The Effect of Incarceration on Mortality*

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January 28, 2022

Abstract

This paper analyzes the effect of incarceration on mortality using administrative data from Ohio between 1992 and 2017. We first document that long-run survival is *higher* among the incarcerated than similar non-incarcerated defendants. Using event study designs centered around the time of release, we show why: mortality risk halves during the period of incarceration, with large reductions in murders, overdoses, and natural causes of death. However, incarceration does not increase post-release mortality, and so the overall effect is increased longevity. These estimates reflect the high-risk environment faced by defendants when not incarcerated, and suggest non-carceral policies to reduce these risks.

*We thank Vittorio Bassi, John Friedman, Jeff Grogger, Matthew Kahn, Michael Light, Matthew Lindquist, Paulina Oliva, Manaswini Rao, Jesse Shapiro, and seminar participants at the Economics of Crime online seminar, Grinnell, the Midwest Health Economics Conference, and USC for helpful comments and suggestions. Daniela Santos-Cardenas and Alison Lodermeier provided excellent research assistance. This project would not have been possible without the assistance of Lisa Locklin, John Paulson, Karen Sorrell, and Kathy Lamb, who took the time to help us access the data and understand the institutional context. Funding for this project was provided by the Becker Friedman Institute. All views expressed are those of the authors and do not necessarily reflect the opinions of any of the funding organizations. Ohio Department of Health data used in this study were obtained from Vital Records, Ohio Department of Health (ODH). Use of these data do not imply ODH agrees or disagrees with any presentations, analyses, interpretations or conclusions.

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Approximately 10% of American men experience incarceration at some point in their lives (Muller and Wildeman, 2016). Past work has found that incarceration has meaningful effects on a variety of economic outcomes, such as labor force participation and future criminal activity. In this paper, we study the effect of incarceration on another critical measure of welfare: mortality.

Conventional wisdom suggests that incarceration has overall negative effects on health and mortality (Spaulding et al., 2011; Daza et al., 2020), but the sign of the effect is theoretically ambiguous. On the one hand, confinement may damage mental and physical health, and proximity to other inmates may expose prisoners to physical danger or encourage risky post-release behavior (Bayer et al., 2009; Stevenson, 2017). On the other hand, inmates are legally guaranteed healthcare that they might not otherwise have access to, and the restricted access to firearms in correctional facilities may lower the risk of being murdered.

Existing empirical evidence is largely based on comparisons of the mortality rate of the incarcerated to a demographically matched sample of the general population (e.g. Binswanger et al. (2007); Rosen et al. (2008); Wildeman et al. (2016); Daza et al. (2020)). These papers often conclude that incarceration increases mortality, but this strategy is unlikely to recover the causal effect of incarceration. Those engaged in criminal activity likely have a higher baseline mortality risk than the matched sample due to differences in unobservable (and thus unmatched) characteristics, biasing estimates of the relationship between mortality and incarceration upwards.

In this paper, we estimate the relationship between incarceration and mortality using 30 years of court and prison records from the three largest counties in Ohio. We observe 2.6 million misdemeanor and felony cases, which we match to individual-level death records for the date and cause of death.

Our empirical setting has two main advantages over previous work. The first is that we can compare mortality among the incarcerated to a much more comparable control group—defendants who were charged but not incarcerated, rather than the general population. The

second is that because we know the predetermined date of release, we can study the sharp changes in mortality rates that occur for groups of prisoners scheduled to be released at particular times.[1]

To first contextualize our analysis, we compare long-term survival for incarcerated and non-incarcerated defendants. We find that conditional on detailed observables, individuals initially incarcerated have a substantially *higher* likelihood of survival over the next 3, 5, and 7 years as compared with similar individuals not initially incarcerated. For example, after five years, a defendant who was incarcerated for one year is 15% less likely to have died than a similar defendant who was not incarcerated.

Next, we investigate the mechanisms underlying this relationship, focusing on two conceptually distinct effects of incarceration on mortality. The first, the *direct effect,* is the effect of incarceration on mortality risk during the period of incarceration. The second, the *post-release effect,* is the effect of past incarceration on mortality risk.

Using a panel of all defendants over the seven years after sentencing, we find that incarcerated defendants have a more than 60% lower mortality rate during the time of incarceration than similar defendants who were not incarcerated (an average of 230 deaths per hundred thousand annually as compared to 587 deaths per hundred thousand annually). The main sources of these differences are dramatically lower risks of mortality from homicide, overdose, or suicide during the period of incarceration. Defendants also have a lower risk of mortality from natural causes of death such as heart disease while incarcerated, potentially due to increased access to medical care.

The estimated lower mortality risk is the sum of two components: the causal effect of incarceration on pre-release mortality and any difference in baseline mortality risk between the two groups related to unobservable characteristics (i.e., not related to covariates we control for, such as charge type, age, race, and past criminal justice involvement). We

[1] A standard quasi-experimental approach would be to instrument for incarceration with the identity of randomly assigned judges. In Appendix G, we show that this strategy is infeasible for this research question: given the rarity of death, it would be highly underpowered to detect an effect even with data from all of the twenty largest court systems in the United States using random assignment of cases to judges.

provide evidence that the incarcerated defendants have weakly *higher* unobservable mortality risk than the non-incarcerated defendants, implying that the estimated difference between the groups is a lower bound on the causal effect of incarceration. First, auxiliary results from the NLSY using variables unobservable in our main dataset show that incarcerated individuals fare similarly or relatively worse on a host of mortality-related variables *prior* to incarceration (e.g., smoking). Second, the incarcerated defendants have higher mortality risk based on observables; unobservables are likely to be similarly signed (Altonji et al., 2005; Oster, 2019). This is also intuitive given that individuals sentenced to incarceration likely committed more serious versions of the same crime.

Next, we show that mortality rates for the two groups converge precisely at the time of release, and remain statistically indistinguishable for at least 15 years.[2] Mortality rates are also similar for each of the individual causes of death that we look at such as homicide or overdose.[3] For similar reasons as the pre-release estimates, unobserved factors imply that the estimated difference is a conservative bound on the post-release effect of incarceration.[4] This means that we can bound the post-release effect as non-positive, i.e., incarceration does not increase average mortality risk over the seven years following release.[5]

We corroborate these findings using a modified difference-in-differences (DiD) approach to estimate the direct effect, reversing the timing of the traditional DiD and exploiting the removal of treatment (incarceration) rather than the onset.[6] This panel-based research

[2]The similar post-release mortality rates provide one potential indication of our improved control group relative to the existing literature, which uses the general population. In addition, that the change in mortality occurs immediately at the time of release is evidence that incarceration rather than other factors explains the pre-release difference in mortality.

[3]Consistent with Binswanger et al. (2007) and Binswanger et al. (2013), there is a post-release effect on overdose deaths over the two weeks after release. However, this short-run effect does not persist and is too small to shift our estimates of aggregate post-release effects.

[4]Section 2.2.4 discusses one additional factor that would also imply this difference is conservative related to unobserved heterogeneity in mortality risk.

[5]Note that our estimates describe average effects—it could be the case that incarceration increases post-release mortality risk for some individuals and decreases it for others, but these forces balance out.

[6]A standard difference-in-differences approach would compare the change in the mortality rate before and after sentencing for those sentenced to incarceration relative to those who were not incarcerated. However, since both incarcerated and non-incarcerated defendants must be alive at the time of sentencing, this estimator is equal to the difference in mortality rates of the incarcerated and non-incarcerated in the post-sentencing period.

design focuses on the sharp changes in mortality rates around the time of release to control for time-invariant, unobservable differences in mortality risk between the incarcerated and non-incarcerated groups. This comes at the cost of additional assumptions on parallel trends, the direction of the post-release effect, and restrictions on individual-specific heterogeneity in mortality risk, but all of these assumptions find support in our data. Estimates are nearly the same as in the earlier models.

The effects of incarceration observed here are largely a function of this population's high mortality risk when not incarcerated: for example, formerly incarcerated individuals' risk of being murdered in their first year after release is approximately 16 times greater than that for similarly aged individuals in the general population. This is further emphasized by the increase in the direct effect over time. Prior to the escalation of the opioid epidemic in 2010, the direct effect was still substantial, with a mortality reduction of 315 deaths per hundred thousand inmates annually. After this escalation, the direct effect nearly doubles in magnitude, primarily due to the sharp increases in overdose mortality risk outside of prison during this period.

The size of the effects we find—and the large number of people who experience incarceration—means that policies that affect incarceration have a substantial effect on aggregate mortality. For example, our estimates indicate that male overdose deaths between 2006 and 2017 would have been 7.2% higher in the absence of incarceration.

Due to the many other costs of incarceration, use of incarceration for the purposes of mortality reduction is not a desirable policy. However, policymakers should consider policies that target the same population and may capture the mortality-reducing properties of incarceration without the associated costs. These might include increasing access to safe, stable living facilities for individuals released on parole, improving safety in the low-income communities from which most of the incarcerated come, regulations that reduce access to firearms, or temporarily separating criminally-involved individuals from high-risk settings. Another possible area of intervention is healthcare: we observe significant decreases in deaths

due to natural causes such as heart disease as a result of incarceration, suggesting that improvements to healthcare outside of prison for this population might also decrease mortality.

This paper contributes to several areas of research. First, it adds to the health economics literature on determinants of mortality, such as climate (Deschenes and Moretti, 2009; Barreca et al., 2016), economic shocks (Ruhm, 2000; Pierce and Schott, 2016), education (Lleras-Muney, 2005; Clark and Royer, 2013) and health care (Finkelstein et al., 2012; Goldin et al., 2019). We find large effects of a different type of policy—our estimates suggest that incarceration has averted an average of approximately 2,000 deaths annually in the US for the last two and a half decades. For context, that is nearly half of the annual mortality reductions from the 2014 Medicaid expansion (Miller et al., 2019). The paper is also closely linked to the literature on "deaths of despair" (Cutler and Lleras-Muney, 2010; Case and Deaton, 2015; Pierce and Schott, 2016); 58% of all overdose deaths in the region we study are of individuals with past criminal justice system involvement. We observe that incarceration particularly reduces mortality risk for the group most prone to deaths of despair, middle-age white men.

