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The relationship between incarceration and premature adult mortality: Gender specific evidence

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Abstract

We examine the relationship between incarceration and premature mortality for men and women. Analyses using the National Longitudinal Survey of Youth (NLSY79) reveal strong gender differences. Using two different analytic procedures the results show that women with a history of incarceration are more likely to die than women without such a history, even after controlling for health status and criminal behavior prior to incarceration, the availability of health insurance, and other socio-demographic factors. In contrast, there is no relationship between incarceration and mortality for men after accounting for these factors. The results point to the importance of examining gender differences in the collateral consequences of incarceration. The results also contribute to a rapidly emerging literature linking incarceration to various health hazards. Although men constitute the bulk of inmates, future research should not neglect the special circumstances of female former inmates and their rapidly growing numbers.

Keywords

Incarceration; Health; Gender; Mortality

1. Introduction

In recent decades, the expansion of the prison system has become one of the defining features of American society. Over three decades, the size of the prison population has increased more than sevenfold. At present, there are well over two million Americans incarcerated in state and federal prisons or jails. Although the average length of a sentence has increased as well, most prisoners are eventually released, creating a cycle of entry and exit affecting large numbers of people. In any given year approximately 700,000 individuals are released from prison, resulting in a large number of community members having some

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contact with the correctional system in their lifetime. Some characterize this group as sufficiently large and distinctive to constitute a “felon class” (Uggen et al., 2006).

Social scientists have investigated many of the negative consequences of a prison sentence with renewed interest. Research has linked incarceration to increased likelihood of divorce (Hagan and Dinovitzer, 1999; Lopoo and Western, 2005), negative parent–child relationships (Pattillo et al., 2004), and diminished wages and employment opportunities (Pager, 2003; Western, 2002). Health has recently been added to the list, providing an important new direction for future research. Research has linked incarceration to infectious disease (Massoglia, 2008a), chronic health problems (Schnittker and John, 2007) and poor mental health (see Haney, 2003 for a review). Other studies have found a relationship between the number of former inmates within a community and rates of infectious disease, including tuberculosis (Farmer, 2002) and HIV/AIDS (Johnson and Raphael, 2009).

Although it is already clear that prison is negatively related to health, a number of elements are still missing from the empirical literature. For one, research has done far more to explore assorted indicators of morbidity than it has to explore mortality (see Patterson, 2013 for a notable exception). This is perhaps surprising given that mortality is the subject of a well-established literature of its own, but its neglect likely reflects some aspects of incarceration that steer researchers toward chronic disease and mental health rather than death. For one, the prison boom is a recent phenomenon and the prison population is relatively young, making death a rare event and, therefore, difficult to observe in numbers sufficient for statistical power. In addition, much of the attention has focused on a handful of mechanisms immediately related to imprisonment, including stress and infectious disease, a focus that renders mortality a more distant outcome and perhaps a less interesting one as well.

More importantly, very few studies have seriously explored gender-differences in the health consequences of incarceration (Steward et al., 2004). Indeed, many studies focus exclusively on men with little discussion of potential gender-specificity (Bird 2004; Joukamaa, 1998; Seaman et al., 1998). This is an especially important limitation given that research in other areas has pointed to gender differences in the experience and consequences of incarceration, differences that are likely relevant to health (see Chesney-Lind, 1997; Lindquist and Lindquist, 1997; Rafter, 1990). The actual role of gender remains unclear—incarceration could plausibly have more or less of an effect on women than it does on men—but it is already clear that gender is relevant to understanding the effects of incarceration and that, without considering gender differences, scholars should be cautious about over-generalizing their findings. Furthermore, recent trends are likely to make gender-differences even more relevant. Although women represent a small fraction of the total inmate population, the number of women in prison has grown (Bloom and Chesney-Lind, 2003).

In this study, we use the National Longitudinal Survey of Youth (NLSY79) to examine the relationship between incarceration and premature adult mortality (mortality between the ages of 24 and 47). The NLSY79 is useful for our purposes. Incarceration is rare among women, as is premature mortality, but the NLSY79 sample size is large enough to estimate gender differences reliably, as we will demonstrate shortly. In addition the NLSY79 includes most of the selective forces that might link incarceration to mortality in a spurious fashion.

These include education, income, drug use, and a history of violence prior to incarceration, but they also include more direct forms of spuriousness such as health status prior to incarceration. Altogether these variables allow us to separate the effects of incarceration from the conditions that predate imprisonment and lead to worse health. Finally, the NLSY data collection period spans almost 30 years in the lives of its respondents, allowing for a much longer period of observation than in previous studies. In short, our study is well positioned to add substantially to the literature.

2. Previous research linking incarceration and health

Despite the rise of incarceration and recognition of its various negative consequences, social scientists have been relatively slow to consider the impact of incarceration on health. Other professionals were quicker to the topic. Correctional officials have long recognized the health needs of inmates (e.g. Spaulding et al., 2002; Weiner and Anno, 1992) and some early reports on the high levels of infectious disease in prisons came from journalists (Herivel and Wright, 2002). Since then a number of studies have begun to explore the topic empirically and, in the fashion of social scientists, with an eye toward inferring whether the relationship between incarceration and health is causal. This research is generally consistent with early claims that incarceration impairs health, but also reveals considerable complexity and, at least implicitly, highlights the importance of gender.

