

The Mortality Penalty of Incarceration: Evidence from a Population-based Case-control Study of Working-age Males

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Abstract

There is a growing body of research on the effects of incarceration on health, though there are few studies in the sociological literature of the association between incarceration and premature mortality. This study examined the risk of male premature mortality associated with incarceration. Data came from the Izhevsk (Russia) Family Study, a large-scale population-based case-control design. Cases ($n = 1,750$) were male deaths aged 25 to 54 in Izhevsk between October 2003 and October 2005. Controls ($n = 1,750$) were selected at random from a city population register. The key independent variable was lifetime prevalence of incarceration. I used logistic regression to estimate mortality odds ratios, controlling for age, hazardous drinking, smoking status, marital status, and education. Seventeen percent of cases and 5 percent of controls had been incarcerated. Men who had been incarcerated were more than twice as likely as those who had not to experience premature mortality (odds ratio = 2.2, 95 percent confidence interval: 1.6–3.0). Relative to cases with no prior incarceration, cases who had been incarcerated were more likely to die from infectious diseases, respiratory diseases, non-alcohol-related accidental poisonings, and homicide. Taken together with other recent research, these results from a rigorous case-control design reveal not only that incarceration has durable effects on illness, but that its consequences extend to a greater risk of early death. I draw on the sociology of health literature on exposure, stress, and social integration to speculate about the reasons for this mortality penalty of incarceration.

Keywords

case-control study, health, incarceration, premature mortality, Russia

Incarceration rates in the geopolitical powers of the United States and Russia are among the highest in the world.¹ The rapid expansion of the U.S. penal system began in earnest in the 1970s, while large-scale imprisonment in Russia dates back further. Although their reasons for resorting to mass incarceration vary, one critical outcome is that both nations imprison a large number of their citizens—730 and 519 per 100,000 residents in the United States and Russia, respectively (International Centre for Prison Studies 2012)—without efficient concomitant structures to reintegrate inmates back into society or to mitigate the impact of incarceration on other negative outcomes. This has led

to substantial research in the United States on the collateral consequences of mass imprisonment for individuals, communities, and society. Studies have examined imprisonment as a barrier to future marriage and employment (Huebner 2005; Pager 2003), its negative effect on already disadvantaged communities (Clear 2007), its role in ethnic and class

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inequality (Pettit and Western 2004; Western 2002), and its influence on the outcomes of senatorial and presidential elections (Uggen and Manza 2002).

Medical sociology largely ignored the potential health effects of imprisonment until recently, though a growing literature has begun to address the topic (Patterson 2010; Wildeman 2012), including studies published in this journal (Massoglia 2008a; Schnittker and John 2007; Schnittker, Massoglia, and Uggen 2012; Turney, Wildeman, and Schnittker 2012). Incarceration may negatively influence health via multiple mechanisms. First, incarceration increases exposure to and the likelihood of contracting infectious diseases like HIV, hepatitis C, and tuberculosis (Johnson and Raphael 2009; Massoglia 2008a; Stuckler et al. 2008). Second, major life events (Thoits 1995) and primary and secondary stressors (Pearlin 1989) can create enduring negative health outcomes. Imprisonment certainly constitutes such an event, as it exposes one to the initial shock of incarceration, the acute stress of the prison environment, and the chronic stress associated with marginalization in multiple domains of life following release. Medical research shows that stress sustained over a long period can have detrimental physiological effects, compromise the immune system, and lead to long-term health problems. Third, incarceration disrupts social ties to spouses and children and to employment and education opportunities that might otherwise provide protective effects against negative health outcomes. Recent studies demonstrate the short- and long-term health effects of incarceration resulting from exposure, stress, and stigma (Fazel and Baillargeon 2011; Massoglia 2008a, 2008b; Schnittker and John 2007).

In spite of the increasing interest in the health effects of imprisonment, the individual-level impact of incarceration on the most serious health outcome, mortality, remains absent from the sociological literature (see Wildeman 2012 for a recent population-level study). While there is detailed information on the distribution and causes of death while incarcerated (Mumola 2007) and on the impact of incarceration on mortality while imprisoned (Patterson 2010), only a few studies address the enduring effects of incarceration on premature mortality upon release from prison (Binswanger et al. 2007; Rosen, Schoenbach, and Wohl 2008; Spaulding et al. 2011). However, these studies usually (1) control only for age, sex, race, and perhaps a simple education variable dichotomized on receipt of a high school diploma and (2) address only briefly the theoretical mechanisms that may

link incarceration to early death. In this study, I used a case-control design to determine the risk of premature mortality among working-age males associated with incarceration, controlling for the known determinants of premature mortality in this sample, including detailed measures of hazardous drinking, smoking status, marital status, and education. Results suggest a substantial mortality penalty of incarceration, and I draw on the literatures on exposure, stress, and social integration to explain the association.

BACKGROUND

There are several reasons incarceration may be associated with negative health outcomes, including premature mortality. Incarceration increases exposure to infectious diseases and subjects one to the prolonged stress of the prison environment. The stress of prison life dissipates upon release, but in the struggle to reintegrate into society the felon and former inmate is faced with a series of new challenges that create their own stress. In addition, both during and following incarceration, one's ties to important protective social bonds and networks like family and employment are threatened or cut off. Each of these factors—exposure to disease, stress, and disruption of social bonds—is associated with negative health outcomes and premature mortality.²

Incarceration and Exposure to Infectious Diseases

Prisons house populations with generally poor health and a high rate of infectious disease. The most common life-threatening infectious diseases in correctional facilities are tuberculosis (TB), hepatitis, and HIV. Sexually transmitted infections and methicillin-resistant staphylococcus aureus are also common (Bureau of Justice Statistics 2008; National Commission on Correctional Health Care 2002). About 10 percent of male inmates in U.S. state prisons have confirmed tuberculosis, about 5 percent have confirmed hepatitis, and about 1.5 percent of inmates in state and federal prisons are HIV-positive (Bureau of Justice Statistics 2008, 2009). The prevalence of each is underenumerated, however, with true infection rates likely much higher. Inmates are not only disproportionately exposed to infectious diseases, but exposure occurs in an environment where group quarters and prison culture create efficient conditions for disease transmission, including overcrowding, poor health care,

poor nutrition, and a host of risky behaviors like sharing hygienic facilities and personal hygiene items, amateur tattooing and piercing, practicing unprotected sex, and using unsterilized drug injection equipment.