We also contribute to the study of the effects of incarceration in the United States. Previous work in economics has focused on outcomes such as recidivism, labor market success, and family spillovers (Kuziemko, 2012; Mueller-Smith, 2015; Rose and Shem-Tov, 2019; Norris et al., 2021). There is a substantial sociology and public health literature on the effects of incarceration on mortality in the United States, where the standard methodology is a comparison of the mortality rates of the incarcerated or formerly incarcerated to samples of the general population that may be matched based on age, race, gender or other observable characteristics. Nearly all of these studies conclude that experiencing incarceration increases post-release mortality (Binswanger et al., 2007; Rosen et al., 2008; Spaulding et al., 2011). More recent papers find relatively consistent evidence of lower mortality while incarcerated for black defendants, but findings are mixed for white defendants (Patterson, 2010; Rosen et al., 2011; Spaulding et al., 2011; Wildeman et al., 2016). However, both the magnitude

and sign of these estimates may be flawed since this method does not account for the many other characteristics on which the incarcerated differ from the general population and which may cause them to experience heightened mortality risk.[7]

Using data with a more comparable comparison group and sharp variation in incarceration status at the time of release, our findings contrast with existing work: we find consistent reductions in mortality during incarceration for all demographic groups and no increase following release. Our paper is most related to the contemporaneous work of Hjalmarsson and Lindquist (2020), which exploits policy reforms to study the effect of incarceration length on post-release mortality in Sweden. Hjalmarsson and Lindquist (2020) focus on the intensive margin, while we focus on the extensive margin. While they find that longer periods of incarceration reduce post-release mortality, it is notable that both studies differ from previous research suggesting detrimental health effects.

1 Background

1.1 Setting

We use data from the three largest counties in Ohio: Franklin County (population of 1.3 million, contains Columbus), Cuyahoga County (population of 1.2 million, contains Cleveland), and Hamilton County (population of 0.8 million, contains Cincinnati). All three counties contain an urban core surrounded by suburbs, and have similar racial compositions (approximately 62% white, 27% black) and median household incomes ($52,000).

Ohio is a good setting to study the relationship between the criminal justice system and mortality, as it is broadly representative of the United States on both dimensions. 790 of

[7] Appendix B uses the National Longitudinal Survey of Youth (1997) to compare mortality risk factors between the general population, incarcerated persons, and those who are charged but not incarcerated (our preferred comparison group). We show that after accounting for age, race, and gender, the general population has far lower mortality risk than the incarcerated, while the incarcerated have similar risk profiles to the charged but non-incarcerated. Section 3.3 discusses further how our estimates contrast with existing literature.

100,000 Ohio adults are incarcerated, and the three-year recidivism rate for the formerly incarcerated is 39.6%, as compared to national averages of 780 and 43.3% respectively (Pew, 2011; Carson, 2018; Kaeble and Cowhig, 2018). Between 1999 and 2018, age-adjusted mortality in Ohio (851.3 per 100,000 annually) was similar to the national average (775.5), and mortality rates for the leading causes of death are also quite similar (National Center for Health Statistics, 2020). Safety in Ohio correctional facilities is also similar to most states so we expect a similar treatment effect of incarceration; 79.4% of the US population lives in states within 25% of the inmate mortality rate in Ohio (Panel A of Figure 1). After 2010, Ohio experienced elevated overdose deaths relative to the rest of the US (Panels B and C of Figure 1), but was otherwise similar to the rest of the US through the sample period. We will later divide our estimates into the pre- and post-2010 period and show that incarceration became substantially more protective after the escalation of the opioid epidemic.

1.2 Data

We collect and match administrative court, prison, and mortality data. Adult court records are available starting in the early 1990s (exact date depending on the county) and contain the full case history, including charges, sentencing date and decisions (punishment type and sentence length), and defendant characteristics (name, date of birth, gender, race, and home address). We collected all available misdemeanor and felony case records from the three counties, totaling 2.6 million cases and 862,505 unique defendants.[8]

We construct the data at the quarterly level, and code whether an individual is incarcerated in a given quarter based on the sentence given at the time of sentencing in the court records. We use this instead of the actual release date to avoid bias induced by compassionate release (when terminally ill individuals are released prior to their death). Ohio has had determinate sentencing since 1996—encompassing nearly all of the study period—meaning that for most inmates, the amount of time served is very close to the sentence. We confirm

[8]See Norris, Pecenco, and Weaver (2021) for details.

that this is true in our data in Appendix A.

We match the court data to mortality records from the Ohio Department of Health, a restricted-access administrative dataset based on the universe of Ohio death certificates between 1990 and 2017. It contains the name, dates of birth and death, and cause of death for the deceased. We match to the court data based on name and date of birth.[9] We also merge the court records with the Social Security Administration Death Master File (DMF) to measure deaths outside of Ohio. This contains the universe of reported deaths in the United States before early 2010 for individuals with social security numbers. Due to the shorter time period and the lack of information on causes of death in the DMF, we rely primarily on the Department of Health records.

2 Results

2.1 Long-run survival and incarceration status

As a first step, we look at how non-incarcerated and incarcerated individuals differ in their likelihood of survival over time. We divide the sample into five groups. The first group is defendants who were charged with a criminal offense but not incarcerated. The remaining four groups are defendants who were sentenced to incarceration for periods of less than 1 year, 1-2 years, 2-3 years, and 3-4 years. In Figure 2, we plot the share who remain alive in each of the seven years following initial sentencing by group. For example, among those who were not incarcerated, just over 99% are alive 1 year after sentencing, but this drops to 95.4% after 7 years.

The figure reveals two striking patterns. First, those who were incarcerated have consistently higher survival probabilities than those who were not. This is somewhat surprising because the incarcerated should have a higher baseline risk of mortality—they are more

[9]For each match, we block on date of birth, and keep matches with a Jaro-Winkler score higher than 0.9 for first and last name.

engaged in other risky behaviors (see Appendix B) and have presumably committed more serious crimes. Yet after 7 years, those who were incarcerated for 3-4 years are nearly 2 percentage points less likely to have died, which amounts to a mortality rate about half that of the non-incarcerated. Furthermore, the probability of survival increases with sentence length, even though individuals receiving longer sentences were on average convicted of more serious crimes.

Second, the shape of the survival curves suggests that these mortality differences are determined largely by incarceration. During the period of incarceration, the slope of the survival curve—which corresponds to the mortality rate—is similar across all of the incarcerated groups. This is followed by a kink at the time of release, where the slope becomes discontinuously more negative. Interestingly, the post-release curves for the incarcerated appear to be roughly parallel to the non-incarcerated curves, suggesting that the period-wise mortality rates for the non-incarcerated and post-release incarcerated are similar.

Table 1 conducts a similar exercise but in regression form. For each case in our data, we regress whether the individual survives for 3, 5, and 7 years after the date of decision on the number of years for which the individual was sentenced to incarceration in that case.[10] An advantage of this approach is that we can control for observable differences between the groups that may be correlated with both sentencing and mortality, such as defendant age or the offense with which they were charged.[11] Again, longer periods of incarceration correspond to higher probabilities of survival, with an additional year of incarceration reducing mortality by about 0.4 percentage points after 7 years. Furthermore, the coefficients are statistically indistinguishable across the 3-, 5-, and 7-year horizons. Since the incarcerated defendants in the longer time horizons spend substantially longer outside of prison, this is most consistent with the differences in long-term survival accruing during the period of incarceration, rather

[10] This is equal to zero if the individual was not sentenced to incarceration. In the regressions, the sample of incarcerated individuals is restricted to individuals with sentence lengths less than the corresponding study period length.

[11] This specification also has the advantage of comparability with the medical literature, which prefers this approach for reasons related to unobserved heterogeneity that will be discussed later in the paper (e.g. Stensrud and Hernán (2020); Bartlett et al. (2020)).

than after release.

The next section of the paper discusses whether this mortality reduction should be interpreted as a causal function of incarceration, as well as how to decompose the effect of incarceration into pre- and post-release effects.

2.2 Pre- and post-release effects of incarceration on mortality

In this section, we investigate why incarceration is associated with reductions in mortality, and discuss whether this relationship should be interpreted as causal. We first document that the release from incarceration leads to large and nearly instantaneous increases in the likelihood of mortality, suggesting that incarceration itself is the key factor, rather than other defendant characteristics. We then compare incarcerated and non-incarcerated defendants in the pre-release and post-release periods. Consistent with Section 2.1, the results indicate that incarceration greatly reduces mortality before release and does not increase post-release mortality. We conclude that the large increases in survival for the incarcerated are explained by the causal direct (pre-release) effect of incarceration.

2.2.1 Prisoner mortality before and after release

We begin by conducting a period-by-period comparison of mortality around the time of release. To do so, we construct a panel dataset over the seven years after sentencing for each defendant, focusing on mortality and incarceration status over this period.[12] We aggregate the data into 14 half-year periods and include all periods in which the individual has not previously died. The relevant empirical specification is:

$$died_{iact} = \sum_{s=-14}^{14} \lambda_s Sentenced_{ic} \times \mathbb{1}[\text{Time to release} = s] + X_{iact}\phi + \eta_t + \gamma_a + \psi_c + \varepsilon_{iact} \quad (1)$$

[12] We select 7 years since 95% of sentence lengths are less than 5 years, so this adequately covers most pre- and post-release periods. We drop individuals with sentences of more than 7 years (0.5% of cases) since they would otherwise only be observed in the pre-release period. For robustness, we will also show a similar analysis in Section 2.2.3 extending up to 15 years post-release and including individuals with longer sentences; the results are similar (Figure A5).

where $died_{iact}$ is a binary indicator for whether individual i died during time t. $Sentenced_{ic}$ is a binary indicator for whether the individual was initially sentenced to incarceration. η_t is a fixed effect for the number of quarters since charges were filed against the defendant, γ_a is a fixed effect for defendant age at time t, and ψ_c is a court-month fixed effect, where each county has one court for misdemeanors and one for felonies. X_{iact} is a vector of controls, including fixed effects for charges against the defendant (1607 charge categories), number of previous cases, and number of previous incarcerations. Standard errors are two-way clustered at the defendant and court-month levels.[13]

In this regression, λ_s represents the difference in mortality between initially incarcerated defendants in the relative-to-release period s (e.g. $s = -1$ is one period before release, $s = 1$ is one period after release) as compared to individuals not sentenced to incarceration.[14] Note that the specification includes fixed effects for time since sentencing, crime type, and other defendant characteristics. λ_s thus reflects differences between observably similar individuals who differ in whether they were initially incarcerated, which may be the result of incarceration or unobservable, pre-existing differences between the groups.