Studies have generally employed one of two strategies for understanding the incarceration-health relationship. Some studies have compared mortality rates in prisons to mortality rates among demographically similar populations who are not incarcerated. In general, these studies find that prisons produce a short term protective effect on mortality by removing high-risk persons from dangerous environments and providing inmates with health care (see for instance, Patterson, 2010; Sattar, 2001). A similar protective effect has been found with respect to morbidity, especially among prisoners from disadvantaged backgrounds (Schnittker and John, 2007).

The consequences of incarceration upon release, however, are very different. Here, too, there are few studies, and what exists has been drawn from diverse contexts, but these studies are generally consistent in their conclusions (see, e.g. Hobbs et al., 2006; Steward et al., 2004; Joukamaa, 1998). Using data from Australia, for example, Hobbs et al. (2006) find that those with a history of incarceration have mortality rates at least twice that of those who have never been incarcerated. Similarly, Seaman et al. (1998) find that injection drug users in the United Kingdom have an unusually high risk of death from overdose following release. Using data from the U.S., Binswanger et al. (2007) find that the risk of death is 3.5 times higher among former inmates than among community members without a prison record, with an especially high risk of death occurring shortly after release, particularly because of drug overdoses.

Although this body of research is innovative, valuable, and consistent in its conclusions, it has at least three limitations (Kinner, 2010). First, studies have generally used an incomplete set of control variables. In some instances, these studies are more interested in drawing attention to an *association*, rather than arguing strongly for an *effect*. Yet without adequate

control variables it is difficult to infer where the risk for premature mortality among former inmates comes from and what mechanisms might account for it. Prisons are, of course, highly selective, and many of the risk factors for incarceration are also risk factors for mortality, including poverty, drug use, involvement with violence, and limited access to social services, including health care. To be sure, studies do control for some of these factors, but even the best studies (see Dirkzwager et al., 2012 analysis of Dutch respondents) rarely control for the full set of potential confounders, leaving the nature of the relationship uncertain. This limitation is not insignificant: according to some skeptical reviews of the literature, prior research “fails to capture the complexity of the circumstances surrounding many unnatural deaths” and, for that reason, is “severely limited” (Kinner, 2010, 1555).

Second, many studies use unusually focused samples. Some, for example, focus only on drug users (Bird, 2004; Seaman et al., 1998), while others focus only on inmates from a single institution (Seaman et al., 1998; Verger, Rotily, Prudhomme, and Bird, 2003) or state (Binswanger et al., 2007). This focus often emerges as a matter of research design. A popular research design, for example, is to link administrative incarceration data with mortality records (Merrall et al., 2010). Linkages of this sort provide an opportunity to study an otherwise inaccessible population, but they have limitations. In the context of regression models, the estimated effect of incarceration on mortality might be peculiar to a subsample, context, or institution, but can easily be misinterpreted as the average effect of incarceration for a much larger population. Misinterpretation of this sort is more likely in a debate where little is known about the effects of prisons in general. Given these concerns, some researchers have called for less reliance on administrative data and more use of representative samples, but such data are hard to find (Kinner, 2010).

Finally, research has generally employed relatively short follow-up periods, often only a few weeks after release (Krinsky et al., 2009; Merrall et al., 2010). As noted by others (Kinner, 2010) this short time period might be good for capturing traumatic deaths, which are not uncommon among former inmates, but it is less useful for capturing deaths due to more chronic conditions, whose effects emerge slowly over time. These causes of death are important too. Research in Australia, for instance, found elevated risk of mortality a decade past release and further found that these deaths are due to a variety of high-prevalence conditions, including cardiovascular disease and cancer (Steward et al., 2004; Kariminia et al., 2007). These causes of death invoke a different set of mechanisms linking incarceration and mortality.

3. Mechanisms linking incarceration and mortality

Assuming there is an effect of incarceration that withstands controls, there are two sets of mechanisms that could explain it. First, the prison environment itself may put inmates at risk for poor health. Infectious disease, for example, is highly prevalent in prisons. Although many inmates are already infected when they begin their sentence, confinement and regular contact increases the likelihood of contagion to those who are not infected (Massoglia and Schnittker, 2009). At the same time, we know from a variety of studies that the prison environment is stressful (Haney, 2003; Kruttschnitt and Gartner, 2005; Lindquist and Lindquist, 1997; Schmid and Jones, 1991). Even if inmates are healthy relative to a

comparable non-institutionalized population, the strategies they use to psychologically adjust to life behind bars, including hyper vigilance, withdrawal, and aggression, are often ill-suited to reintegration (see Haney, 2006). Once they return to the community, these strategies could increase the risk for stress-related conditions, including cardiovascular disease and substance abuse, as well as increase the risk for poor health behavior (e.g., smoking) (Aneshensel, 1999; Baum, Garofalo and Yali, 1999; Massoglia, 2008a; Pearlin, 1989). Post-incarceration stress may also lead some to suicide (Tremblay and Pare, 2003).

Second, incarceration could be linked to mortality through assorted other challenges of reintegration. Following release, former inmates often have difficulty maintaining relationships and securing stable employment (e.g. Pager, 2003; Petersilia, 2000; Lopoo and Western, 2005; Massoglia et al., 2011), both of which are related to health (Ross et al., 1990; Ross and Mirowsky, 1995). In the U.S., even when a former inmate is able to find steady employment, the job is unlikely to provide health benefits. The stigma of incarceration could also lead to poor health more directly through, for example, discrimination and diminished social status (Braman, 2004; Schnittker and McLeod, 2005; see Williams et al., 2003 on the health effects of discrimination).