It is impossible to know for sure what proportion of ex-inmates with an infectious disease contracted their illness while in prison. There is little doubt, however, that the prison is an especially important setting for TB: increasing incarceration rates in several nations are associated not only with a greater number of TB infections among inmates and ex-inmates but also with growing TB infection rates among their general populations (Stuckler et al. 2008). Similarly, U.S. prisons are seen as a primary engine driving the non-institutionalized population's exposure to hepatitis C as infected inmates are released. In a study of imprisonment and infectious disease, Massoglia (2008a) found that those previously incarcerated were about four times more likely than those not previously incarcerated to self-report urinary tract infections, hepatitis, and tuberculosis. Johnson and Raphael (2009) found that changes in black incarceration rates between 1982 and 1996 were nearly entirely responsible for the black-white disparity in AIDS infection rates during this period.

Incarceration, Stress, and Health in Prison and Following Release

Massoglia (2008a) draws on the medical sociology literature to explain how incarceration can create negative health outcomes. He argues that the shock of incarceration as a major life event, the stressful immediate conditions of imprisonment, and the long-term extralegal consequences of incarceration following release lead to a greater prevalence of stress-related illnesses among former inmates.

Incarceration is both an acute and an enduring stressor. The initial shock of imprisonment is a traumatic event on par with marital separation and the death of a close family member (Holmes and Rahe 1967). Major life events that precipitate dramatic change in a short time period can create substantial stress and lead to detrimental health effects (Thoits 1995; Wheaton 1994). However, as Pearlin (1989) notes, it is important not to confuse what may seem like discrete events with more enduring stressors, of which he cites incarceration as one example. One element of incarceration's enduring nature is that it is not an isolated event but can last for years, thereby presenting chronic stress on top of the initial shock. The proximate sources of stress

in prison are manifold and constant; they include lack of privacy, overcrowded conditions, antagonistic relationships with guards and fellow inmates, witnessing violence, and the threat of violent victimization.

Another element of the enduring impact of imprisonment is that even upon release, former inmates face stressful consequences of prison life that have health effects. I discuss some of these in the next section on the disruption of social bonds and networks. In short, these ongoing consequences of incarceration—social stigma, poor employment prospects, decreased earnings, family problems, the inability to participate fully in society, lack of control over important aspects of one's life—are secondary stressors and durable strains (Pearlin 1989) that not only are problematic in themselves but also negatively influence health (Massoglia 2008a; Schnittker and John 2007).

There is substantial evidence from the psychoneuroimmunology literature that chronic stress is associated with immune dysfunction (Glaser and Kiecolt-Glaser 2005). Psychological stress occurs when environmental conditions exceed the individual's coping ability, producing distress, anxiety, and negative thoughts, emotions, and moods. The central nervous, endocrine, and immune systems interact with each other in complex ways, and stressors can disrupt their function and weaken immune response. Chronic stressors are especially detrimental and can result in negative health effects by increasing susceptibility to and the severity of infectious diseases. Examples of continuous stressors that have been shown to alter immune functions include isolation and exposure to hostile conditions, both of which are characteristics of imprisonment, and the resulting immunological dysfunction can last for extended periods (Glaser and Kiecolt-Glaser 2005). Stress-induced immune dysfunction perversely increases the inmate's vulnerability at precisely the time when exposure to infectious diseases is greatest, that is, during imprisonment. Of course, the health effects of contracting an infectious disease while incarcerated persist upon release from prison.

While the evidence is weaker, there is some suggestion that chronic stress may also be associated with cardiovascular disease (Dimsdale 2008). Incarceration is a chronic stressor in itself, and the hypertension resulting from stress can persist far beyond the duration of the initial stressor. Since different types of stressors—again, of the type often examined in studies of stress and cardiovascular disease like those associated with jobs,

relationships, and feelings of unfair treatment and discrimination (Krieger and Sidney 1996)—continue following release from prison, the former inmate remains at risk of premature mortality long after release from prison.

Massoglia (2008a) found that relative to those who had not been incarcerated, those who had were at greater risk of being medically diagnosed with stress-related illnesses like psychological problems, hypertension, heart disease, and chronic lung disease. They were also at a greater risk of self-reporting stress-related conditions like chest pain, depression, and general health problems. Schnittker and John (2007) also found significant long-term health effects of imprisonment following release. Consistent with the argument that these effects are stress related, Schnittker and John's results provided considerable evidence that the effects were due less to incarceration itself and more to the enduring stigma associated with it. Similarly, Schnittker et al. (2012) and Turney et al. (2012) both found incarceration to be associated with mental health, including major depression and other serious psychiatric disorders.

Incarceration, Disruption of Social Integration, and Health Effects

Social bonds, networks, support, and capital provide important protective effects against negative health outcomes. From a life course perspective, incarceration is a major turning point and a remarkably disruptive force against social integration (Sampson and Laub 1993; Western 2002). It has corrosive effects on employment, marriage, and other forms of social integration that are otherwise protective of health. The effects of incarceration on labor market opportunities are considerable, diminishing the chances of both employment and earnings. There is substantial evidence of an association between incarceration and subsequent employment, and experimental research reveals that employers are reluctant to hire former inmates (Pager 2003). This effect extends to wages, with prior imprisonment restricting access to career-oriented occupations and often leaving ex-inmates stuck in poor jobs. This cuts off the potential for earnings growth over the employment career (Sampson and Laub 1993) and results in a wage penalty of 10 percent to 30 percent for ex-inmates (Western, Kling, and Weiman 2001). Western (2002) found that the individual-level effect on wage mobility is so strong and imprisonment so widespread among young black men in the United

States that incarceration plays a key role in ethnic wage inequality at the aggregate level. Finally, a simple yet often overlooked fact is that ex-inmates' employment opportunities are often limited to jobs that do not provide health insurance.

Incarceration has negative effects on maintaining stable relationships.³ Being in prison imposes a separation—exacerbated because inmates are often institutionalized far from home—that makes it difficult to maintain the friendship and trust required of a stable relationship (Nurse 2002). Similarly, the social and psychological adaptations required for life in prison are not easily discarded upon release, making it difficult to reintegrate and to maintain healthy relationships with family and friends (Braman 2004). Following release, the stigma of incarceration and the inability to provide for one's family due to the employment effects of prison make an ex-inmate less desirable as a mate (Wilson 1987). These effects are manifested in lower marriage and higher divorce rates among formerly incarcerated men (Huebner 2005; Western 2006).

The past two decades have produced a voluminous literature on the individual and social determinants of health and health inequalities. This literature shows that the relationships with health of employment and marital status are complex, and both relationships are bidirectional. Nevertheless, this literature also clearly shows that employment, income, and the strength of family ties are associated with morbidity and mortality, as are social capital and social support. Ross and Mirowsky (1995) showed that full-time employment slowed declines in perceived health and physical functioning, and studies of the United States (Backlund, Sorlie, and Johnson 1996) and Europe (Ecob and Davey Smith 1999) revealed relationships between income and morbidity and mortality. An analysis of the same case-control data employed in the present study found inverse relationships with premature mortality for both education (as a measure of socioeconomic status) and marriage (Pridemore et al. 2010). The protective effect of marriage against morbidity and mortality, especially for men, is a common finding and has been for decades (Verbrugge 1979).