Figure 3 plots the coefficients λ_s.[15] Incarcerated defendants have much lower mortality hazards while imprisoned than observably similar non-incarcerated defendants. For each of the periods before release, this difference is more than 60% of the post-release mean of 587 deaths per 100,000. However, after release the difference between the two groups disappears, and is statistically indistinguishable from zero in each of the subsequent periods. This sharp change—consistent with what we saw in Figure 2—suggests that differences in the mortality between the incarcerated and non-incarcerated groups in the pre-release period stem from incarceration itself as opposed to other factors.

[13] It is necessary to cluster at the defendant level due to serial correlation and because defendants may be charged multiple times. The appropriate unit for the second level of clustering is less clear. Our goal is to account for correlation in potential outcomes that may arise because of common shocks (e.g. crime waves). We take the court-month as a reasonable level at which this might be a concern.

[14] Using the sentenced rather than actual release date is key with mortality as the outcome, since releases would otherwise be observed only for those who are still alive.

[15] Figure A2 is a standard event study in which the second post-release period is the reference period, as compared to Figure 3 which has no reference period.

As a means of aggregating the estimates, we estimate the following specification:

$$died_{iact} = \beta_1 Sentenced_{ic} \times PreRelease_{ict} + \beta_2 Sentenced_{ic} \times PostRelease_{ict} + X_{iact}\phi + \eta_t + \gamma_a + \psi_c + \varepsilon_{iact} \quad (2)$$

Equation 2 is the same as Equation 1, but aggregates the pre- and post-release coefficients: β_1 corresponds to the average difference in mortality risk between the incarcerated and non-incarcerated over the pre-release period after controlling for observables, while β_2 corresponds to the average difference over the post-release period.

Column (1) of Table 2 estimates the aggregate differences in pre- and post-release mortality, conditional on the same controls used in Equation 1. It reveals that during the period of incarceration, the mortality rate for the incarcerated is approximately 357 deaths per hundred thousand lower than among non-incarcerated individuals who are otherwise similar on observable characteristics. Consistent with Figure 3, we cannot reject that the mortality rates are the same during the post-release period. Columns (3)-(8) of the table break down these differences by individual causes of death. While this will be discussed in more detail later in the paper, two main patterns emerge: all causes of death are lower during the period of incarceration, and the largest differences arise from lower risk of homicide and drug overdose. This analysis has so far been descriptive, but the next two sections discuss how these estimates can be used to understand the causal effect of incarceration in both the pre- and post-release periods.

2.2.2 Pre-release mortality

Table 2 provides estimates of the difference in mortality risk between the incarcerated and observably similar individuals who were accused of criminal offenses but not incarcerated. In the pre-release period, this difference will be equal to the causal effect of incarceration on pre-release mortality plus any difference in baseline mortality risk that is not accounted for by the covariates. If we were able to determine the sign of the differences in unobservable

mortality risks, this would allow us to use the estimated difference to bound the causal effect of incarceration on mortality in the pre-release period. For example, if the incarcerated have unobservably higher mortality risk than the non-incarcerated, this would mean that the pre-release mortality difference *understates* the extent to which incarceration reduces mortality, and so this difference is a conservative lower bound on the causal effect of incarceration.[16]

It is intuitive that the incarcerated should have unobservably higher mortality risk: given the charge and criminal history fixed effects, individuals sentenced to incarceration are likely to have committed more serious versions of the same crime, or have unobservable characteristics that dispose the judge towards incarcerating them.[17] It seems plausible that both factors would be positively correlated with mortality risk.[18] However, there are two concrete pieces of evidence that further suggest that the underlying unobservable mortality risk is higher for the incarcerated.

First, we assess selection on observables. Columns (1)-(2) of Table 2 show our main regression specification with and without controls, X_{iact}. Including controls shrinks the estimated difference between the incarcerated and non-incarcerated in the post-release period by 44 deaths per 100,000, meaning that the observable characteristics of those sentenced to incarceration indicate a higher mortality risk as compared to the non-incarcerated. The positive selection along observable characteristics suggests positive selection along unobservable characteristics, as is commonly assumed in the economics literature (e.g. Altonji et al.

[16] Another way of estimating the incarceration-mortality relationship would be to instrument for incarceration using the random assignment of cases to judges, as in other work with this data (Norris et al., 2021). Appendix G shows that despite a strong instrument (first stage $F = 5,605$) and large number of randomly assigned cases (809,347 cases), the IV estimates are imprecise due to the low likelihood of mortality. Monte Carlo simulations in Appendix G further show that a judge-design is unlikely to ever be empirically feasible: this approach would be severely underpowered even with 20 years of data from the twenty largest court systems in the US using random assignment of cases to judges, an order of magnitude more data than has ever been used with this research design.

[17] They may also be incarcerated due to idiosyncratic factors unrelated to their individual characteristics (e.g., severity of the randomly assigned judge), but that would not bias our estimates.

[18] One possible reason incarceration could be negatively correlated with mortality risk is if judges were reluctant to incarcerate terminally ill defendants. However, when we examine mortality among the non-incarcerated population in the weeks after sentencing in Figure A3, we see no spike in deaths immediately following the decision. We take this as evidence against this hypothesis.

(2005); Oster (2019)).[19]

Second, Appendix B tests explicitly for differences between the incarcerated and non-incarcerated in mortality risks that are unobservable in the main data. We use the National Longitudinal Survey of Youth (NLSY), which contains information on behaviors and characteristics related to mortality risk that are unobservable in our court data, such as use of hard drugs or membership in a gang. We can thus assess these mortality risks for NLSY individuals who are incarcerated as compared to those who are charged with a crime but are not incarcerated; in other words, we can use the NLSY to observe factors that are unobservable in the Ohio court data. Across the board, the incarcerated have either higher or similar "unobservable" mortality risks as compared to the non-incarcerated, indicating that the unobservable differences in mortality risk in our main data should be positive or close to zero.

Put together, this evidence indicates that our estimate of β_1 in column (1) of Table 2 is at worst a conservative lower bound on the extent to which incarceration reduces mortality during the period of incarceration.[20] This indicates that incarceration reduces mortality risk by at least 357 deaths per hundred thousand ($p < 0.001$) relative to the post-release mean of 587. This is a 61% reduction in mortality risk, almost the same as the difference in mortality between smokers and non-smokers aged 45-54 (Banks et al., 2015).

We also use detailed cause of death information to understand the factors underlying this effect. The most common non-natural cause of death in our sample is overdose (32% of deaths in the first year after release), which approximately halves during the period of incarceration, declining by 101 deaths per hundred thousand (column 3).[21] This reduction may reflect

[19] Using the same data as this study, Norris et al. (2021) employs an examiners design and finds positive selection along both observables and unobservables in the relationship between incarceration and post-release criminal activity. While criminal activity and mortality risk are different, they are almost certainly positively correlated along unobservable dimensions such as risk-taking, providing evidence in favor of positive unobservable selection here.

[20] Section 2.2.4 will discuss how the presence of compositional changes due to survivorship bias could imply that this estimate is even more conservative as to the extent that incarceration reduces mortality, but shows that this is unlikely to be important in this context.

[21] There are fewer observations in column 3 since we restrict to the period when ODH began coding overdose deaths (after 1999).

addiction treatment, greater ability to treat overdoses, or more difficulty obtaining narcotics while incarcerated.

Contrary to portrayals in popular media, murder is greatly reduced during the period of incarceration (column 4).[22] Murder is particularly important since it is the third most common risk-factor in this sample (25% of deaths in the first year after release), highlighting the dangerous environment faced by defendants outside of correctional facilities. The presence of correctional officers and lack of access to firearms while incarcerated both likely play a significant role; firearms are involved in 85% of homicides in our sample.

Inmates are constitutionally guaranteed medical care, and there may also be changes to diet or lifestyle that affect mortality risk. We find a large (52 per hundred thousand) reduction in deaths due to natural causes during the period of incarceration (column 5 of Table 2). Panel B of Table 2 finds the gains mostly come from reductions in heart disease, infections, and non-classified causes.[23] Even if the quality of prison medical care is poor, many inmates may still receive better care than they would otherwise. For example, 79.9% of inmates with persistent medical problems report being examined by a medical professional upon intake and twice as many inmates with serious mental health conditions receive psychiatric medication during incarceration as compared to prior to arrest (Wilper et al., 2009); a recent paper using data from Norway finds that prison mental health services can improve long-run patient outcomes (Bhuller et al., 2021).

Mortality due to non-overdose unintentional injuries and suicides declines by 33 and 35 deaths per hundred thousand respectively (columns 6 and 7) respectively during the period of incarceration. The largest category of unintentional injuries is car accidents, and so that effect is mostly explained by reduced use of vehicles while incarcerated. The reduction in

[22] It is not completely eliminated. We observe 76 homicides during incarceration. Murders or other causes of death are unlikely to be misreported as the coroner or medical examiner has jurisdiction over prison deaths and is not employed by the correctional facility.

[23] Prior work has found a strong relationship between cohort-level incarceration rates and AIDS rates before 1996 (Johnson and Raphael, 2009). In contrast, even though half of deaths due to infections are attributed to AIDS in our data, we find that incarceration slightly *reduces* infection-related deaths. One potential explanation for this discrepancy is that our data come mostly from the years after 1996, when the introduction of antiretroviral drugs substantially reduced AIDS-related mortality.

suicides may reflect the presence of correctional officers or reduced access to firearms (which are involved in 38% of suicides in our sample). Importantly, the next section of the paper examines the longer-term effects of incarceration on mortality and finds no evidence that suicides averted by incarceration are systematically displaced to the post-release period.

The cause-specific results are informative about the key factors underlying the overall reduction in mortality risk during the period of incarceration. However, we view them as only approximations due to competing risks: incarceration may cause switches in cause of death, such as if an individual would have died from an overdose if they had not been incarcerated, and instead dies of a heart attack while incarcerated. If such switching occurs, our estimates may not fully reflect how incarceration changes risks for a particular cause (e.g. Stensrud et al. (2020)). In practice, this should mostly affect the estimates for natural-cause mortality. This is because during the period of incarceration, the absolute risks of homicide, suicide, and accidents are close to zero. As a result, there will be some individuals who would have died due to those factors if they were not incarcerated, but instead die while incarcerated from other causes. Natural causes of death are the most common cause of death while incarcerated, so the likely "switch" will be towards natural causes of death. As a result, our estimates are probably conservative as to the extent to which incarceration reduces natural-cause mortality.