The challenges of re-integration go beyond housing, relationships, and employment. Regardless of the restrictions inmates face post-release, former inmates nonetheless have considerably more autonomy than they had while incarcerated. This autonomy can lead to a host of problems, some related to a return to illegal behavior. Drug use, for example, is quite common during the period immediately after release and can lead to drug overdose insofar as incarceration reduces drug resistance and familiarity with appropriate drug dosing. In addition, former inmates have to manage other aspects of their lives, including medical care. With new-found independence, this can be challenging. Research has shown that adherence to HIV therapy declines after release (Milloy et al., 2011; Small et al., 2009).

4. Gender, incarceration, and health outcomes

Although there has been some research on incarceration and health, there has been little discussion of gender differences, and none that focuses on gendered differences in mortality. This omission could reflect the general neglect of gender in criminology (see Chesney-Lind and Irwin, 2007), but it seems particularly problematic with respect to the area of health, both from the standpoint of selection and causation. For one, there are gender differences in the forces of selection into prison, and many of these differences are relevant to health and mortality. As a percent of inmates, for example, more female inmates than male inmates are drug users (Greenfeld and Snell, 1999). To the extent that drug use drives the incarceration-mortality association, the association between incarceration and mortality may be larger for women than for men but also more likely to be eliminated by simple controls for prior drug use and other behaviors associated both with crime and mortality. In addition, some of the basic demographic characteristics of incarceration differ between the sexes. These have mixed implications for health. On the one hand, female prisoners are somewhat better-educated than male prisoners and more likely to have grown up in a two-parent household, which suggests a more advantaged and health-promoting upbringing (Snell, 1994). On the other hand, female inmates often suffer from a history of victimization, which is related to

women's patterns of offending (Browne, 1987; Snell, 1994). Many female offenders, for example, turned to drug use following physical and sexual abuse, suggesting the criminal careers of women reflect ongoing disadvantages more than they might for men (Browne, 1987; Chesney-Lind, 1997). Such differences have implications for health outcomes, including mortality.

All these factors are also related to selection into prison, but assuming that incarceration has at least some effect on mortality among women (and men), there are still several reasons to expect gender differences in the effects of incarceration. For one, the conditions of confinement may differ between the sexes. On the one hand, the prison environment may be better for women. Male prisoners tend to suffer more injuries behind bars than female prisoners (Maruschak and Beck, 2001; Pare and Logan, 2011) and female prisoners tend to form more supportive relationships in prison (see Kruttschnitt and Gartner, 2005). On the other hand, many other aspects of incarceration are more difficult for women. For instance, a larger fraction of female prisoners report developing health problems while incarcerated (Maruschak and Beck, 2001) and female inmates also report higher levels of mental distress (Lindquist and Lindquist, 1997). These differences point to the importance of a specific inquiry into gendered differences in the impact of incarceration on later mortality.

Moreover, the quality of services available to women in prison may be worse than that available to men, resulting in more women leaving prison with uncontrolled chronic conditions (see for instance, Belknap, 1996). Female prisoners are generally offered fewer educational and vocational programs (Rafter, 1990), in part because female offenders may be seen as less open to rehabilitation than male offenders, and therefore less likely to benefit from such programs (Kruttschnitt and Gartner, 2005, Chapter 5). Finally, the geography of incarceration may offset some of the support female inmates are able to secure. Female inmates are more likely to be sent to prisons far removed from family, children, friends, and legal representation (Braithwait and Arriola, 2003), which partly explains higher levels of abuse by correctional officials in women's facilities (Human Rights Watch, 1996: Ford V. County of Oakland, 6th Cir., 2002).

While these processes suggest differences in the prison experience itself may produce gender differences in health outcomes, the relevance of gender does not end upon release. Female inmates may face greater cultural obstacles to reintegration than men, especially given the inconsistency between traditional notions of femininity and beliefs about criminality. The violent, willful, and predatory image of the typical prisoner clashes with stereotypical notions of femininity (see for instance, Schur, 1984). Among men, however, the image of the prisoner may reinforce traditional notions of masculinity, including toughness or power (Anderson, 2000; see also Katz, 1988). This tension has not abated over time, even as beliefs about gender have progressed. Indeed, the demonization of female offenders has been especially severe in recent years (Chesney-Lind and Irwin, 2007), with female offenders periodically blamed for the disintegration of the family in urban areas (Kruttschnitt and Gartner, 2005, esp. 38 and 119). Given gender differences in the stigma of incarceration, female inmates may face unique challenges with reintegration (see Riche (2001) and Morash et al. (1998)). To the extent that women are held accountable for maintaining the family, for example, a prison sentence may result in more stigma for wives

and mothers than for husbands and fathers (Chesney-Lind and Irwin, 2007). In addition, many female offenders return to the abusive relationships that initiated their criminal careers, further undermining their ability to reintegrate (Morash, 2010).

In sum, prior literature points to a number of reasons to suspect differences by gender in the incarceration-mortality relationship. First, there are meaningful differences in the lives of men and women before prison. Second, men and women often face different institutional challenges while they are incarcerated, ranging from correctional services to maintaining contact with family. Finally, the post release experience is often vastly different for men and women. Prior research has linked these factors to health and mortality, so a focused investigation on gender differences in the mortality-incarceration relationship is particularly timely and relevant.

5. The current study

This study focuses on (i) the relationship between incarceration and premature mortality (between the ages of 24 and 47), (ii) whether there are gender differences in the incarceration-mortality relationship, and (iii) whether there are gender differences in the impact of some of the factors related to both incarceration and health, including educational attainment and family background variables. Understanding the incarceration-mortality relationship necessitates longitudinal data, as well as an assortment of variables related to incarceration and mortality. The National Longitudinal Survey of Youth (NLSY) is uniquely suited for these purposes.