Individual-level Studies of Incarceration and Mortality

A few studies have examined the association between incarceration and mortality. Most of these studies looked at the effects in the period immediately following release from prison, which is an especially risky time for former inmates. A retrospective cohort

study by Binswanger et al. (2007) of all inmates released from the Washington State Department of Corrections between 1999 and 2003 linked prison records to the National Death Index, comparing mortality rates of the previously incarcerated with the state population. With a mean follow-up period of about two years, they found that those who had been incarcerated were more than three times more likely to die than other residents in the same region. European studies in Scotland (Seaman, Brettle, and Gore 1998) and France (Verger et al. 2003) also found an increased risk of mortality soon after release from prison. Lim et al. (2012) found similar results for those released from New York City jails.

A few studies have looked at the long-term effects of incarceration on mortality. Relative to injuries and other external causes of death that were the focus of studies with shorter follow-up periods, these studies allow the authors to examine the impact of incarceration on deaths due to internal and chronic causes. Rosen et al. (2008) compared the mortality of ex-inmates to other state residents, linking prison records to state death records from North Carolina from 1980 to 2005. They found that ex-prisoners were significantly more likely to experience early death. Spaulding et al. (2011) carried out a cohort study of all persons incarcerated in Georgia state prisons on June 30, 1991. They linked prison and mortality records to determine 15.5-year survival, finding high standardized mortality rates for those who had been released from prison.

DATA AND METHODS

Study Design and Population

This was a large-scale population-based case-control study. It was large scale in both practical and formal terms. Practically, the original project and the present analysis were (1) undertaken by institutions and researchers in multiple countries including Russia, Germany, the United Kingdom, and the United States and (2) scholars from multiple disciplines including epidemiology, demography, medicine, and sociology and (3) involved multiple local agencies like the police, narcology clinics, and the vital registration system. It was also large scale in more formal methodological terms: A prior research project in the city acted as a *de facto* pilot study for the current project, a team of over 30 people interviewed more than 3,500 case and control proxies, the research team collected forensic autopsy data on decedent cases, and measurement research was undertaken to determine

which types of questions were more reliable when using proxy respondents.

A study is population based if the cases come from a precisely defined and identified population and the controls are sampled directly from this population. The most desirable alternative for a population-based case-control study is to sample controls randomly, which was feasible in this study because a population register was available for Izhevsk. As opposed to disease-based research undertaken to treat or cure a specific disease, population-based research refers to human subjects research where the objective is to measure, determine the causes of, and improve the health of populations. This was also the case with the larger project carried out here, as the mission was to address the mortality crisis in the population.

A case-control study is an observational study in which two existing groups that differ on some outcome are identified and compared. This design is usually employed to identify factors that contribute to some condition. In this case, that condition was premature mortality. The potential relationship of the suspected risk factor—in this case incarceration—is examined by comparing the cases and controls with regard to how frequently exposure is present in each group. The use of case-control designs are rare in sociological analyses.

Data for this analysis were collected as part of the Izhevsk Family Study (IFS), which was designed to examine premature mortality among working-age Russian males (Leon et al. 2007; Tomkins et al. 2007). Human subjects and ethical approvals for the study were obtained from the committees of the Izhevsk Medical Academy and the London School of Hygiene and Tropical Medicine. Izhevsk is an industrial city on the western side of the Ural Mountains. Prior research in the region revealed the feasibility of carrying out such a complex investigation in this location (e.g., Shkolnikov, Meslé, and Leon 2001). Izhevsk had a population of about 630,000 residents in 2002 according to the Russian Census carried out that year. It is a typical Russian industrial city, and residents had both an average life expectancy and a distribution of deaths by cause in working-age men that was very similar to that of Russia as a whole. Russia rivals the United States in its incarceration rate, ensuring a high enough lifetime prevalence of incarceration for meaningful analysis given the number of cases and controls in the study.

Cases were deceased men aged 25 to 54 years who died from any cause between October 20,

2003, and October 3, 2005, and who were living in an Izhevsk household with at least one other person at the time of death. The upper age range was purposely truncated at 55 years because the IFS was designed to examine premature mortality (and life expectancy of Russian males was about 60 years). The IFS team was notified of deaths by the registrar of deaths. Cause of death was coded using the 10th Revision of the International Classification of Diseases. Of the 1,750 cases with a proxy interview, cause of death was established in 72 percent of the cases by forensic autopsy, in 11 percent by a non-forensic pathologist, in 11 percent by a doctor who had treated the decedent, and in 5 percent by a doctor who had not treated the decedent. There were no significant differences in the distribution of causes of death between cases for which a proxy interview was obtained and those for which a proxy interview was not obtained (Leon et al. 2007:2003, Figure 1). Controls were living men selected from a 2002 population register and who were living in an Izhevsk household with at least one other person. Each month new controls were randomly selected from within five-year age bands from the sampling frame, such that the control sample with proxy interviews reflected the same ages as the accumulating series of cases with proxy interviews. This sample selection process yielded 1,750 cases and 1,750 controls.

A team of 34 trained interviewers used a structured questionnaire to obtain information about cases and controls from proxy respondents living in the same household. Oral consent was obtained from proxy informants. Nearly all case proxy interviews took place six to eight weeks after death. Proxy interviews were done between December 11, 2003, and November 16, 2005. Case and control proxy interviews were carried out at the same rate throughout the data accumulation period. Interviewers returned to an address up to three times to get a response. When more than one proxy was available, a prespecified priority order was used, with wives or partners being the first choice. Most proxy interviews were with wives or partners (59 percent for cases, 85 percent for controls), followed by mothers (21 percent for cases, 9 percent for controls). Other less common informants included adult offspring, siblings, fathers, or other relatives. For validation purposes, the research team obtained proxy interviews from two informants living in the same household in a subset of 200 cases and 200 controls. The interviews with proxies took place in private to avoid contamination of responses. The questionnaire covered a

wide range of topics, including alcohol consumption, smoking, and social, economic, and demographic information. Most questions were derived from established and validated instruments, and in most cases the reference period for the surveyed behaviors and experiences was the prior 12 months.

Measures

The main independent variable in the present study was lifetime prevalence of incarceration of any sort for any length of time. The question on the instrument asked: "Had he [i.e., the decedent or the control] ever been in any kind of prison?" Response categories included: "yes, in the previous year"; "yes, between 1 and 5 years ago"; "yes, more than 5 years ago"; "no, never"; "difficult to answer"; and "refuse to answer." For the purposes of this analysis, I created a dichotomous variable for lifetime prevalence and a separate categorical variable in which no/never was the reference group, with yes in the prior year, yes 1 to 5 years ago, and yes more than 5 years ago as the other categories.