In summary, we find that mortality is dramatically lower during the period of incarceration. The sharp shift in mortality around the time of release reinforces the point that this is not just due to pre-existing negative selection on mortality risk, and instead provides an informative lower bound on the causal effect of incarceration.[24] The results on individual causes of death provide further support for this interpretation: for example, it is intuitive that the risk of homicide and overdose would be lower in an institutionalized setting where it may be more difficult to access firearms or opioids, and prison staff are present. The magnitude of our estimates indicate that the relationship between incarceration and pre-release

[24] Section 2.3 will use the timing of release as a source of identification, but that will require some additional assumptions.

mortality is economically important.

2.2.3 Post-release mortality

Incarceration might also affect prisoner health and mortality risk after release. On the one hand, the reduction in mortality risk during incarceration could persist. For example, the previous section showed that mortality from natural causes declines during the period of incarceration; experiencing medical care during the period of incarceration might conceivably have persistent positive effects on health. On the other hand, prisoners may be exposed to infection risks or form peer connections that increase mortality after release. Given that the post-release period is far longer than the period of incarceration for most inmates, it is important to consider post-release effects when determining the overall effect of incarceration on mortality.

As with pre-release mortality, the difference in mortality between formerly incarcerated and non-incarcerated defendants (β_2 in Equation 2) can be used to bound the causal effect of incarceration on post-release mortality risk. This difference is the sum of three components: (1) the causal effect of incarceration on post-release mortality (post-release effect); (2) differences in unobservable mortality risks between the incarcerated and non-incarcerated that existed at the time of sentencing; and (3) differential changes in the mortality risk profile of the incarcerated/non-incarcerated due to survivorship bias (Hernán, 2010; Stensrud and Hernán, 2020).

The first component is the one that we would like to estimate. The second component is the same unobservable selection issue addressed in the previous section: the evidence shows that the incarcerated have, if anything, slightly higher pre-existing unobservable mortality risks. The third component reflects the fact that mortality risk is lower for the incarcerated during the period of incarceration; as a result, the sample of incarcerated individuals who survive into the post-release period may include more high mortality risk individuals than among the surviving non-incarcerated who were sentenced at the same time. The next

section of the paper, Section 2.2.4, investigates this third component in greater detail—by estimating it directly and using simulations to bound how it could affect our estimates—and finds that any compositional issues are likely to have negligible effects on our results.

In column (1) of Table 2, we cannot reject equality of β_2 with zero. Our estimates are quite precise; we can rule out differences in post-release mortality that are greater than 8% of our estimate of the direct effect at the 5% significance level. Given that both the second and third components discussed above are at least weakly positive, this means that the causal effect of incarceration on post-release mortality must be at least non-positive, i.e., incarceration does not increase average post-release mortality risk.

Despite the lack of evidence for aggregate post-release effects, there might be modest effects for specific causes of death. For example, there could also be time-varying post-release effects that average out to zero over the period of study, such as if medical care during incarceration causes short-run reductions in post-release mortality, but other factors increase longer-term post-release mortality. Figure 3 evaluates this possibility by estimating the period-by-period comparison for each cause of death. We cannot reject that the average values of the post-release coefficients are equal to zero for any individual cause of death and nearly all coefficients are statistically indistinguishable from zero, indicating a consistent lack of post-release effects.

Previous work has found that over the two weeks following release from prison, overdose mortality is elevated relative to later periods (Binswanger et al., 2007, 2013). Using prison release data, we also find that overdoses are elevated in the two weeks following release, and that the magnitude of our estimates are comparable to this previous work (Figure A4). However, overdose deaths enter into a steady state after that, and the short-lived effect does not result in aggregate post-release effects since two weeks is only a small portion of the post-release half-year. This reinforces that the two weeks after release are a priority period for public health interventions.

While deaths averted by incarceration must occur eventually, the impact on overall

longevity will depend on how long they are delayed for. To help answer this question, we extend the period of study to include the 15 years following release in Figure A5. We still cannot reject a joint null of no mortality difference between the previously and never-incarcerated over all post-release periods ($p = 0.331$). This means that the deaths are averted for at least 15 years and implies that the change in overall longevity as a function of incarceration is likely to be substantial.

Taken together, the results indicate that incarceration increases longevity. As will be discussed further in Section 3.3, this finding contrasts with existing research which argues that incarceration increases post-release mortality (e.g. Rosen et al. (2008); Spaulding et al. (2011); Binswanger et al. (2013); Daza et al. (2020)). We also do not observe measurable increases in causes of death that could potentially be displaced from the incarceration period into the post-release period due to individual behavior, such as suicide or overdose. At the same time, the formerly incarcerated have the same risk of overdose as the non-incarcerated, suggesting that prison drug treatment is not particularly effective at reducing post-release overdose.

2.2.4 Effects of unobserved heterogeneity

In the previous sections, we found that mortality is dramatically reduced during incarceration, which might generate further compositional differences between the incarcerated and non-incarcerated. In this section, we assess the magnitude of this effect and find that it indeed is unlikely to affect our results.

As is usual in hazard models, persistent unobserved heterogeneity in mortality risk across individuals can lead to compositional effects as individuals exit the sample over time due to mortality. Appendix C uses a simple proportional hazards model to formally develop the implications of unobserved heterogeneity for our estimates, but we summarize them here. With unobserved heterogeneity, if incarceration reduces the risk of death during the time of incarceration, as we find, the incarcerated population would progressively develop

a higher average unobserved mortality risk relative to the non-incarcerated group. This is because individuals in the incarcerated population with high unobserved mortality risk would be less likely to die. This will reduce the pre-release difference in mortality risk between the non-incarcerated and incarcerated, implying that the estimated difference is a more conservative estimate of how incarceration reduces pre-release mortality risk. After release, there would be more individuals with high unobserved mortality risk in the surviving ever-incarcerated population as compared to the never-incarcerated; again, this will bias the post-release difference upwards and make that estimate conservative as to the extent that incarceration reduces post-release mortality.[25]

We use two empirical approaches to establish the extent to which this survivorship bias may affect our estimates. First, we directly estimate the variance of the unobserved heterogeneity in a parametric hazard model.[26] Second, we conduct simulations to assess the likely degree of bias. Both approaches suggest minimal bias.

To directly estimate the degree of heterogeneity, we model each individual i's likelihood of mortality in day t as taking the proportional hazards form $\lambda_i(t|\mu) = \mu\lambda_0(t)\theta_i$, where $\mu = \exp\{x'\beta\}$ reflects differences in mortality risk arising from observed covariates x and $\lambda_0(t)$ is a commonly-shared baseline hazard. θ_i is time-invariant individual-specific mortality risk that has mean 1 and variance σ^2. The variance term reflects the extent of dispersion in latent mortality risk (conditional on observed covariates x): if there is significant dispersion across individuals, then this would be reflected in a higher value of σ^2.[27,28]

[25] A reduced form way to think about the effects of unobserved heterogeneity are as mortality displacement—deaths averted during incarceration are shifted to the time periods following release. This is the reverse of the "harvesting" effect in the temperature-mortality relationship (Deschenes and Moretti, 2009; Chirakijja et al., 2019).

[26] This approach to testing for unobserved heterogeneity follows recent papers in economics (Mastrobuoni and Pinotti, 2015).

[27] Identification of mixed proportional hazards models with single-spell data requires variation in covariates to pin down the unobserved heterogeneity parameters (Van den Berg, 2001). In our context, time-varying binary and predictable covariates of whether an individual is incarcerated identifies the variance of unobserved heterogeneity without further assumptions required, such as continuity (Honoré, 1991).

[28] Unobserved heterogeneity is operationalized at the person by prison spell level; there is no additional information from allowing person-specific unobserved heterogeneity since this is a hazard model with only a single possible failure (i.e., death).

Table 3 presents the results. In column (1), we model the hazard rate as a function of whether an individual is incarcerated pre-release, incarcerated post-release, a gamma-distributed unobserved heterogeneity component, Weibull hazard, and a set of baseline characteristics of the individual. We find large reductions in mortality risk during incarceration ($\beta = -1.06, p < .001$)[29] and a difference in post-release mortality risk close to 0; both results are consistent with our main findings. We estimate the variance of unobserved heterogeneity to be negligible, ($\theta < 10^{-5}$, $p = 1$) in column (1), providing strong evidence against unobserved heterogeneity of a magnitude that would substantively affect our results. Column (2) changes the distributional assumption of unobserved heterogeneity to inverse-Gaussian; the models are indistinguishable. The results are also robust to changing the covariates included and the assumption of a Weibull-distributed hazard. Column (3) estimates the same Weibull model with no unobserved heterogeneity, column (4) adds court-year and offense fixed effects, and column (5) instead flexibly estimates the hazard using a semiparametric Cox model. All results are qualitatively the same.

Second, Section C1 runs simulations to examine how the presence of unobserved heterogeneity affects our estimates. Using our data, we generate simulated data under assumptions of: (i) no unobserved heterogeneity, i.e., mortality risk is only determined by observable factors; and (ii) time-invariant unobserved heterogeneity, modeled using a gamma distribution that preserves the overall mean mortality risk from the no unobserved heterogeneity case but progressively increases the variance. We run our benchmark regression in each of the data sets and compare the distribution of coefficients under each assumption. We find that even a high degree of unobserved heterogeneity does not substantially affect our coefficient estimates: for example, even under unobserved heterogeneity that quadruples the variance in mortality risk as compared to the no unobserved heterogeneity case, both the pre- and post-release coefficients are biased upwards by only 15 deaths per 100,000 (relative to the estimated pre-release effect of -357). This reflects the fact that the probability of mortal-

[29] This is a 65% reduction in mortality risk relative to the baseline hazard rate, which is very similar to the estimates in Table 2.

ity in any given period is not very high for even the riskiest set of individuals—and as a result, the compositional changes induced by stints of incarceration are not very large.[30] The simulations also reinforce that any bias from unobserved heterogeneity would cause the pre/post-release differences to be conservative estimates of the extent that incarceration reduces mortality risk, so would not change the paper's main take-aways.

Taken together, this indicates both that there is not significant persistent unobserved heterogeneity in mortality risk during our study period,[31] and that even if there were, this would not substantially influence our estimates. Further, the quantitatively and qualitatively similar estimates from both the proportional hazards models and the more reduced-form models in the previous subsections are additional evidence that the results are not driven by the imposition or lack thereof of the proportional hazards assumption.