6. Data and methods

We are not the first to use the NLSY to study incarceration (Huebner, 2005; Massoglia, 2008a; Schnittker and John, 2007; Western, 2002). Because of its high quality and longitudinal nature, the NLSY has been used to examine the effects of incarceration on health, marriage, wages, and other outcomes (Lopoo and Western, 2005; Massoglia, 2008b; Western, 2002), but it has not, to our knowledge, been used to explore gender differences in mortality. Data collection began in 1979 and is ongoing. The original NLSY sample included 12,686 individuals and utilized a multi-stage stratified probability sample of U.S. dwellings. Respondents were interviewed annually in the early years of the survey and biennially starting in 1994. Given the design of the study, we observe all respondents at the age of 24 and follow them until death or censoring at age 47. During the period of observation, 349 individuals died.

The main independent variable is, of course, incarceration. Incarceration is measured indirectly, as the place of residence during the interview. Because the NLSY sought to collect data in each wave from all respondents in the initial sample, a special effort was made to interview respondents on schedule and regardless of their place of residence. Although it is possible to approximate the length of a prison sentence using information on the number of consecutive interviews in prison/jail, previous research indicates that exposure to the prison system is more consequential than the length of exposure, a result we also find here (Schnittker and John, 2007; Massoglia, 2008a).¹ Incarceration is coded 1 if respondents

were interviewed in prison or jail for any panel of observation and 0 otherwise (see Harper and McLanahan, 2004). Although the NLSY occasionally overlooks detainments for short periods of time, including brief jail spells between interviews, a contaminated reference category (those coded as having no period of incarceration were actually incarcerated for some amount of time) biases our estimates downward and, as we show shortly, our estimates are still quite strong, especially for women (for a more complete discussion see Western, 2002).

The models include numerous control variables, starting with the demographic characteristics of the respondent (see Cutler, Deaton, and Lleras-Muney, 2006 for a review of the determinants of mortality). Apart from gender, the models include age and race (coded as black, other race, and white, which serves as the reference category). Respondent's education is time varying to allow for individuals advancing through the educational system as the survey progresses (even though, of course, most educational attainment is complete by the time respondents are in their mid-twenties). The variable is coded as either high school degree or no degree, with those having at least a high school diploma serving as the reference category.

We also account for a series of life course and health-related processes, allowing us to explore influences anterior to incarceration and health. Parental level of education indicates whether either of the respondent's parents earned a BA degree or higher. Welfare indicates whether the family of origin received welfare assistance at any point when the respondent was growing up. Given the importance of socioeconomic background, we included an additional measure beyond the receipt of welfare. Poverty status is a dichotomous variable coded 1 if respondents reported living at or below 125% of the national poverty level at any point in previous panels and 0 otherwise. We include time-varying covariates for marital status and employment status.

In addition to these life course processes, we also include an indicator of locus of control, based on agreement with four items from an established scale (e.g., "what happens to me is my own doing" and "sometimes I feel that I don't have enough control over the direction my life is taking") (Rotter, 1966). A score of 0 indicates the belief that one does not control his/her life, while a score of 4 indicates the belief that one has complete control (see Zerega Tseng and Greever 1976). Locus of control has been associated with health and may also play a role in offending (Bandura, 1997; Gallo and Matthews, 2003; Pearlin et al., 1981; Taylor and Seeman, 1999). Finally, the models include controls for health insurance and self-reported poor health prior to incarceration. Health insurance is a time varying dichotomous variable coded 1 if respondents reported being covered by health insurance at the time of the interview and 0 otherwise. Assessing health problems prior to incarceration was based on reports of whether the respondents have a health limitation that limits the type and amount of work they can do, coded 1 if they report such a limitation prior to also reporting incarceration.

¹We also estimate the models with length of incarceration as a continuous variable, and the results were substantively identical. Moreover, the relationship between incarceration and later mortality did not change significantly with year over year changes in incarceration length, suggesting exposure, rather than length of exposure, is driving the incarceration mortality relationship.

In addition to these background variables, the models account for more direct health risks. For instance, the models account for serious drug use with a dichotomous variable coded 1 if respondents reported using illegal drugs in any prior panel, including intravenous drugs, and 0 otherwise. The variable is time varying, but logically the variable never reverts to zero following a positive score. Using dummy variables, we also account for those who report current cigarette use and those who do not. Along similar lines we also account for violent behavior. We model violent behavior with a series of dummy variables based on whether respondents reported attacking someone in the last year. We retained information on the degree of violence: “sporadic violence” denotes those who report such acts four times or less and “frequent violence” denotes five acts or more, with no violence serving as the reference category.

7. Methods

The NLSY provides information about the year when a respondent died, but does not specify the month or day. Given this level of detail, discrete-time event history analysis is the preferred method of analysis (Allison, 1984).² Discrete-time event history models can be interpreted in terms of a logit regression model fit to person-panel observations, wherein the log-odds of dying are estimated at each time point for each individual based on the covariates included in the model (described below). Formally, the model can be expressed as:

$$\text{Log}[P(t)/(1 - P(t))] = \alpha(t) + b_1x_1\text{incarceration}(t) + b_2x_2(t) + \epsilon$$

where the left side of the equation is the log of the odds of death at time t , given survival to $t - 1$. The hazard function, then, is the conditional odds that an individual will die in each time period (t), given they did not die at an earlier time period (Allison, 1984). This hazard function is expressed through a constant ($\alpha(t)$), which represents the baseline logit hazard functions for each time period, while b_1x_1 represents the impact of incarceration, b_2x_2 represents the impact of a vector of relevant covariates and control variables.