I included controls for age group and for the known determinants of premature mortality among this group (Leon et al. 2007; Pridemore et al. 2010), including smoking status, marital status, education, and hazardous drinking. Age was included as seven five-year age categories: 25–29, 30–34, 35–39, 40–44, 45–49, 50–54, and 55+ (the latter contained only one case and 11 controls). Smoking status consisted of three categories: non-smokers, ex-smokers, and current smokers. Marital status consisted of five categories: living together in a registered marriage, living together in an unregistered marriage, never married, divorced/separated, and widowed. Educational status consisted of six categories: complete higher education, incomplete higher, specialized secondary, complete secondary, professional, and incomplete secondary. The "specialized" and "professional" categories may be unfamiliar to Western readers; more information can be found in Pridemore et al. (2010). Hazardous drinking was a dichotomous variable coded 1 if during the previous 12 months the person had gone on a drinking binge of at least two days and/or at least twice per week had an occurrence of drunkenness or a hangover or went to sleep with his clothes on due to drinking. The type of drinking binge mentioned here is known as *zapoï* in Russian and is defined as going on a spree of continuous drunkenness lasting at least two days during which the person is completely removed from normal social life. At the design stage of the IFS, a systematic review

(Tomkins 2006) of the validity of proxy informant data related to alcohol consumption led to the key conclusion that the validity of proxy responses was improved if questions were restricted to behaviors that were directly observable. Further, subsequent analyses (Leon et al. 2007) revealed problem drinking as defined here was a strong determinant of mortality among this population, with over 40 percent of all premature deaths among this group shown to be attributable to a similar measure of hazardous drinking.

Analysis

I used logistic regression to estimate mortality odds ratios (ORs) comparing cases to living controls. I estimated two models: the first was for lifetime prevalence of incarceration; the second was for how long before the interview (if ever) incarceration occurred. All ORs were adjusted for the controls described previously. Given the results of model estimation, I compared the distributions of causes of death for cases ever incarcerated relative to cases never incarcerated.

RESULTS

Table 1 shows the distribution of all variables among the cases and controls. As revealed elsewhere (e.g., Leon et al. 2007; Pridemore et al. 2010), there are differentials in premature mortality among this group based on hazardous drinking, smoking status, marital status, and education. Table 1 suggests there may also be significant differences between cases and controls in their experience with incarceration. While only about 5 percent of controls were ever incarcerated, around 17 percent of cases had been incarcerated. One percent of cases and 0.1 percent of controls had been incarcerated in the past year, 3.4 percent of cases and 1.0 percent of controls had been incarcerated between one and five years prior to the proxy interviews, and 12.3 percent of cases and 3.7 percent of controls had been incarcerated at some point at least five years prior to the proxy interviews.

Table 2 shows the mortality odds ratios for the association between incarceration and premature mortality, mutually adjusted for age, hazardous drinking, smoking status, marital status, and education. As expected, problem drinking, smoking status, marital status, and education were all associated with premature male mortality in this sample. Model 1 shows the association of premature mortality with lifetime prevalence of incarceration. Net

of the control variables, males who had been incarcerated were more than twice as likely to experience premature mortality compared to males who had never been incarcerated (OR = 2.2, CI: 1.6–3.0). For Model 2, caution should be taken when interpreting the results for time since incarceration as the cells for incarceration during the last year contain very small counts. Nevertheless, it is interesting to note the substantially heightened risk of premature mortality for those who had been incarcerated in the 12 months prior to the proxy interview (OR = 11.8, CI: 1.5–92.5). The mortality odds ratios for incarcerated 1–5 years prior (OR = 2.0, CI: 1.1–3.8) and more than 5 years prior (OR = 2.1, CI: 1.5–2.9) were essentially the same as each other and the same as the mortality odds ratio for lifetime prevalence of incarceration shown in Model 1.

Finally, Table 3 provides the age-adjusted odds of specific causes of death by lifetime prevalence of incarceration. The comparison category no longer consists of the original controls, but instead of decedent cases who had never been incarcerated. ORs are presented in terms of those ever incarcerated relative to those never incarcerated. Table 3 shows that relative to cases who had never experienced incarceration, those who had been incarcerated were significantly more likely to have died from respiratory diseases (OR = 1.79, CI: 1.19–2.71), infectious diseases (OR = 3.10, CI: 1.74–5.50), accidental poisonings that were not from alcohol (usually drug overdoses; OR = 2.32, CI: 1.11–4.87), and homicide (OR = 1.99, CI: 1.04–3.82).⁴

In summary, the findings revealed that males who had ever been incarcerated were more than twice as likely to die prematurely, that this risk of death was heightened further by incarceration within the past year, and that decedents who had been incarcerated were more likely than other decedents to die from infectious and respiratory diseases, drug overdoses, and homicide.

DISCUSSION

More than 10 million people are imprisoned worldwide, with nearly 2.5 million incarcerated in the United States alone (Walmsley 2009). While the United States and Russia already resort to mass imprisonment, the incarceration rate in two-thirds of the world's nations has increased in recent years (Walmsley 2009). Since 95 percent of all prisoners will eventually be released, the number of former inmates is large and will continue to grow. Estimates

Table 1. Distribution of Variables among Cases and Controls ($n = 3,500$).

	Cases		Controls		Total	
	($n = 1,750$)	Percentage	($n = 1,750$)	Percentage	($n = 3,500$)	Percentage
Ever incarcerated						
No	1,449	82.8	1,662	95.0	3,111	88.8
Yes	292	16.7	83	4.7	375	10.7
Missing	9	.5	5	.3	14	.4
When incarcerated						
Never	1,449	82.8	1,662	95.0	3,111	88.8
In last year	17	1.0	1	.1	18	.5
1–5 years ago	60	3.4	17	1.0	77	2.2
>5 years ago	215	12.3	65	3.7	280	8.0
Missing	9	.5	5	.3	14	.4
Age group						
25–29	131	7.5	130	7.4	261	7.5
30–34	144	8.2	145	8.3	289	8.3
35–39	136	7.8	145	8.3	281	8.0
40–44	306	17.5	293	16.7	599	17.1
45–50	441	25.2	429	24.5	870	24.9
50–54	591	33.8	597	34.1	1,188	33.9
55+	1	.1	11	.6	12	.3
Problem drinker						
No	679	38.8	1,223	69.9	1,902	54.3
Yes	917	52.4	309	17.7	1,226	35.0
Missing	154	8.8	218	12.5	372	10.6
Smoking status						
Non-smoker	133	7.6	373	21.3	506	14.5
Ex-smoker	149	8.5	218	12.5	367	10.5
Current smoker	1,468	83.9	1,158	66.2	2,626	75.0
Missing	—	—	1	.1	1	.0
Marital status						
Married (registered)	930	53.1	1,351	77.2	2,281	65.2
Married (unregistered)	205	11.7	174	9.9	379	10.8
Never married	215	12.3	104	5.9	319	9.1
Divorced or separated	342	19.5	106	6.1	448	12.8
Widowed	57	3.3	15	0.9	72	2.1
Missing	1	.1	—	—	1	.0
Education						
Complete higher	135	7.7	354	20.2	489	14.0
Incomplete higher	36	2.1	43	2.5	79	2.3
Specialized secondary	340	19.4	405	23.1	745	21.3
Complete secondary	560	32.0	535	30.6	1,095	31.3
Professional	442	25.3	299	17.1	741	21.2
Incomplete secondary	209	11.9	100	5.7	309	8.8
Missing	28	1.6	14	.8	42	1.2