2.3 Difference-in-differences estimates of the effect of incarceration

In the previous two sections, we compared defendants who are initially incarcerated to those who are not initially incarcerated. The initially-incarcerated defendants have substantially lower mortality before release, but the mortality rates for the two groups converge precisely at the time of release. In this section, we use the sharp change in incarceration status to create a difference-in-differences-style estimate of the effect of incarceration on pre-release mortality. This estimator can account for unobservable differences in mortality risk across incarcerated and non-incarcerated defendants under some additional assumptions that are plausibly satisfied or close to satisfied in this context.

The standard DiD approach studies changes in outcomes at the time that one group receives the treatment. In our setting, however, this would mean comparing changes in outcomes before and after sentencing for those sentenced to incarceration relative to those not sentenced to incarceration. This presents a challenge when studying mortality, since

[30] Results are similar with time-varying unobserved heterogeneity.

[31] One explanation for this result is that while individuals in our sample lead relatively risky lives, the extent of *persistent* unobserved risk is small relative to baseline risk or observable determinants of risk.

all defendants are alive at the time of sentencing. As a result, the standard DID estimator collapses to the post-sentencing difference in outcomes between the groups, which is the comparison we already use in Section 2.2.

We instead conduct a *prospective DiD* around the *removal* of treatment, i.e., exit from incarceration.[32] Using only periods after sentencing, we define the prospective DiD as the change in mortality risk between incarceration and the post-release period for those sentenced to incarceration relative to the changes over time in mortality risk observed in the comparable non-incarcerated group. Note that this closely corresponds to two parameters that we have already estimated—β_1 and β_2 in Equation 2. The prospective DID is equal to $\beta_1 - \beta_2$, i.e., how the difference in mortality risk between the incarcerated and non-incarcerated changes around the time of release.[33] Intuitively, the similar non-incarcerated individuals provide the trend in mortality that we would expect the incarcerated to experience over that period, such as might happen due to aging. By looking at the change in mortality risk immediately at the time of release, we attempt to recover the effect of incarceration on mortality risk due to being incarcerated.

To implement our estimator, we construct a quarter-level panel dataset that begins after sentencing, continues for seven years, and includes all quarters in which the individual has not previously died. The prospective DiD is estimated using:

$$died_{iact} = \alpha Sentenced_{ic} + \beta Sentenced_{ic} \times PreRelease_{ict} + X_{iact}\phi + \eta_t + \gamma_a + \psi_c + \varepsilon_{iact} \quad (3)$$

where the variables are defined as in Equation 2, $Sentenced_{ic}$ is a binary indicator for whether the individual was sentenced to incarceration, and $Sentenced_{ic} \times PreRelease_{ict}$ is an indicator for whether they are sentenced to be incarcerated during quarter t. Standard errors are two-way clustered at the defendant and court-month levels. β equals the change in mortality risk

[32] Other studies, such as Banerjee and Duflo (2014) and Alexander and Schnell (2019), have used a prospective DiD. We clarify the assumptions needed for identification in Appendix D.

[33] A standard DiD estimator would be equal to $\beta_2 - \beta_1$ because the treatment is in the post period. It is the reverse here because the treatment is in the pre-period.

between the period of incarceration and post-release period for the incarcerated relative to the change in mortality risk over time observed among the non-incarcerated, as accounted for with fixed effects for the number of quarters since charges were filed.

Appendix D formally describes this design using a potential outcomes framework. We show this design will identify the ATT with two additional assumptions beyond standard DiD parallel trends. The first is that there must not be post-release effects on mortality. However, if there are post-release effects but their sign is the same as β^{ATT}, then $\widehat{\beta}$ will be a conservative estimate of β^{ATT}; this follows from $\widehat{\beta}$ being equal to $\beta_1 - \beta_2$. Section 2.2.3 shows that the post-release effects are zero or weakly negative (i.e., past experience of incarceration reduces mortality risk). Since our estimate of $\widehat{\beta}$ is also negative, this force would cause our DiD estimate to be at worst conservative.

The second assumption is that conditional on controls, there should not be significant persistent, individual-specific unobserved heterogeneity in mortality risk. Although Section 2.2.3 presents evidence for negligible unobserved heterogeneity in this setting, Section C1 implements simulations to examine the extent to which unmeasured unobserved heterogeneity may bias our difference-in-differences estimates. Under time-invariant unobserved heterogeneity, the bias in the difference-in-differences estimator is quite small. This is because the unobserved heterogeneity biases the post-release and pre-release differences in mortality risk between the incarcerated/non-incarcerated upwards by similar amounts, so they cancel each other out. The extent to which they cancel each other out depends on the form of the unobserved heterogeneity, but in all simulations, the DiD bias is relatively small.

The third assumption is the standard assumption of parallel trends. Intuitively, this design is comparing the change in the mortality of the incarcerated between the period before release and that of release as compared to the change in mortality over the same number of periods since sentencing among the non-incarcerated. In other words, the parallel trends assumption in this case is that the age-mortality trajectory is the same among the incarcerated and non-incarcerated. Figure 3 shows that for all outcomes, the relative difference

in mortality between the incarcerated and non-incarcerated is flat in the years before and after release, consistent with similar age-mortality trajectories. But more importantly, given the large and nearly instantaneous change in mortality risk at the time of release seen in the period-by-period comparison, there would have to be other time-varying factors of an implausibly large magnitude and exact timing for our results to be explained by a failure in this parallel trends assumption.

Column (1) of Table 4 estimates the DiD specification from Equation 3 on mortality risk, measured in deaths per hundred thousand individuals annually. The coefficient on "Incarcerated in quarter" (corresponding to β) measures the direct effect of incarceration. We estimate that incarceration reduces mortality risk by 368 deaths per hundred thousand ($p < 0.001$).[34] This is quite similar to our earlier estimate since unobservable differences in mortality risk between the incarcerated and non-incarcerated appear to be similar with our detailed set of controls.

Appendix E shows the results are also very similar when excluding the non-incarcerated group. In this specification, the estimated treatment effects are primarily identified from changes in the mortality risk of incarcerated individuals at the time of release rather than factors like comparisons to the initially-not-incarcerated individuals, which we used in our earlier analyses. The similar results from two different identification approaches supports the robustness of our findings. For the robustness checks and heterogeneity analysis in the remainder of the paper, we will focus on the DiD specification—however, all of the results go through when using the specification in Equation 2.

[34] With multiple time periods, two-way FE estimators return a weighted average of group-period treatment effects with no guarantee that these weights are all positive (Goodman-Bacon, 2018; de Chaisemartin and D'Haultfœuille, 2020). Thus, under heterogeneous effects, β might be misleading or even wrong-signed. However, these weights are identified in a version of Equation 3 that excludes controls and conditions on period-of-release × court-month FEs as opposed to court-month FEs (this makes the design sharp in the sense that all members of each group change treatment status at the same time). We conduct this analysis, and find that the sum of the negative weights is only 0.03%. This means that any potential bias from negative weights is extremely small.

3 Additional analysis

3.1 Heterogeneous effects

Our data run from the early 1990s through 2016, and the effect of incarceration may have changed over this period. Panel A of Figure 4 re-estimates Equation 3 for each year of sentencing and plots the estimated β. The direct effect of incarceration has tripled over our study period. The remaining panels show that this change is primarily driven by overdoses, although there is also a moderate increase in the effect of incarceration on homicides. The mortality rate within prison in Ohio barely changes over this time period—from 256 deaths per 100,000 prisoners annually in 2001 to 255 in 2016—indicating that these changes are explained by heightened mortality outside of correctional institutions rather than changes within them (Bureau of Justice Statistics, 2020).

Our long study period permits us to look at the effect of incarceration on mortality in periods where the population mortality rates in Ohio are similar to the rest of the US, and hence more generalizable, as well as how the effect of incarceration changes in response to the opioid epidemic in Ohio. We estimate the difference-in-differences model with the sample split based on whether the sentencing date was prior to 2010 or post-2010, as the mortality rates in Ohio begin to increase in 2010 as compared to the US average. Columns (6) and (7) of Table 4 present the results. Column (6) shows the direct effect of incarceration on mortality prior to 2010 ($\beta = -315$, $p < 0.001$) is about 40% smaller than post-2010 (column (7), $\beta = -547$, $p < 0.001$). The estimates prior to 2010 are also similar to the full sample results with respect to specific causes of death (Table A1): incarceration reduces mortality from all causes of death, and the largest reductions still come from homicides and overdoses.

In addition to varying effects of incarceration over time, there may be heterogeneous effects related to defendant characteristics. Table 5 finds that the direct effect of incarceration is 49% larger for individuals above the age of 30, consistent with their higher baseline mortality risk. Reductions are significantly larger for white and male defendants. White males

above the age of 30 are exactly the population believed to be affected by "deaths of despair," consistent with the rising importance of overdose deaths in our sample period. We do not observe heterogeneity related to whether the individual was charged with a drug crime, or the poverty level of their neighborhood. On the other hand, we do observe that the direct effect of incarceration on mortality is slightly smaller for sentences of less than a year. Since sentences less than one year are typically served in jail—rather than prison—this may reflect differences in provision of services across jails and prisons or differences in baseline mortality risks of their populations.[35,36]

Individuals involved in the criminal justice system overlap substantially with the population experiencing rising "deaths of despair." Between 2001 and 2017, 58% of overdose-related deaths in the three study counties are of individuals who previously appeared in court.[37] The rise in deaths of despair, particularly those related to the opioid epidemic, appears to have substantially increased the protective effects of incarceration. To estimate the extent to which incarceration has affected the rise in deaths of despair, we calculate the fraction of the Ohio population incarcerated in each year between 2006 and 2016 within racial and ten year age bins using the American Community Survey. We then multiply those figures by the treatment effect of incarceration on mortality within those bins for the estimated reduction in mortality due to incarceration in that year. Over this period, we find that overdose deaths for men would have been 7.2% higher in the absence of incarceration.

3.2 Robustness checks

This section examines the robustness of our results. One concern is that Ohio death certificates cover only deaths in Ohio, but incarceration could affect the likelihood of residency in

[35] The heterogeneous effects by defendant characteristics are qualitatively the same prior to 2010 as compared to the full study period.

[36] Unlike the direct effect of incarceration, we do not observe heterogeneity in post-release effects that is related to sentence length. This contrasts somewhat with Hjalmarsson and Lindquist (2020), who find that longer sentences in Swedish prisons reduces post-release mortality risk. This difference may be due to the better provision of services in Swedish prisons relative to the US.

[37] Other states are similar: State of Connecticut (2019) finds that 49.7% of Connecticut overdose deaths are former prisoners.