There were 349 deaths observed in the study, as death by age 47 is uncommon. For this reason, we estimated discrete-time event history models suitable for rare events (see discussions in King and Zeng 2001a, 2001b; Tomz et al., 2003). As discussed by King et al. (2001), traditional event-history models fit to data with rare events can underestimate the probability of such events and introduce considerable downward bias to the coefficients (King and Zeng, 2001a). The rare event model corrects for this problem and generate bias-corrected coefficients (see King and Zeng, 2001a, 147 for a technical discussion).

Event-history models are not the only way to address the effects of incarceration. As a robustness check, we re-analyze the data using propensity score models (Morgan, 2001; Rosenbaum and Rubin, 1983), another popular approach among those interested in the effects of incarceration (Massoglia, 2008a; Mears and Bales, 2009). Propensity score models

²Respondents enter the NLSY at various ages between 15 and 24. However, we array the data to begin tracking all individuals at age 24, thereby avoiding potential estimation biases due to right truncation. (Allison, 1984).

are common in incarceration research because they closely approximate a counterfactual condition that is hard to realize given the topic. Propensity score models match subjects in a sample based on their likelihood of incarceration (the propensity score), and then examine differences in an outcome (in this case, mortality) between those who experienced incarceration and those who did not. This difference between the two groups of subjects represents an effect of incarceration insofar as the treatments and controls are appropriately matched and differ only with respect to the treatment. The propensity score represents the conditional probability of incarceration and can be written as follows (Rosenbaum and Rubin 1983):

$$(2)p(\text{incarceration}) = \Pr(T_i = 1 | \mathbf{X}_i)$$

where T_i is 1 if individual i has been incarcerated and zero otherwise and \mathbf{X}_i is a vector of covariates that predict incarceration. As in the case of event-history models, we use a set of covariates related to gender, crime, and health, including demographic variables, prior health status, criminal behavior, and drug use and a series of interactions to create gender specific propensity scores. Every individual in the sample is assigned a propensity score regardless of whether they are in the treatment (ever incarcerated) or control (never incarcerated) group.

The analytic sample is constructed by matching treated individuals to control individuals. This is done using a propensity score, but different procedures can be used to assign a match between similar scores. As these have different strengths and weaknesses, our analysis uses two complementary procedures: radius matching and kernel matching. Kernel matching weights the contribution of a comparison based on its quality: the best matches between treatment and control contribute the most to estimating the treatment effect. In this way, kernel matching accounts for potential outliers or cases when treated individuals are not perfectly matched to non-treated individuals. Radius matching is different. Radius matching assigns a radius – in our case 0.001 – around the propensity score of each treated individual. Controls within that radius are used to calculate the treatment effect, while controls outside that radius are not.

Sample attrition is minimal in the NLSY. Between 1983 and 1998, 14 interviews were scheduled for each respondent and respondents only missed, on average, 2 interviews (see Macurduy et al., 1998). According to Western (2002), the small amount of panel-wise attrition does not cause systematic sampling biases in studies concerned with incarceration. Nevertheless, we address attrition in multiple ways. For one, we use discrete time event history models, which use all available information for each respondent and, thus, do not exclude respondents who occasionally miss a panel (Allison, 1984). Second, the results were replicated using survey weights that take attrition into account. Although we present unweighted results, weighted results were substantively identical. In addition, attrition does not appear to be related to our variables of interest: in Appendix A we present models in which attrition is treated as the dependent variable.

The longitudinal nature of the data is relevant in another way. As discussed earlier, recent work has been sensitive to the longer term impact of incarceration on mortality (Dirkzwager et al., 2012) and preliminary analysis of our data shows the importance of accounting for an extended period of time after release. Our data show the time period immediately after release is the most frequent period when respondents die, but there is considerable variability in time to death after release. For instance, among those ex-inmates who die during the survey period, female ex-inmates lived and average of just under 4 years after release and men on average lived almost 5 years post release. Moreover, these averages are not just a function of a few outliers, as approximately 70% lived more than 2 years after release. Such descriptive data demonstrate the importance of the period immediately post release, but also reveal that incarceration may have far reaching effects on mortality.

Given our interest in discerning the influence of selection, we estimate several progressively more stringent models. We also estimate all the models separately by gender. Presented in this fashion, the models can be interpreted along three dimensions: (i) whether the effects of incarceration on mortality are sensitive to controls, (ii) whether there are gender differences in the effects of incarceration, and (iii) whether there are gender differences in the most important variables for understanding the effects of incarceration.

8. Results

Descriptive statistics are presented in Table 1, stratified by gender. A number of gender differences are important to highlight. Gender differences in morbidity and mortality follow the familiar paradox. Men are at greater risk for premature mortality: there were 239 deaths among men and 110 among women. Women are, however, at greater risk for poor health: 7.2% of men report poor health compared to 9.9% of women. Men are slightly less likely to have health insurance: approximately 67% of men have insurance compared to 74% of women. Not surprisingly, men are more likely to be violent and experience incarceration. A number of variables, however, show no meaningful gender differences, including parental education levels, poverty status, and locus of control.