suggest nearly 3 percent of U.S. adults and 5 percent of adult males have spent time in a state or federal prison (Bonczar 2003). Similarly, nearly 5 percent of

this sample of Russian males aged 25 to 54 had been incarcerated in their lifetime.⁵ The large number of people ever incarcerated in nations that rely heavily

Table 2. Mortality Odds Ratios for the Association between Premature Mortality from All Causes and Measures of Incarceration ($n = 3,085$).

	Model 1: Ever Incarcerated	Model 2: When Incarcerated
	OR (95 percent CI)	OR (95 percent CI)
Ever incarcerated		
No	1.0 [reference]	
Yes	2.2 (1.6–3.0)	
When incarcerated		
Never		1.0 [reference]
In last year		11.8 (1.5–92.5)
1–5 years ago		2.0 (1.1–3.8)
>5 years ago		2.1 (1.5–2.9)
Age group		
25–29	1.0 [reference]	1.0 [reference]
30–34	1.0 (.7–1.5)	1.0 (.7–1.5)
35–39	1.0 (.6–1.5)	1.0 (.6–1.5)
40–44	1.3 (.9–1.8)	1.3 (.9–1.8)
45–50	1.3 (.9–1.9)	1.3 (.9–1.9)
50–54	1.4 (1.0–1.9)	1.4 (1.0–1.9)
55+	.1 (.0–1.2)	.1 (.0–1.2)
Problem drinker		
No	1.0 [reference]	1.0 [reference]
Yes	3.7 (3.1–4.4)	3.7 (3.1–4.4)
Smoking status		
Non-smoker	1.0 [reference]	1.0 [reference]
Ex-smoker	1.5 (1.1–2.2)	1.5 (1.1–2.2)
Current smoker	2.1 (1.7–2.8)	2.2 (1.7–2.8)
Marital status		
Married (registered)	1.0 [reference]	1.0 [reference]
Married (unregistered)	1.4 (1.1–1.8)	1.4 (1.1–1.8)
Never married	2.6 (1.9–3.6)	2.5 (1.8–3.5)
Divorced or separated	3.3 (2.5–4.3)	3.2 (2.5–4.3)
Widowed	3.6 (1.8–7.2)	3.6 (1.8–7.2)
Education		
Complete higher	1.0 [reference]	1.0 [reference]
Incomplete higher	1.8 (1.0–3.1)	1.8 (1.0–3.2)
Specialized secondary	1.6 (1.2–2.1)	1.6 (1.2–2.1)
Complete secondary	1.7 (1.3–2.2)	1.7 (1.3–2.2)
Professional	2.2 (1.7–3.0)	2.2 (1.7–3.0)
Incomplete secondary	2.7 (1.8–3.9)	2.7 (1.8–3.9)

on imprisonment reveals the potentially broad impact of any association between incarceration and morbidity and mortality.

Mass incarceration disrupts marriage and labor markets (Huebner 2005; Lopoo and Western 2005; Pager 2003; Wilson 1987), perpetuates ethnic and class inequality (Pettit and Western 2004; Western 2002), influences the outcome of national elections

(Uggen and Manza 2002), and places the broader population at greater risk of infectious diseases (Stucker et al. 2008). Recent studies provide evidence that the effects of incarceration extend to negative health outcomes, revealing individual-level effects of incarceration on morbidity (Fazel and Baillargeon 2011; Massoglia 2008a, 2008b; Schnittker and John 2007) and mortality (Binswanger et al. 2007; Rosen

Table 3. Age-adjusted Odds of Specific Causes of Death by Lifetime Prevalence of Incarceration ($n = 1,741$).

Cause of Death	Proportion of Deaths		OR (95 percent CI)
	Ever Incarcerated ($n = 292$)	Never Incarcerated ($n = 1,449$)	
Cancer	7.2	10.3	.75 (.47–1.22)
Cerebrovascular diseases	3.8	6.1	.70 (.37–1.34)
Ischaemic heart disease	11.6	15.5	.85 (.57–1.27)
Other cardiovascular	12.0	12.2	.96 (.65–1.42)
Mental disorder	1.4	1.0	1.36 (.45–4.14)
Respiratory disease	11.6	7.0	1.79 (1.19–2.71)
Infectious disease	6.8	2.3	3.10 (1.74–5.50)
Chronic liver disease + cirrhosis	6.5	6.8	.93 (.56–1.55)
Other digestive disease	4.1	3.7	1.17 (.61–2.22)
Drowning	0.7	1.1	.58 (.13–2.56)
Acute alcohol poisoning	3.1	5.9	.50 (.25–1.00)
Other accidental poisoning	4.1	1.4	2.32 (1.11–4.87)
Homicide	4.8	2.1	1.99 (1.04–3.82)
Suicide	5.5	7.2	.68 (.39–1.18)
External, cause undetermined	4.1	6.7	.53 (.29–.99)
Transport injuries	3.1	2.3	1.24 (.59–2.64)
Other external causes	5.8	4.3	1.26 (.72–2.19)
All other causes	3.8	4.2	.83 (.43–1.61)

et al. 2008; Spaulding et al. 2011) and population-level effects of incarceration rates on life expectancy (Wildeman 2012).

The results of this rigorous large-scale population-based case-control study of incarceration and premature mortality are clear. Men who had been incarcerated were more than twice as likely as those who had not to experience premature mortality. Relative to decedent males who had never been incarcerated, decedent ex-inmates were significantly more likely to die from infectious diseases, respiratory diseases, non-alcohol-related accidental poisonings (usually drug overdoses), and homicide. Considered alongside other research, these findings reveal not only that incarceration has immediate and durable effects on morbidity upon release from prison, but that its consequences may extend to a greater risk of early death. Thus, to the list of extralegal sanctions experienced by those who have been imprisoned, we add this mortality penalty of incarceration.