Ohio. Column (3) of Table 4 reruns the main specification, measuring death with appearances in either the Ohio Death Certificates or the SSA Death Master File. The sample size is smaller because the Death Master File only covers deaths through 2010, but the results are similar. Column (4) uses the same sample of years as column (3), but measures deaths using only the Ohio records for a more direct comparison; the estimates in columns (3) and (4) are statistically indistinguishable.[38]

Another concern is that the main estimates implicitly assume that those who are not initially sentenced to incarceration were not incarcerated on later charges and that after release, individuals are not reincarcerated. Both would cause our estimates to understate the direct effect of incarceration. In Appendix F, we instrument for whether the defendant is incarcerated in a quarter with their sentenced incarceration status in our DiD framework. The estimates of β are approximately twenty percent larger than our main results, but the conclusions are qualitatively the same.

A final interpretational issue is that many non-incarcerated defendants are placed on probation, and this heightened monitoring may reduce mortality risk. In this case, our estimates are still policy-relevant as to the effect of incarceration relative to counterfactual punishments, but would underestimate the effect relative to no punishment. Column (5) of Table 4 re-runs the main difference-in-differences specification, but excludes non-incarcerated defendants sentenced to probation. Estimates are similar, consistent with probation not having a major effect on mortality risk.

3.3 Comparisons with related work

There is an extensive sociology and public health literature on mortality and incarceration in the United States, with some of the most prominent papers cited over a thousand times (e.g. Binswanger et al. (2007)). This literature has primarily estimated the effects of incarceration by comparing mortality among the incarcerated or formerly incarcerated to the general

[38] The estimate of β in column (4) is slightly smaller than in column (1) of Table 2 because the treatment effect of incarceration increases over time (Section 3.1) and column (4) excludes later years.

population while adjusting for demographics such as age, race, and gender. However, this strategy does not account for the many other characteristics on which the incarcerated differ from the general population and so is unlikely to produce causal effects. This issue is well-known in the literature, and two major recent review papers on incarceration and mortality have called for alternative empirical approaches (Massoglia and Pridemore, 2015; Wildeman and Wang, 2017). This section briefly summarizes the existing work, and then discusses how and why our own estimates contrast with them.

Most recent work on the direct effect of incarceration have found that there are protective effects of incarceration for black inmates (Patterson, 2010; Rosen et al., 2011; Spaulding et al., 2011; Wildeman et al., 2016), with Wildeman et al. (2016) finding a decline of 294 deaths per 100,000. However, there are mixed results for white inmates. Rosen et al. (2011), Patterson (2010), and Spaulding et al. (2011) find that white inmates have higher mortality than the white general population (e.g. 28% higher in Spaulding et al. (2011)), while Wildeman et al. (2016) finds a modest decline of 27 deaths per 100,000.

Our estimates of the direct effect of incarceration are similar to existing work for black defendants (we find a decline of 314 deaths per 100,000 annually), but differ substantially for white defendants (decline of 450 deaths per 100,000). This difference in estimates for whites is important given that the total populations of white and black inmates in the United States are roughly the same (Wildeman et al., 2016).The likely reason that our results differ from previous work is that our method provides a more appropriate control group. While previous work compares mortality for the incarcerated to the general population conditional on some basic demographic controls (typically age, race, and gender), we compare them to defendants who are charged but not incarcerated. Since the general population likely has lower unobservable mortality risk than criminal defendants, this means that previous estimates would be biased towards finding that incarceration increases mortality relative to our estimates. In Appendix B, we provide support for this hypothesis using the National Longitudinal Survey of Youth. We compare pre-existing mortality risk factors, such as general health and use

of hard drugs, between those who will be incarcerated, those who will be charged but not incarcerated, and those without future criminal justice involvement. The incarcerated have similar mortality risks to those who are charged but not incarcerated. However, their risk is substantially higher than those without criminal justice system involvement, consistent with other criminal defendants being a more appropriate comparison group.

The NLSY analysis also helps explain the discrepancies between our estimates and the previous literature for white inmates. When we rerun the analysis split by race, the difference in underlying mortality risk factors between incarcerated and non-criminal justice involved respondents is consistently much smaller among black respondents than white respondents. We take this as one possible reason why previous work has found similar results for black defendants, but not for white defendants.

The results in this paper also conflict with the recent literature on post-release effects. A recent review paper nicely summarizes the academic consensus: "[a]lthough current incarceration has mixed effects on prisoners' health, incarceration has a clearly deleterious impact on health" (Wildeman and Wang, 2017). A number of papers argue that the post-release effect of incarceration outweighs the protective effect of incarceration, meaning that the aggregate effect of incarceration is to increase mortality (e.g. Spaulding et al. (2011); Daza et al. (2020)). The difference between our estimate of the post-release effect and the consensus in the literature also likely stems from our control group of non-incarcerated, criminally-charged individuals rather than the general population. Using this more comparable control group, we find instead that incarceration has no detectable effect on post-release mortality for at least 15 years, and thus likely increases overall longevity.

4 Conclusion

This paper documents that incarceration leads to sizable reductions in mortality during the period of confinement, with most of the decline coming from homicides and overdoses. This

protective effect has grown stronger in recent years. We also find that past experience of incarceration does not increase mortality, meaning that the aggregate effect of incarceration is to reduce mortality. Taking our year-by-year estimates of the effect of incarceration on mortality, we estimate that 1,869 deaths were averted in Ohio over the period of our study as a result of incarceration. Given that Ohio has similar rates of incarceration, mortality while incarcerated, and age-adjusted mortality to the rest of the United States, a simple extrapolation indicates over the study period, incarceration averted an annual average of approximately 2,000 deaths nationally.[39]

These results are a function of the high mortality risk that criminal justice-involved individuals face when not incarcerated and demonstrate how short-term separation of high-risk individuals from unsafe environments can substantially increase longevity. This has several implications. First, while incarceration is clearly not a desirable policy for mortality reduction due to its many other costs, policymakers might consider adopting aspects of the mortality-reducing aspects of incarceration into other policies. For example, increasing access to safe, stable living facilities for individuals released from prison may be particularly effective. The reductions in deaths from natural causes during incarceration also suggests potential gains from increasing access to healthcare for this population, such as enrolling inmates in Medicaid upon release. Second, the results emphasize the high-risk environment that incarcerated individuals face after release, and suggest there might be a high social value from other policies aimed at increasing community safety in this population. Third and finally, our results indicate that reforms to reduce incarceration may benefit from complementary policies to improve the health of at-risk individuals.

[39] Our study does not cover periods of major transmissible disease outbreaks including the peak of the AIDS epidemic or COVID-19, when these conclusions may differ. Also our estimates are of the effect of incarceration on mortality for individuals, and do not account for any potential community-level effects.

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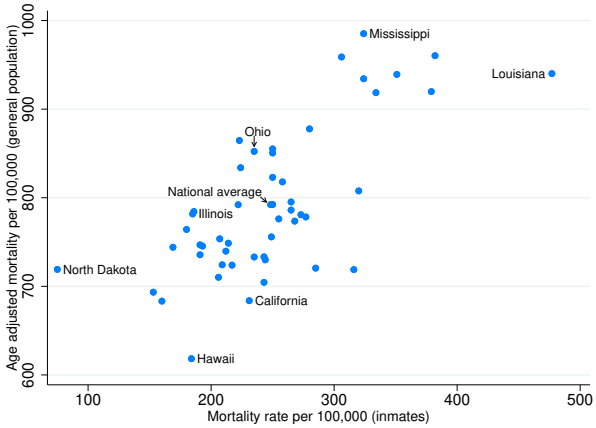
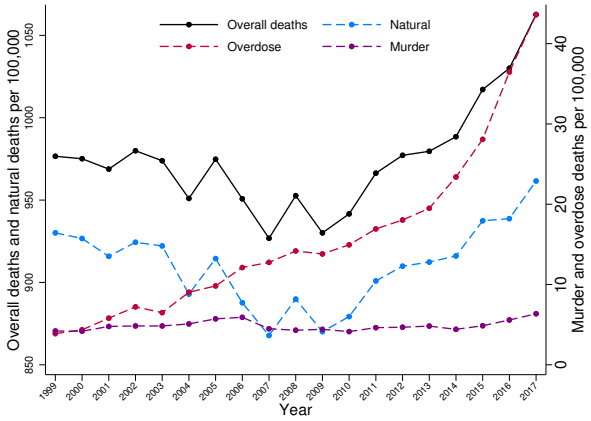
Figures