9. The relationship between incarceration and premature mortality

Table 2 presents four models predicting the odds of premature mortality. Model 1 suggests a significant relationship between incarceration and premature mortality for both men and women (the parameter estimates are 0.354 and 1.702 respectively). After exponentiating the coefficients, the odds of dying are at least 41% higher for men who have been incarcerated relative to men who have not been incarcerated. The same odds among women are more than 5 times higher ($\exp 1.702 = 5.48$). Although the first model is merely descriptive and does not control for confounding factors, the results suggest a powerful relationship between incarceration and premature mortality, especially among women.

Model 2 introduces a series of background variables. Introducing controls for age, race, and family background (parental education level and whether the respondent's family of origin received public assistance or welfare) eliminates the relationship between incarceration and mortality for men. In contrast, the relationship between incarceration and mortality among

women remains significant and declines only marginally (the parameter estimate is 1.621; $p < 0.01$).

Model 3 considers the impact of a number of individual-level attributes, including education and a variety of behaviors. In contrast to the factors considered in Model 2, the factors considered in Model 3 are generally more predictive of female mortality. Education, violence, drug use, and cigarette use are all predictive of premature mortality among women, in marked contrast to family background. Yet despite the introduction of these measures, the key relationship observed in Model 2 remains: Model 3 shows incarceration is a significant predictor of premature mortality for women but not for men.

Model 4 introduces two additional blocks of variables. The first block includes three life course factors often associated with health and mortality: marital status, poverty status and employment status. The second block includes locus of control, health problems prior to incarceration and whether the respondent has health insurance. Not surprisingly, many of these variables – marriage and employment status, whether the respondent had insurance and health problems prior to incarceration – are associated with mortality. Model 4 provides the most stringent test of the incarceration–mortality relationship, but reveals patterns consistent with the earlier models. Even in the presence of many control variables significantly related to mortality, a robust relationship remains between incarceration and premature mortality for women, but not for men. Formerly incarcerated women have 2.5 times (exp. 0.907; $p < .05$) higher odds of early mortality than women without a history of incarceration. While the magnitude of this coefficient is reduced between Model 1 and Model 4, women who were incarcerated consistently have higher risks of premature mortality across all models. Of particular note, we find this relationship even after accounting for a range of factors, including family background factors (such as parental education level), behavioral factors (such as drug use), and life course factors (such as marriage and employment). In an even more direct test of a spurious relationship, the relationship remains significant even after accounting for prior health problems. In Model 4, we also formally tested the difference in mortality risk across genders, and find that female ex-inmates are at a significantly higher mortality risk than male ($p < .01$).

10. Propensity score models

Propensity score models offer supplementary evidence that speaks to our main finding but using a different set of assumptions. As discussed earlier, propensity score models use observed covariates to create two groups that are statistically homogeneous except for incarceration. Assuming a properly specified matching model, any difference in mortality between the two groups can be attributed to incarceration. The results from the propensity score models, presented in Table 3, are similar to the event-history models. Incarceration is not related to premature mortality among men but is related to premature mortality among women. Consistent with the results from the event history models, the magnitude of the coefficients indicates that the effect of incarceration on mortality is at least five times stronger for women (estimates .133 and .136) than men (estimates $-.006$ and $.025$). As a further test of the conclusions drawn from the propensity score models, we assessed the robustness of our estimates through a series of simulations. This allows us to empirically

assess how strong the impact of unobserved variables would have to be in order to render the effect of incarceration on mortality for women non-significant (DiPrete and Gangl 2004). In this case, the bias from unobservables would have to exceed 50% for our estimates to not be supported by the data.³

11. Discussion

This study assessed whether men and women who experienced incarceration are at greater risk of premature mortality. The analyses reveal strong gender-specific patterns. Women who experienced incarceration are more likely to die prematurely than their non-incarcerated counterparts, even after controlling for socio-demographic factors and a variety of covariates that are themselves significantly related to mortality. Men who experienced incarceration are also more likely to die prematurely than non-incarcerated men, but this relationship, unlike the relationship found among women, is explained entirely by socio-demographic factors, especially education and race. This result is robust to the analytic approach we use and the other covariates in the models fit well-established epidemiological patterns, lending further credibility to the results.

We view this analysis as an initial step in understanding gender differences in the incarceration-mortality relationship and have several suggestions for future research. The breadth of information available in the NLSY allows us to address selection and other mortality risks to some degree, but it nevertheless remains difficult to specify the mechanisms linking incarceration to mortality or to explain the gender difference. There are a number of possibilities, but we start with the most general. The incarceration-health relationship may be stronger for women because incarceration entails more stigma than it does for men. Evaluating this possibility involves assessing both the relationship between incarceration and stigma and the relationship between stigma and mortality. Regarding the former, the role of stigma is usually inferred in light of evidence linking contact with the criminal justice system with a variety of negative outcomes (see Western, 2006; Pager, 2005; Schnittker and John, 2007), but there is little direct evidence bearing on stigma directly, especially for female inmates, or even on how the public views female offenders. It is possible that the stigma of incarceration is higher among female than male offenders, given its relative prevalence, but explicit measures of stigma, including stigma consciousness (Pinel, 1999), would help to illuminate the role of stigma in a more precise fashion. Such measures could also empirically link stigma to mortality. Stigma is a compelling explanation in part because it likely operates through many of the mechanisms described earlier, including reduced socioeconomic resources, as when a former inmate is not hired because of the mark of her criminal record, or because of the stress of marginal status (see Link and Phelan, 2001).

Stigma is not the only potential explanation. According to Morash (2010), women on probation or parole face different challenges than men in the same situation. Among the most salient are higher rates of drug addiction and ongoing relationships with abusive partners. These possibilities can be at least indirectly addressed by exploring cause of death.