The findings presented here are consistent with results from prior research, not only in terms of

incarceration's effect on negative health outcomes but also with regard to causes of death and effect size. Relative to decedents who had not been incarcerated, I found that decedents who had been incarcerated were more likely to die from infectious diseases, respiratory diseases, non-alcohol-related accidental poisonings, and homicide. Earlier studies of incarceration and mortality also often found an excess of deaths from these causes, especially infectious diseases, drug overdose, and homicide. The higher risk of death due to infectious and respiratory diseases is consistent with the literature on greater exposure to infectious diseases while imprisoned, including HIV, hepatitis C, and tuberculosis (Johnson and Raphael 2009; Massoglia 2008a; Stuckler et al. 2008).

Further, while effect sizes vary between studies, the ORs estimated here, both for men who had been incarcerated in the year prior to death (OR = 11.8) and for men who had been released at least one year before death (OR = 2.2), are generally similar to those from earlier studies.

Implications for Other Health-related Phenomena

Incarceration and its association with morbidity and mortality likely have implications and collateral consequences for other health-related phenomena. First, the empirical literature provides consistent evidence of an association between criminal offending and premature mortality, especially for chronic and violent offenders, in both Europe and the United States (Lattimore, Linster, and MacDonald 1997; Laub and Vaillant 2000; Paanila, Hakola, and Tiihonen 1999; Piquero et al. 2014; Sattar and Killias 2005). Incarceration, however, is rarely if ever discussed as a contributing factor. Piquero et al. (2014), for example, relied on the work of Gottfredson and Hirschi (1990) and Moffitt (1993) to explain their finding that high-rate chronic offenders experience excess mortality. Laub and Vaillant (2000:96) suggested and tested four competing hypotheses—risk taking and impulsiveness, substance use, poor self-care, and economic and educational deprivation—but found only modest support and concluded that “[a]lthough delinquency is strongly associated with premature mortality, the etiological links remain unclear.” Incarceration is absent from these discussions but may provide a partial explanation, especially since the association between offending and early death is stronger among those most likely to come into contact with the penal system (i.e., chronic, high-rate, and violent offenders) and since offenders are more likely to die from infectious diseases.

Second, not only are ex-inmates a vulnerable population, but they can serve as vectors for disease and other health problems, and thus the negative health consequences of imprisonment extend beyond the incarcerated to family members, communities, and the public. Partners and children of incarcerated men suffer higher rates of negative physical and mental health outcomes (Wildeman, Schnittker, and Turney 2012), and children of incarcerated men are also at greater risk of behavioral problems (Wakefield and Wildeman 2011). Ex-inmates’ partners are at high risk of sexually transmitted infections (STIs) that the men contracted while in prison. Similarly, there is a positive association between a neighborhood’s incarceration rate and its prevalence of STIs (Johnson and Raphael 2009; Thomas and Torrone 2006) and other negative health outcomes, especially in cities (Freudenberg 2001). Further, increasing incarceration rates are directly responsible for the growing

prevalence of TB and multidrug-resistant TB in European and Central Asian nations (Stuckler et al. 2008). A similar pattern exists in the United States, especially with hepatitis C, which spreads to the public when inmates are released (Weinbaum, Lyerla, and Margolis 2003). Through these and other mechanisms, the burden of illness spills over from prisons and ex-inmates, with important consequences for family, community, and population health.

The association between incarceration and morbidity and mortality may contribute to other known health inequalities.⁶ As Thacher (2004:90) stated in his study of inequality in crime victimization by socioeconomic status, “inequality in the distribution of any social burden is of greatest concern when it exacerbates the inequalities that accumulate in other spheres of life.” The tremendous expansion of the penal system has had differential effects by ethnicity (Pettit and Western 2004). For example, Western and Wildeman (2009) estimated that about 23 percent of black men born in the first half of the 1970s could expect to have been incarcerated at some point by the time they were in their mid-30s. The corresponding estimate for white men was less than 3 percent. We also know there are ethnic disparities in morbidity and mortality. This includes differences in life expectancy, TB, and HIV, all of which have now been shown to be associated with incarceration. If blacks are incarcerated at a much higher rate, and if incarceration has both immediate and enduring effects on morbidity and mortality, then it is plausible that incarceration is partially responsible for ethnic disparities in health. Johnson and Raphael (2009) showed, for example, that changes in black incarceration rates are closely linked to changes in AIDS prevalence among black men and women and that the black-white racial disparity in AIDS infection rates during the 1980s and 1990s was almost completely attributable to changes in black incarceration rates during this period. Using state-level panel data from between 1980 and 2004, Wildeman (2012) found that incarceration rates were negatively associated with population health. He also found that the strength of the association was substantial for blacks and that mass imprisonment likely played a role in (1) diminishing health gains in blacks over this period and (2) racial differences in life expectancy. As Thoits (2010) noted in her review, a key conclusion drawn from the literature on stress and health is that the unequal distribution of stress in the general population results in inequalities in physical well-being. Massoglia

(2008a) contends that incarceration is a powerful sorting mechanism that acts to unequally distribute exposure to acute and enduring stressors, thereby increasing the risk of poor health among those who have been imprisoned. The penal system is already recognized as a powerful source of social stratification generally (Wakefield and Uggen 2010), and Massoglia (2008b:275) concluded that “due primarily to disproportionate rates of incarceration, the penal system plays a role in perpetuating racial differences” in health.

Limitations

There are a few main limitations to this study that must be considered. First, the original instrument was not designed specifically to examine the association between incarceration and premature mortality, and thus the question about incarceration was vague. Specifically, it asked, “Had he [i.e., the decedent or the control] ever been in any kind of prison?” This question is not precise as to the type of institution and how long the person was incarcerated. Proxy respondents may have interpreted this question to mean a variety of things, including pretrial detention. On the other hand, while there is considerable heterogeneity in the conditions and length of confinement experienced by formerly incarcerated men, prior studies also did not assess the impact of these characteristics. Further, the lack of precision in the wording of the question likely means the mortality odds ratios represent conservative estimates since the question probably captured not only those who had spent years in prison but also those who spent lesser time in local jails and detention centers.

Second, there are two further design limitations that may lead to underestimating the association between incarceration and premature mortality. The controls who had been incarcerated had yet to reach life expectancy, meaning they could still experience premature mortality. Additionally, to be included in the sample, cases had to have been living with at least one other person at the time of death, and controls had to be living with at least one other person at the time of their random selection into the control series. This was necessary to collect information about cases (and thus controls) via proxy respondents. Evidence shows that previously incarcerated men are more likely to be single and to have relationship difficulties; therefore, they may be more likely to live alone and thus more likely to have been excluded from the IFS sample. Again, however, both limitations would serve to

downwardly bias the mortality odds ratios estimated here and thus result in conservative estimates of the association between incarceration and premature mortality.