Figure 1: Descriptive statistics on Ohio

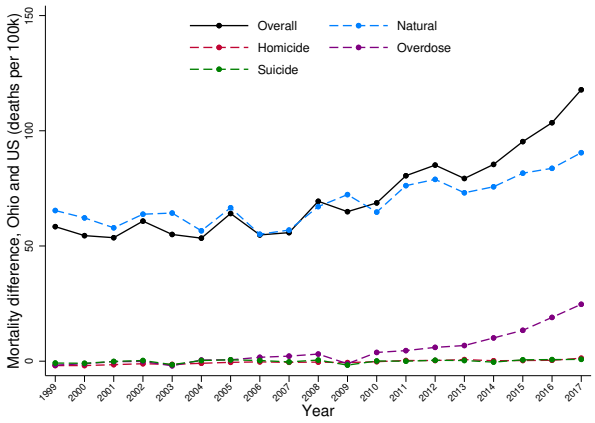
(a) State-level in-prison mortality



(b) Trends in causes of death over time in Ohio

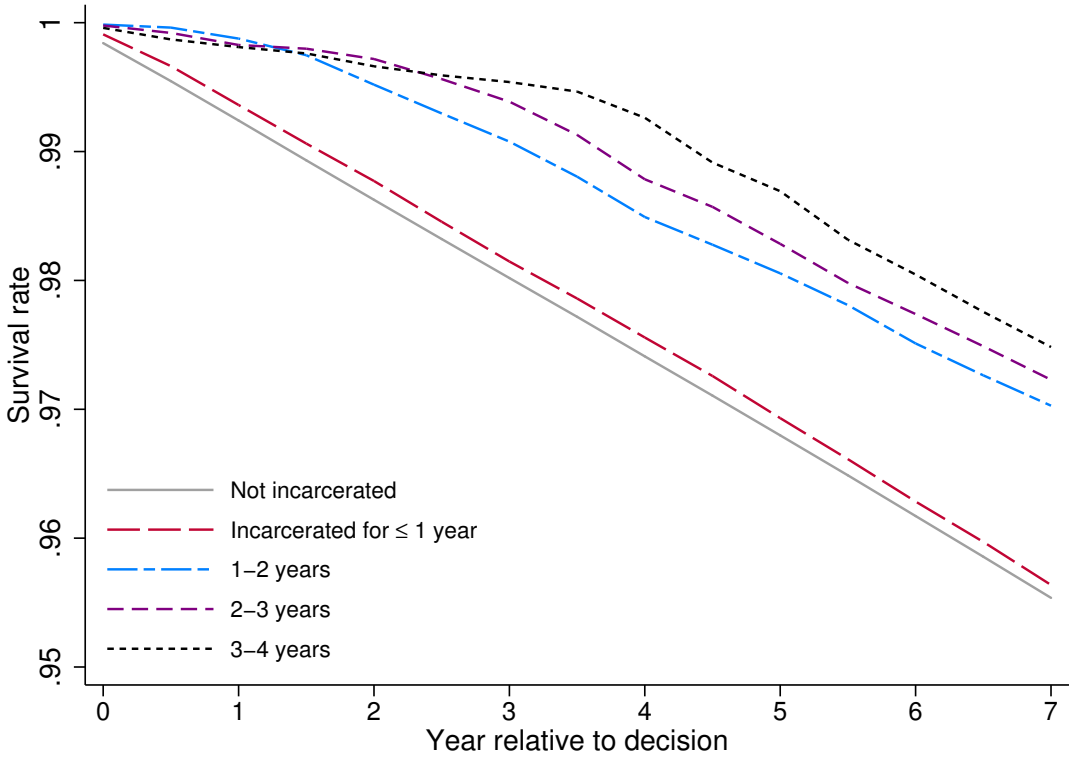


(c) Mortality differences, Ohio relative to US



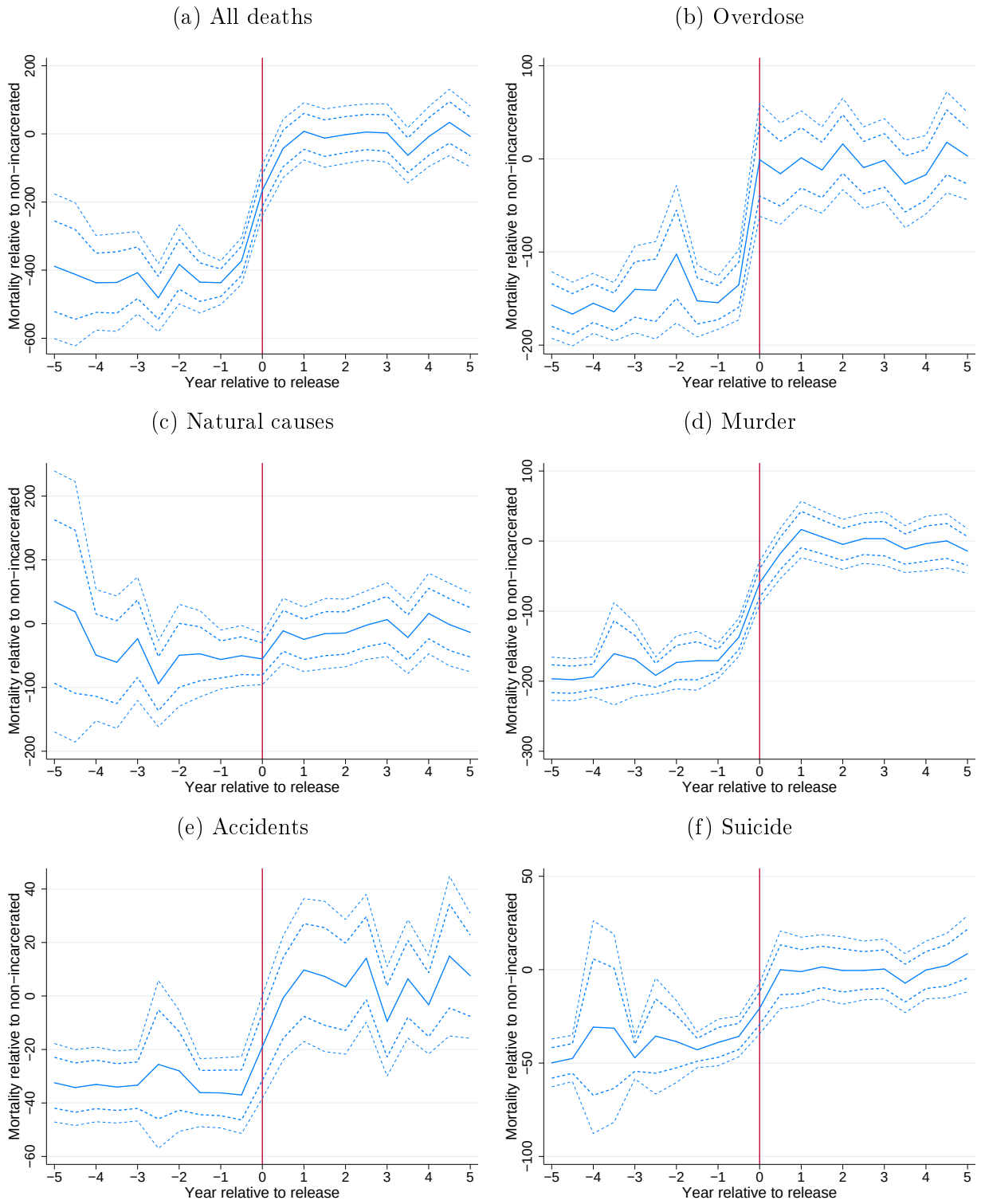
Panel (a) displays a scatterplot of state-level age-adjusted mortality rate and within-prison mortality rate. Source: BJS, Deaths in Custody Reporting Program, 2001 and 2005-2014; National Prisoner Statistics, 2001 and 2005-2014; and Federal Bureau of Prisons, 2001 and 2005-2014. Panel (b) displays overall and cause-specific Ohio mortality rates by year. Source: Ohio Department of Health death records. Panel (c) plots the difference between the age-adjusted mortality rate in Ohio and the rest of the United States by cause of death. Source: CDC WONDER

Figure 2: Survival rates by sentence length



This figure displays Kaplan-Meier estimates of the survival rate split by sentence length groups, in each half-year relative to decision. The data consist of seven years following sentencing for each defendant.

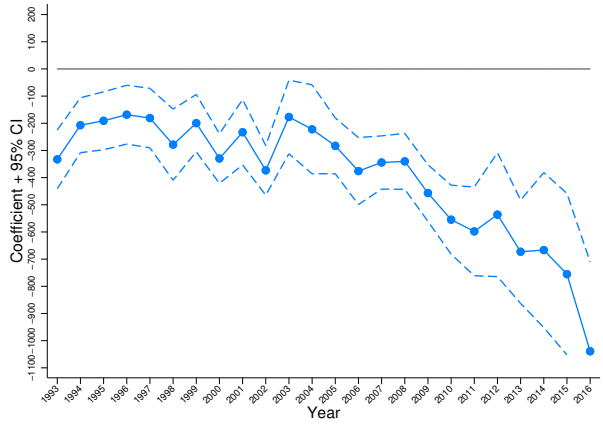
Figure 3: Period-by-period estimates of differential mortality risk around exit from incarceration



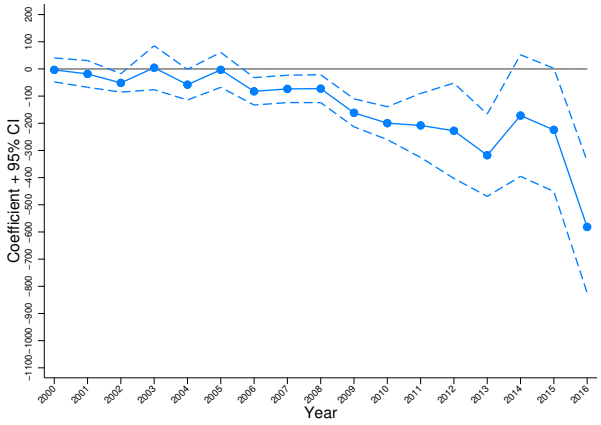
This figure displays regression estimates of the difference in annual mortality per 100,000 between the initially non-incarcerated and the initially incarcerated in each half-year relative to release. Overall mortality in Panel (a), cause of death-specific results in Panels (b)-(f). Regressions estimated according to Equation 1, and include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The data consist of seven years following sentencing for each defendant. Dotted lines represent 95% confidence intervals two-way clustered at the individual and court-month level, and thin dashed lines represent the sup-t confidence band (Montiel Olea and Plagborg-Møller, 2019).

