie Naomi F. and Turney Kristin. 2017. “Beyond Incarceration: Criminal Justice Contact and Mental Health.” American Sociological Review 82(4):719–743.

The Sentencing Project. 2017. Trends in U.S. Corrections. Washington, D.C.:.

Testa Alexander and Jackson Dylan B.. 2019. “Food insecurity among formerly incarcerated adults.” Criminal Justice and Behavior 46(10): 1493–1511.

Turney Kristin, and Goodsell Rebecca. 2018. “Parental incarceration and children’s wellbeing.” The Future of Children 28(1): 147–164.

U.S. Department of Justice. 2013. Uniform Crime Report: Crime in the United States, 2012. Washington, DC: Federal Bureau of Investigation.

Valeri Linda, and VanderWeele Tyler J.. 2013. “Mediation analysis allowing for exposure–mediator interactions and causal interpretation: Theoretical assumptions and implementation with SAS and SPSS macros.” Psychological Methods 18(2): 137. [PubMed: 23379553]

Wakefield Sara and Uggen Christopher. 2010. “Incarceration and Stratification.” Annual Review of Sociology 36:387–406.

Western Bruce and Wildeman Christopher. 2009. “The Black Family and Mass Incarceration.” The Annals of the American Academy of Political and Social Science 621(1):221–242.

Wildeman Christopher and Muller Christopher. 2012. “Mass Imprisonment and Inequality in Health and Family Life.” Annual Review of Law and Social Science 8:11–30.

Wildeman Christopher. 2011. “Invited Commentary: (Mass) Imprisonment and (Inequities in) Health.” American Journal of Epidemiology 173(5):488–491. [PubMed: 21239521]

Zamble Edward. 1992. “Behavior and adaptation in long-term prison inmates: Descriptive longitudinal results.” Criminal Justice and Behavior 19(4): 409–425.

Table 1.

Descriptive Statistics by Incarceration History and Race

	Full sample	*By Incarceration History*			*By Race*		
		Never incar.	Formerly incar.		White	Black	
	Mean/prop.	Mean/prop.	Mean/prop.	*p-value*	Mean/prop.	Mean/prop.	*p*-value
Outcomes							
C-reactive protein (log + 1)	1.305	1.304	1.308	0.939	1.287	1.400	0.017
Depressive symptoms (0–15)	2.442	2.357	3.005	<0.001	2.337	2.961	<0.001
Incarceration history							
Formerly incarcerated (1=yes)	0.130	-	-	-	0.124	0.157	0.046
Age at first incarceration *(among formerly incarcerated)*							
Under age 18 years	0.193	-	-	-	0.198	0.173	0.084
18 years or older	0.807	-	-	-	0.801	0.827	
Duration of incarceration *(among formerly incarcerated)*							
Less than one year	0.530	-	-	-	0.532	0.524	0.336
One year or longer	0.469	-	-	-	0.468	0.476	
Incarceration dosage *(among formerly incarcerated)*							
Once	0.593	-	-	-	0.605	0.545	0.071
Two or more times	0.407	-	-	-	0.395	0.455	
Pre-incarceration criminal justice contacts							
Ever stopped by police	0.198	0.150	0.450	<0.001	0.208	0.148	0.001
Ever arrested	0.265	0.160	1.000	<0.001	0.255	0.316	0.032
Ever convicted	0.121	0.050	0.570	<0.001	0.120	0.129	0.922
Covariates							
Race (1=Black)	0.168	0.162	0.203	0.046	-	-	-
Age	14.623	14.611	14.709	0.301	14.589	14.799	0.386
Gender (1=female)	0.519	0.557	0.269	<0.001	0.510	0.570	0.029
Family SES in adolescence	0.080	0.103	−0.080	<0.001	0.128	−0.163	<0.001
Neighborhood racial composition in adolescence (% non-White)	16.790	16.426	19.229	0.083	8.564	57.674	<0.001
Neighborhood economic disadvantage in adolescence	0.804	0.791	0.889	0.216	0.521	2.218	<0.001
Depressive symptoms in adolescence	2.889	2.831	3.275	0.002	2.814	3.258	0.002

	Full sample	*By Incarceration History*			*By Race*		
		Never incar.	**Formerly incar.**		**White**	**Black**	
	Mean/prop.	**Mean/prop.**	**Mean/prop.**	***p-value***	**Mean/prop.**	**Mean/prop.**	***p*-value**
Delinquency in adolescence	0.087	0.078	0.150	<0.001	0.087	0.087	0.881
Repeat arrestee	0.122	0.052	0.592	<0.001	0.109	0.183	0.002
Violent arrestee	0.034	0.016	0.151	<0.001	0.029	0.059	0.004
Parental incarceration	0.148	0.127	0.29	<0.001	0.134	0.219	<0.001

Notes: p-value of difference in between never incarcerated and formerly incarcerated and Blacks and Whites; two-tailed test. Measures of pre-incarceration criminal justice contact are not mutually exclusive. Sample size (N=5,488) based on analytic sample for depressive symptoms, with exception of C-reactive protein (N=4,976). Descriptive statistics are weighted and adjust for survey design effects.