³See DiPrete and Gangl (2004) for full discussion.

Knowing whether incarcerated women are more likely than men to die of, for instance, homicide, drug overdose, suicide, or infectious disease would provide clues regarding the specific mechanisms involved. Along similar lines, measures of sexual behavior would be informative. Some female inmates have participated in sex work (Kassira et al., 2001) and many return to such work following release. As a general statement, inmates have comparatively high levels of sexually transmitted disease, which could play a role in explaining the gender difference. It is doubtful, however, that our gender-specific effects are being driven by HIV alone. For instance, in state and federal prisons, rates of HIV have declined substantially. In recent years, less than 2.5% of women and 2% of men in prison are known to be HIV positive (Maruschak, 2008). More detail information on drug use would be informative as well. We control for general drug use, but more detailed drug use indicators might shed more light on gender differences. It may be, for instance, that a unique pattern of drug use among female ex-inmates, related to issues around the timing and frequency of use, rather than simply whether they use drugs at all, contributes to the higher mortality of female ex-inmates (Morash, 2010). In general, the gender differences likely reflects a number of influences rather than one particular thing. In line with Morash (2010), we think it reflects the combination of challenges that fall on female offenders, including stigma, abusive partners, drug use, mental illness and infectious disease.

In thinking about gender-specific challenges, it is also worth considering the multiple sorts of effects incarceration can have. Even if one believes incarceration exerts most of its impact through infectious disease or drugs, the process leading to these mechanisms likely reflects more than exposure to infections or increased use of drugs. Prior literature has shown that incarceration, for example, lowers drug resistance, which contributes to the likelihood of drug overdose after release. At the same time, incarceration lowers adherence to HIV drug therapy. In this way, diminished access to treatment following release increases the likelihood of complications (Milloy et al., 2011; Small et al., 2009). Overall, we emphasize the importance of social, behavioral, and medical processes when understanding how incarceration impacts the health of released felons. Furthermore, we emphasize the importance of thinking about how selection and causation work in tandem: although some women may use drugs or have infections prior to incarceration, the effects of incarceration might be to increase the risk of mortality associated with those conditions.

It is important to note that the NLSY has at least three limitations that are relevant to our conclusions. First, as a function of its design, the NLSY does not contain older individuals. Given the recency of the prison boom, older persons are less likely to have served time, but they are, of course, at greater risk of mortality. Some of the effects of incarceration might emerge only in later life, especially if incarceration is related to mortality through chronic stress, whose effects will be realized only years later. This possibility has not been explored extensively, but there is some evidence bearing on the age-specific effects of incarceration. Using a more age-representative sample, Binswanger and colleagues find the relative risk of mortality is *lowest* among inmates 45 and older and highest among those 25–44 (Binswanger et al., 2007). The NLSY may capture those age groups most at risk, but it is unclear whether the same age-specificity applies to both men and women.

Second, while approximately 350 individuals died prematurely in the NLSY, the NLSY contains relatively few deaths. The sample is not unusual in this regard: the age-specific rates of mortality found in the NLSY are similar to those reported elsewhere (Binswanger et al., 2007). Furthermore, we used models developed for rare-events and the number of deaths was, in any case, sufficiently large to identify significant effects among the group that experiences the fewest deaths. A larger and/or older sample would not necessarily alter this basic result, but it would allow for the consideration of the more complex pathways, processes, and interactions.

Third and finally, along with a larger and older sample, it is important to consider the measures used in our analysis. While the NLSY has many desirable features and has been widely used to study the effects of incarceration, it was not expressly designed to study the health effects of incarceration. A more focused research design, perhaps one with data focusing on causes of death and specific drug usage would help us move toward understanding the precise mechanisms linking incarceration to later mortality. Along similar lines, careful studies linking administrative data with other data sources has the potential to be particularly informative (Kinner et al., 2013).

12. Conclusion

The growth of incarceration is a relatively new phenomenon but it will be an important consideration for years to come. As the correctional system assumes an ever larger place in American society and affects a wider range of people, the consequences of the prison boom will likely compound. Our study points to a number of areas for future research, but it specifically encourages further consideration of gender differences in the health consequences of incarceration. To date, most of the evidence on this topic has neglected gender entirely or considered gender mostly in terms of its effects on families and children. Women themselves have been neglected. Incarceration undoubtedly has numerous collateral consequences, but it also affects the health of female offenders directly. More men might suffer behind bars than women, but some of the effects of incarceration on health might be stronger among women.

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Appendix A. Discrete event history predicting attrition for the full sample - probit coefficients

	Male	Female
Incarceration	0.423***	0.565***
	(.03)	(.07)
Age	-0.259***	-0.265***
	(.00)	(.00)
Race (Black = 1)	0.343***	0.206***

	Male	Female
	(.02)	(.02)
Two parents	0.452 ***	0.376 ***
	(.02)	(.02)
Parents' education	-0.090 **	-0.034
	(.03)	(.03)
Gov. assistance	-0.012	0.072 ***
	(.02)	(.02)
College degree	0.583 ***	0.437 ***
	(.03)	(.03)
High S. degree	0.648 ***	0.562 ***
	(.02)	(.02)
Poverty status	0.203 ***	0.260 ***
	(.02)	(.02)
Drug use	0.142 ***	0.031
	(.02)	(.02)
Violence (sporadic)	-0.109 ***	-0.100 ***
	(.02)	(.02)
Violence (often)	-0.179 ***	-0.113 ***
	(.02)	(.03)
Locus of control	0.013	0.031 ***
	(.01)	(.01)
Health Problems	0.047	0.164 ***
	(.03)	(.02)
Health Insurance	1.391 ***	1.598 ***
	(.02)	(.02)
Constant	7.271 ***	7.417 ***
	(.05)	(.05)
Observations	142,304	140,224

* *Note:* $p < .05$.