Third, this analysis was undertaken using a male sample, and results may not hold for females. Similarly, the study used Russian data and thus may not generalize to other nations. One example is the high rate in Russia of tuberculosis and multi-drug-resistant tuberculosis (MDR TB). In 2005, Russia had TB incidence and mortality rates of 119 and 22 per 100,000 residents, respectively. These high rates are driven largely by mass incarceration in Russia (Stuckler et al. 2008), and MDR TB cases represent about one-quarter of all treated cases in Eastern Europe (Euro TB and the National Coordinators for Tuberculosis in the WHO European Region 2007). Another example is that premature mortality due to stress-related heart disease is very high in Russia following the collapse of the Soviet Union (Leon and Shkolnikov 1998). This might explain the failure to find a higher proportion of deaths due to heart disease among decedents who had been incarcerated. A similar example is suicide. Prior research showed elevated rates of suicide among ex-prisoners relative to the comparison population (Binswanger et al. 2007; Lim et al. 2012; Spaulding et al. 2011). However, Russia's suicide rate, and especially its male suicide rate, is among the highest in the world and increased substantially following the collapse of the Soviet Union (Pridemore and Spivak 2003). Nevertheless, my findings are consistent with theory and with key conclusions drawn from recent research on incarceration and morbidity in the United States (Massoglia 2008a, 2008b; Schnittker and John 2007) and from studies of the impact of incarceration on morbidity and mortality in the United States and elsewhere (Binswanger et al. 2007; Fazel and Baillargeon 2011; Spaulding et al. 2011).

Fourth, it may be that the effect on premature mortality of incarceration and of the time since incarceration varies by age, which could lead to an over- or underestimate of the mortality odds ratio based on the two-year timeframe of data collection. While this may be true, especially since the number of cases and controls who had been incarcerated was relatively small, any bias in the estimates resulting from this is likely minimal for several reasons. First, the sample includes subjects with a range of ages at which premature mortality occurred and a range of the number of years since incarceration, meaning the sample includes those at greater and lower risk points. Next, the vast

majority of those who had been incarcerated fell into one of two groups: 1–5 years or more than 5 years since incarceration. As shown in Table 2, the risk of premature mortality associated with incarceration is the same for both of these groups. Additionally, as described in Footnote 4, I carried out further analyses stratifying cases and controls by age. The results showed no differences in the size of the mortality odds ratios for those less than 40 and greater than 40 years of age. Further, the original IFS design deliberately selected the 25 to 54 male age group because it is a relatively narrow age range where (in the Russian context) the distribution of mortality outcomes is more or less similar, and selection does not begin markedly until 65 years. Nevertheless, the chance for over- or underestimation remains, and this potential bias must be considered.

Finally, while case-control designs provide many advantages, they are vulnerable to selection effects. The Izhevsk Family Study was not focused on this specific question and thus steps were not taken to minimize the risk of selection bias (e.g., there was no way of measuring potential confounders before incarceration occurred).⁷ However, it is important to point out that prior research found that (1) several key childhood correlates of offending, including IQ and early onset delinquency, were not related to premature mortality (Laub and Vaillant 2000); (2) no childhood traits predicted both later offending and physical health (Farrington 1995); (3) the association between chronic offending and early death held even after controlling for individual and environmental risk factors from childhood (Piquero et al. 2014), including substance abuse and psychiatric diagnoses (Stenbacka et al. 2012); and (4) the association between incarceration and morbidity remained after controlling for health prior to imprisonment (Massoglia 2008a). Still, selection effects are a threat to the validity of the results and we cannot rule them out.⁸

Future Research

These and related findings point to a number of avenues for future research. First, further theorizing and theory testing is required to determine the precise pathways through which incarceration influences morbidity and mortality. While I rely on exposure to infectious diseases, stress, and disruption of social integration to explain this association, alternative explanations deserve attention. For example, criminological theories like Gottfredson and Hirschi's (1990) general theory of crime (with

its attention on low self-control and analogous risky behaviors) and Moffitt's (1993) developmental taxonomy (with a focus on the neuropsychological deficits of life course persistent offenders) may help explain the poorer health and earlier death of those who come in contact with the penal system. Further, there may be other causes of negative health outcomes that are specific to time spent in prison, including poor nutrition while incarcerated, drug use in prison, and the likelihood of starting smoking in prison for those who did not already smoke or increasing the amount of smoking in prison among prior smokers. Second, we should examine more closely the impact of incarceration and its association with morbidity and mortality on the relationship between offending and health; family, community, and population health; and health inequalities. The latter seems especially salient, as disparities in the distribution of incarceration by ethnicity may be driving ethnic health inequalities, and the mortality penalty of incarceration might even help explain the black-white gap in life expectancy (Wildeman 2012).

Third, we should test to see if sentence length conditions the association between incarceration and health. As with estimating the impact of incarceration, estimating the causal effect of sentence length is difficult because both it and health outcomes may be endogenous to individual characteristics. As suggested by Wildeman (2011), however, one way to overcome this limitation is to take advantage of exogenous variation in sentence length unrelated to the offender. One such source, employed by Green and Winik (2010) in their analysis of recidivism, is variation in sentence length due to the sentencing decisions made by different judges. Fourth, time since release may have an effect on risk and type of morbidity and mortality. Several studies show a highly elevated risk of mortality, especially from drug overdose and suicide, in the days and weeks immediately following release from prison (Binswanger et al. 2007; Pratt et al. 2006). Lim et al. (2012) provide similar results for those released from jails, and the findings presented here, while based on small cell counts, also show a very high risk of mortality for those who had been incarcerated at some point in the prior year.

Fifth, there are likely important moderating and mediating effects to consider in the relationship between incarceration and premature mortality. It seems plausible that education, employment, and social capital may diminish the strength of this association. There may also be contextual effects

by facility type (e.g., security level, federal vs. state prisons, jails vs. prisons). Prison and community health programs vary in their quality of screening and care, making it likely that the risk of illness due to and the mortality penalty of incarceration vary by penal system. Further, while the association between incarceration and death due to infectious disease is likely direct, the association between incarceration and premature mortality due to other causes may operate indirectly via imprisonment's negative effect on other factors known to influence health, like employment and family stability. Sixth, there is of course the necessity of creating research designs that allow for the estimation of causal effects (Wildeman 2011). There is now clear evidence of an association between incarceration and morbidity and mortality, both soon immediately after release and longer term. However, the designs of these studies remain vulnerable to spurious and selection effects.