Table 2.

	CRP (log)							*CESD*							
	: OLS Regression[a]			**Stage 2: Treatment-Control[b]**				**Stage 3: IPTW with RA[c]**				**Stage 1: NB Regression[a]**			
	M2	**M3**	**M4**	**M5**	**M6**	**M7**	**M8**	**M9**	**M10**	**M11**	**M12**	**M13**	**M14**	**M15**	**M16**
				0.230 *				0.204 †				0.169 **			
				(0.108)				(0.111)				(0.049)			
	031				−0.007				−0.030				0.251 †		
	.77)				(0.215)				(0.234)				(0.144)		
	90 †				0.073				−0.094				0.126		
	.08)				(0.159)				(0.113)				(0.114)		
		0.082				0.171				0.103				0.170 **	
		(0.071)				(0.126)				(0.116)				(0.055)	
		0.246 **				0.290 *				0.300 *				0.186 *	
		(0.087)				(0.115)				(0.121)				(0.073)	
			0.152 *				0.319 **				0.309 **				0.151 **
			(0.065)				(0.114)				(0.118)				(0.055)
			0.111				0.074				−0.144				0.218 *
			(0.092)				(0.134)				(0.111)				(0.095)

	Stage 2: Treatment-Control[b]				**Stage 3: IPTW with RA[c]**			
	M17	**M18**	**M19**	**M20**	**M21**	**M22**	**M23**	**M24**
	0.201 †				0.265			
	(0.103)				(0.281)			
		0.528 *				1.563 †		
		(0.207)				(0.894)		
		0.130				0.728 *		
		(0.120)				(0.357)		
			0.163				0.266	
			(0.129)				(0.317)	
			0.301 *				0.596 †	
			(0.116)				(0.349)	
				0.150				0.194
				(0.116)				(0.306)
				0.260 *				0.628
				(0.120)				(0.335)

J Health Soc Behav. Author manuscript; available in PMC 2021 September 01.

***$p<0.001$;

**$p<0.01$;

*$p<0.05$;

†$p<0.1$

a: Results of the OLS regression (CRP; N=4,976) and negative binomial (CESD; N=5,488) models. Coefficients and standard errors presented. Models are weighted and adjust for age, gender, race, family SES at Wave I, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, violent arrestee, and parental incarceration.

b: Results of the regression models that include only those convicted of a crime (CRP: N=585; CESD: N=634). Coefficients and standard errors presented. Models are weighted and adjust for age, gender, race, family SES at Wave I, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, violent arrestee, and parental incarceration.

c: Results of the models using inverse probability of treatment weighting with a regression adjustment estimator. Average treatment effects (ATE) and standard errors presented. Sample is restricted to only those convicted of a crime (CRP: N=592; CESD: N=641). The models predicting the "treatment" (e.g., incarceration history, age at first incarceration, duration of incarceration, incarceration dosage) and outcomes include age, gender, race, family SES at Wave I, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, violent arrestee, and parental incarceration.

d: The reference group is never incarcerated.

Table 3.

Criminal Justice Contacts and Health

	CRP (log)		CESD	
	Model 1a	**Model 1b**	**Model 2a**	**Model 2b**
Stopped by police	−0.022	−0.001	0.073	0.060
	(0.048)	(0.047)	(0.044)	(0.046)
Arrested	−0.149**	−0.146**	0.146*	0.117*
	(0.046)	(0.047)	(0.056)	(0.055)
Convicted	0.089	0.090	−0.080	−0.091
	(0.066)	(0.066)	(0.070)	(0.069)
Incarcerated	0.207***	0.181**	0.216**	0.162**
	(0.058)	(0.059)	(0.063)	(0.059)

Note: Coefficient estimates and standard errors presented.

*** p<0.001;

** p<0.01;

* p<0.05;

† p<0.1.

Models “a” present results of weighted OLS (for log CRP; N=4,976) and negative binomial (for CESD; N=5,488) regression where outcomes are regressed on the measures of criminal justice contact jointly with basic adjustment for race, age, and gender. Models “b” present results of weighted models where outcomes are regressed on the measures of criminal justice contact jointly with adjustment for race, age, gender, family SES at Wave I, neighborhood racial composition at Wave I, neighborhood economic disadvantage at Wave I, depressive symptoms at Wave I, delinquency at Wave I, repeat arrestee, violent arrestee, and parental incarceration.