Responses of 1 indicate respondents remained in the study for any given survey year and responses of 0 indicate that respondents fell out of the sample (attrition) in any given year.

** $p < .01$.

*** $p < .001$.

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Table 1

Descriptive statistics: person year data file.

	Male	Female
Premature mortality	0.004	0.002
Age	31.2	31.3
Race (Black = 1)	0.282	0.275
Other race	0.062	0.061
Parents education	0.153	0.152
Welfare	0.129	0.158
High S. degree	0.589	0.620
No degree	0.253	0.218
Poverty status	0.296	0.307
Early delinquency	0.231	0.092
Drug use	0.294	0.229
Violence (sporadic)	0.247	0.152
Violence (often)	0.169	0.047
Violence (never reference)	0.584	0.801
Locus of control	2.561	2.501
Poverty	0.079	0.0471
Married	0.679	0.709
Employed	0.923	0.889
Health problems	0.072	0.099
Health Insurance (1 = yes)	0.669	0.740
Incarceration	0.099	0.012
Observations	68,276	70,869
Respondents	6225	6134

Notes: For time stable variables (e.g., race) the numbers in Table 1 represent the proportion of sample that are in a given state or category.

Notes: For time varying variables (incarceration) the numbers in Table 1 represent the wave specific probability a respondent enters a given state or category.

Table 2

Coefficients of a discrete event history analysis for rare events: the relationship between incarceration and premature mortality.

	<u>Model 1</u>		<u>Model 2</u>		<u>Model 3</u>		<u>Model 4</u>	
	Male	Female	Male	Female	Male	Female	Male	Female
Incarceration	0.354 [*] (.18)	1.702 ^{***} (.39)	-0.004 (.20)	1.621 ^{***} (.39)	-0.126 (.21)	1.281 ^{***} (.41)	-0.296 [*] (.21)	0.907 ^{**} (.41)
Age			0.048 (.03)	-0.069 (.04)	-0.083 ^{***} (.03)	-0.095 [*] (.04)	-0.138 ^{***} (.03)	-0.126 ^{***} (.05)
Black			0.619 ^{***} (.14)	0.399 [*] (0.20)	0.723 ^{***} (.15)	0.546 [*] (.21)	0.777 ^{***} (.15)	0.541 [*] (.22)
Other Race			-0.043 (.15)	0.313 (.36)	-0.125 (.30)	0.265 (.37)	-0.216 (.31)	0.177 (.37)
Parents Edu.			-0.506 [*] (.24)	-0.367 (.32)	-0.546 [*] (.24)	-0.434 (.33)	-0.473 [*] (.24)	-0.271 (.32)
Welfare			0.475 [*] (.16)	-0.193 (.26)	0.449 [*] (.16)	-0.269 (.26)	0.413 ^{**} (.17)	-0.404 (.26)
High S. Degree					0.413 ^{***} (.16)	-0.302 (.22)	0.361 ^{***} (.16)	0.212 (.22)
No Degree					1.45 ^{***} (.17)	1.11 ^{***} (.26)	0.567 [*] (.21)	0.414 (.31)
Violence (sporadic)					-0.062 (.19)	-0.089 (0.26)	-0.056 (.16)	-0.131 (.26)
Violence (often)					-0.037 (.24)	0.441 [*] (.07)	-0.141 (.19)	0.292 [*] (.12)
Drug use					0.106 (.15)	0.421 [*] (.20)	0.221 (.15)	0.451 [*] (.22)
Cigarette Use					0.381 [*] (.16)	0.401 [*] (.19)	0.171 (.16)	0.362 (.23)
Locus of Control							0.013 (.06)	-0.081 (.49)
Married							-1.484 ^{***} (.18)	-1.511 ^{***} (.28)
Poverty							-0.191 (.16)	.403 (.25)
Job							-1.615 ^{***} (.39)	-0.884 ^{**} (.34)
Health Problems							0.483 (.41)	0.841 ^{***} (.36)
Health H. Insurance (1 = yes)							-1.535 ^{***} (.15)	-1.187 ^{***} (.21)
Constant	-6.20	-7.109	-4.717	-5.556	-5.044	-5.702	-2.068	-4.258

	Model 1		Model 2		Model 3		Model 4	
	Male	Female	Male	Female	Male	Female	Male	Female
	(.12)	(.19)	(.56)	(.55)	(.57)	(.84)	(.15)	(.64)
Pseudo <i>R</i> -Square	0.0278	0.0513	0.0429	0.0561	0.0702	0.0989	0.1336	0.1499
Observations	68,276	70,869	68,276	70,869	68,276	70,869	68,276	70,869

Note: Standard errors in parenthesis.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

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Table 3

Propensity score estimators with multiple matching procedures: the treatment effect of incarceration on mortality by gender.

Matching estimator	Incarceration effect	Normal theory 95% bounds		Empirical distribution 95% bounds	
		Lower	Upper	Lower	Upper
Kernel (men)	-0.006	-.037	.024	-.038	.023
Kernel (women)	0.133*	.038	.228	.054	.252
Radius (men)	0.025	-.002	.051	-.003	.054
Radius (women)	0.136*	.041	.229	.045	.231

All analyses restricted to regions of common support.

* $p < 0.05$.