Finally, this is an area where truly interdisciplinary research teams are required. Epidemiologists and medical and public health researchers are not only trained in the methods of studying morbidity and mortality, but they are also often employed by or have close relationships with administrative agencies and even prisons themselves. With a focus on patterns, proximate risk factors, and harm reduction, however, their research is often less concerned with the theoretical mechanisms through which the association operates, leaving potentially helpful measures and tests of association unexplored. This is a key sphere in which sociologists can be of benefit, especially given a history of excellent work in discovering the complex causes of health inequalities. There is an increasing awareness of the role of social forces in seemingly individual health outcomes (Link and Phelan 1995), and greater cooperation between disciplines and recognition of the "genes-to-global culture" and "social symbiome" approach to health (Pescosolido et al. 2012) will be fruitful in this area.

CONCLUSION

The findings from this large-scale population-based case-control study revealed that men who had been incarcerated were more than twice as likely to experience early death relative to men who had not been incarcerated. In other words, a mortality penalty accompanies incarceration. I identified four causes of death to which these decedent ex-inmates were significantly more vulnerable compared to other decedent men: infectious diseases, respiratory

diseases, non-alcohol-related accidental poisonings (usually drug overdoses), and homicide. Although both incarceration and premature mortality are endogenous to individual characteristics, the association between them remained after controlling for several of these important characteristics, including hazardous alcohol consumption, family status, and socioeconomic status. While other studies have shown that the period immediately following release from prison is a dangerous time for ex-inmates, the current research design de facto provided for a longer term follow-up, making this study among the first in the literature to do so. I found not only that the association between incarceration and negative health outcomes extends to premature mortality, but that the causes of death to which ex-inmates are vulnerable several years after release are similar to those to which they are at risk soon after release. These causes of death also correspond to the findings from the literature that examines the long-term effects of incarceration on illness.

An often overlooked aspect of the incarceration-health association is that prisons actually provide an opportunity for screening and treating a population that is otherwise unlikely or unable to take advantage of community-based health care. In addition to screening and treatment during incarceration, institutions should work with inmates prior to release on planning for their short- and long-term health care needs. This will benefit not only the individual health of prisoners and former prisoners, but population health as well. Nevertheless, screening, planning, and especially treating inmates are expensive endeavors, and penal systems and their staff are already tasked with serving populations and undertaking functions that would be more efficiently, effectively, and humanely addressed in other, more appropriate settings.

Given the size of penal systems in nations like the United States and Russia, and the impact of incarceration on morbidity and mortality, more research must be done to better understand if and how mass imprisonment is related to population health generally and health inequalities specifically. The causes of health inequalities can be dynamic and may change over time (Link and Phelan 1995), and given the growth of penal systems worldwide (and especially in the United States) in recent decades, Massoglia's (2008b) hypothesis that the penal system has become a system of health stratification should be tested thoroughly (Wildeman 2012).

In the United States, resorting to mass imprisonment was a political decision. Careful research

now shows that many of the extralegal consequences of contact with the penal system—especially the mortality penalty of incarceration—go well beyond what we think about when we think about punishment. Excess morbidity and mortality are inconsistent with the tenets of the main philosophies of punishment—retribution, deterrence, and rehabilitation—and in fact run counter to their ideals. In light of the collateral consequences of mass imprisonment to individuals, communities, and society, the size and the mission of the penal system require fundamental reconsideration. The mortality penalty of incarceration should be part of the debate.

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NOTES

1. While I use case-control data collected in Russia, and while this study is one of the first to address the topic in that country, it is also one of the few studies to address the incarceration-mortality association more generally. Therefore, I focus attention less on Russia specifically and more on the general association between incarceration and premature mortality.
2. Social science research in Russia was stunted during the Soviet era and is only beginning to recover. Thus, there is little research in Russia on the collateral consequences of mass imprisonment at the population-level or on the individual-level effects of incarceration (see Bobrik et al. 2005 for a summary of the scant research and data that have been published). There is little reason to believe, however, that incarceration in Russia is dissimilar to the United States in terms of these main potential explanations for its effect on morbidity and mortality. Exposure to infectious diseases while incarcerated

is greater in Russia, and having been incarcerated increases the risk of the development of tuberculosis 6 to 12 times in the country depending upon place type of incarceration (Coker et al. 2006). Prior incarceration in Russia also influences social stigma and employability and sometimes comes with restrictions on where one can reside.

3. Men at a higher risk of unstable relationships are also at a higher risk of incarceration. For example, while there is little difference in the prevalence of fatherhood between incarcerated and non-incarcerated men, the former are much less likely to be married (see Chapter 6 in Western 2006). Beyond any selection effect, however, imprisonment can be harmful to existing relationships and to the formation of stable relationships upon release from prison.
4. Based on a reviewer's comment, in analyses not shown here, I stratified by age to explore how the time reference of the incarceration measure (i.e., within prior year, 1–5 years before, more than 5 years before) may bias results. For men in their 40s and 50s (when most deaths in this sample occurred), this might produce some variation. Further, a man in his 50s incarcerated within the prior year may be different from a man in his 20s incarcerated within the prior year. As suggested by the reviewer, I stratified the sample into less than 40 years old and more than 40 years old. I was unable to do this stratified analysis for those incarcerated within the past year due to very small cell counts. Among those less than 40 years old, there were only eight cases and one control who had been incarcerated in the prior year. Among those more than 40 years old, there were nine cases and no controls who had been incarcerated in the prior year. I did stratify by age for lifetime prevalence of incarceration. In that case, the results were almost exactly the same for all groups. For the full sample: odds ratio (OR) = 2.2, 95 percent confidence interval (CI): 1.6–3.0; for the <40 sample: OR = 2.1, CI: 1.3–3.7; for the >40 sample: OR = 2.2, CI: 1.5–3.2.
5. Massoglia (2008a, 2008b) also found that 5 percent of his subsample of the National Longitudinal Survey of Youth who were at least 40 years old and had been administered the "Health 40" module had been incarcerated.
6. The data employed in this analysis were from Russia and thus cannot be used to make direct inferences about health inequalities by ethnicity in the United States. However, given the similarity in findings from this study and those from other European nations and from the United States, there is little reason to expect major differences in the general conclusions drawn, nor thus in their implications.
7. Similarly, in this study we cannot discern what proportion of the risk of premature mortality might be due directly to chronic and/or serious offending instead of incarceration.

8. Although one way to address selection effects is propensity score matching (see Massoglia 2008a for an application to incarceration and health), this method is inappropriate for case-control designs. As pointed out by Allen and Satten (2011:52), this method should be limited to prospective studies because (a) "exposure probabilities in a case-control study are not representative of the target population, so the estimated propensity score does not correspond to that in the target population" and (b) "comparing the difference in proportions of persons with disease in the exposed and the unexposed (the typical effect measure for a propensity score analysis) is problematic with case-control sampling, since the proportion of persons with disease in the study population is fixed by design." Månsson et al. (2007) show that when using propensity score matching in case-control studies there is the likelihood of artifactual effects on the odds ratios and (due to the failure of propensity scores to converge to the true value) a reduction in the ability to control for confounding factors.

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