Figure 4: Direct effect of incarceration on mortality by year of sentencing

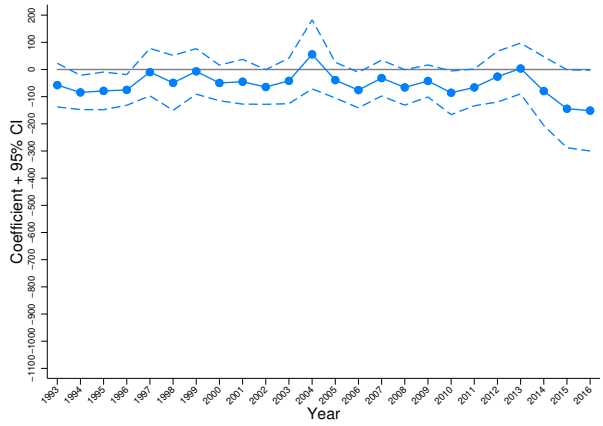
(a) Overall deaths



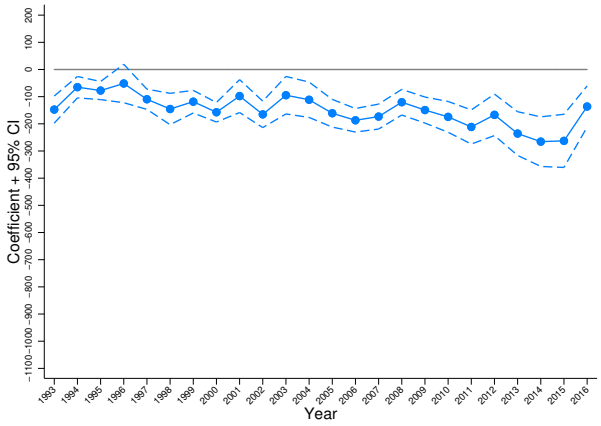
(b) Overdose



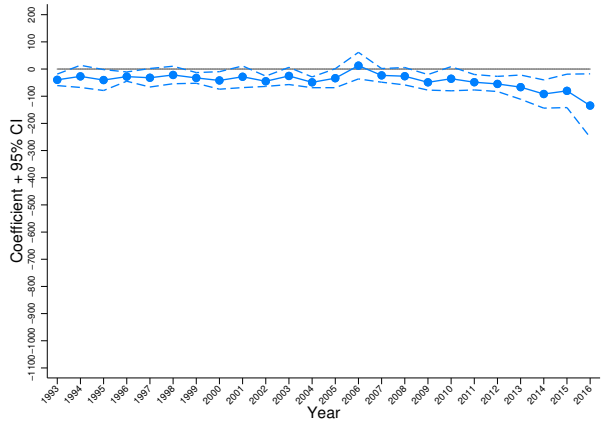
(c) Natural



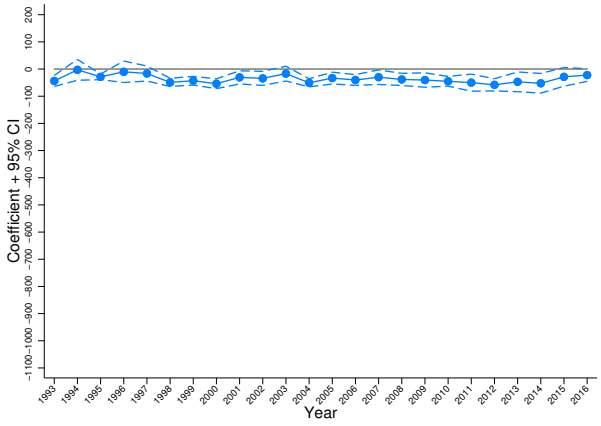
(d) Murder



(e) Accidents



(f) Suicide



This figure displays year of sentencing-specific difference-in-differences regression estimates of the effect of incarceration on the outcome in the panel header. Mortality is measured in terms of deaths per 100,000 person-years. Regressions estimated according to Equation 3, and include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The data consist of seven years following sentencing for each defendant. Dotted lines represent 95% confidence intervals two-way clustered at the individual and court-month level.

Tables

Table 1: Relationship between incarceration and survival within varying years

	Survival (per 100,000)		
	3 years	5 years	7 years
	(1)	(2)	(3)
Years incarcerated	399.7***	409.1***	414.5***
	(42.20)	(37.70)	(36.48)
Survival mean	98239.2	97197.3	96231.8
Observations	1,591,821	1,601,420	1,605,182

This table reports the relationship between years of sentence and the probability of survival within 3, 5, and 7 years of court decision. Regressions include court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The sample is restricted to individuals with sentence length less than or equal to the number of years of the outcome. Standard errors in parentheses and clustered at the individual and court-month level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 2: Effect of incarceration on annual mortality per 100,000

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Overall		*Cause of death*					
			Overdose	Murder	Natural	Accident	Suicide	Other external
Incarcerated × pre-release	-357.33***	-355.88***	-100.94***	-144.22***	-51.98***	-32.77***	-35.10***	-5.87***
	(19.292)	(14.881)	(12.304)	(7.976)	(11.763)	(5.203)	(3.769)	(2.180)
Incarcerated × post-release	11.14	55.59***	-0.79	1.19	2.87	5.70*	0.87	0.35
	(9.808)	(11.476)	(5.119)	(3.470)	(7.440)	(2.908)	(1.766)	(0.958)
Controls	Yes	No	Yes	Yes	Yes	Yes	Yes	Yes
DiD estimate	-368.47***	-411.47***	-100.15***	-145.40***	-54.85***	-38.47***	-35.98***	-6.22***
Post-release mean (1 year)	587.4	587.4	189.2	146.9	185.6	52	33.1	8.6
Observations	41,211,056	41,211,056	37,219,360	41,211,056	41,211,056	4,1211,056	41,211,056	41,211,056
			Cause of natural death					
		Heart	Cancer	Infection	Respiratory	Liver/alcohol	Circulatory	Other
Incarcerated × pre-release		-18.51***	5.90	-6.64	-2.92	-3.06	-2.34	-24.42***
		(5.836)	(6.009)	(4.064)	(3.086)	(3.007)	(2.311)	(5.374)
Incarcerated × post-release		-2.74	5.76	1.73	-3.22**	-0.19	-1.70	3.23
		(3.340)	(3.865)	(2.904)	(1.589)	(2.071)	(1.940)	(3.143)
Controls		Yes	Yes	Yes	Yes	Yes	Yes	Yes
DiD estimate		-15.77***	0.13	-8.36***	0.30	-2.87	-0.63	-27.64***
Post-release mean (1 year)		42.5	36.5	17.2	11.2	8.6	12.9	56.7
Observations	92,907,982	41,211,056	41,211,056	41,211,056	41,211,056	41,211,056	41,211,056	41,211,056

This table reports difference-in-differences estimates of the effect of incarceration on annual mortality per 100,000. Regressions estimated according to Equation 3, and include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The post-release mean is the death rate for the first year after release. Standard errors in parentheses and clustered at the individual and court-month level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 3: Proportional hazard models of the effect of incarceration on days to death

	Weibull-Gamma	Weibull-InvG.	Weibull		Cox
	(1)	(2)	(3)	(4)	(5)
Incarcerated × Prerelease	-1.06***	-1.06***	-1.06***	-1.00***	-1.08***
	(0.056)	(0.056)	(0.073)	(0.073)	(0.073)
Incarcerated × Postrelease	0.03**	0.03**	0.03	0.03	0.03
	(0.013)	(0.013)	(0.024)	(0.021)	(0.024)
θ	$< 10^{-5}$	$< 10^{-5}$			
$\theta = 0$ p-value	1	1			
Court-year FE	No	No	No	Yes	No
Offense FE	No	No	No	Yes	No
Observations	1,840,879	1,840,879	1,840,879	1,840,879	1,840,879

This table reports coefficients from parametric and semiparametric hazard models. All models include the covariates of incarcerated pre-release, incarcerated post-release, age, race, poverty decile, female, previous charges and incarcerations. Columns (1-4) assume the hazard rate follows the Weibull distribution, while Column (5) estimates a Cox model and therefore does not restrict the hazard rate. Column (4) includes court-year and offense fixed effects. Column (1) assumes individual-specific frailty follows the Gamma model, while column (2) assumes frailty follows the Inverse Gaussian distribution. θ is the variance of unobserved heterogeneity or frailty. Standard errors in parentheses. Standard errors in columns (3-5) clustered at the level of the individual. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 4: Difference-in-differences estimates of the effect of incarceration on annual mortality per 100,000

	Ohio Death Records		ODR or DMF	ODR	Excluding probation	Results by period	
						Pre 2010	Post 2010
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Ever incarcerated (α)	11.14	73.44***	-0.830	2.855	4.989	3.106	42.12
	(11.39)	(11.02)	(9.808)	(10.29)	(10.77)	(9.952)	(25.62)
Incarcarated in quarter (β)	-368.5***	-389.0***	-351.4***	-333.3***	-392.0***	-315.0***	-546.8***
	(17.93)	(17.27)	(18.06)	(17.53)	(18.75)	(17.25)	(47.61)
Post-release mean (1 year)	587.4	587.4	534.3	502.6	587.4	486	911.8
Controls	Yes	No	Yes	Yes	Yes	Yes	Yes
Observations	41,211,056	41,211,056	31,628,445	31,628,445	32,468,671	30,596,576	10,614,480

This table reports difference-in-differences estimates of the effect of incarceration on annual mortality per 100,000 across varying datasets and sample restrictions. Our main results use the Ohio Death Records (ODR), which provide comprehensive coverage of all deaths in Ohio until 2018 and include cause of death information. As robustness we also provide results using the Social Security Death Master File (DMF), which covers all deaths in the United States for Social Security Number holders to 2010, and does not include cause of death information. Pre-2010 includes the sample who were scheduled for release prior to 2010. Post-2010 includes the sample were scheduled for release in 2010 or later. Regressions estimated according to Equation 3, and include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. Standard errors in parentheses and clustered at the individual and court-month level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 5: Heterogeneity by personal characteristics

	(1)	(2)	(3)	(4)	(5)	(6)
Incarcarated in quarter (β)	-302.9***	-370.0***	-450.1***	-375.8***	-397.3***	-384.9***
	(19.792)	(20.282)	(32.614)	(28.450)	(18.994)	(24.188)
Age>30 × Incar in quarter	-147.2***					
	(30.520)					
Drug charge × Incar in quarter		2.5				
		(28.415)				
Black × Incar in quarter			136.3***			
			(36.241)			
SES ≤ 25th perc. × Incar in quarter				-4.1		
				(31.777)		
Female × Incar in quarter					199.5***	
					(48.360)	
Less than 1 year × Incar in quarter						64.8*
						(33.269)
Left-out group post-release mean (1 year)	447.4	571.2	732	628.3	604.3	499.6
Interaction group post-release mean (1 year)	724.9	624.9	483.8	562.7	518.8	611.6
Observations	41,211,056	41,211,056	41,211,056	41,211,056	41,211,056	41,211,056

This table reports heterogeneous difference-in-differences estimates of the effect of incarceration on annual mortality per 100,000. Regressions include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, sentence less than a year, previous charges and incarcerations, and offense type fixed effects. Post-release means are reported by group for the first year after release. For example, in column (1) the left-out group is those younger than 30, and the interaction group is those older than 30. Standard errors in parentheses and clustered at the individual and court-month level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Appendix for *The Effect of Incarceration on Mortality*

A Comparing sentences and time served

To avoid issues arising from potentially endogenous releases from incarceration because of impending death, we code whether an individual is incarcerated in a given quarter based on the sentence given at the time of sentencing in the court records. To check how accurately these sentences reflect time served, we match court records to Ohio Department of Rehabilitation and Corrections prison records, which contain the exact date of release. The sentence length is similar to time actually spent incarcerated—72.3% are released within two quarters of their sentenced date of release. Even this likely overstates measurement error since it only includes prison sentences. Most sentences are served in jail, not prison, and are for less than a year, so measurement error will be minimal given our aggregation to the quarter level. The measurement error of the prison stints is also roughly symmetrical around zero. This means that our estimates would be biased towards zero, and therefore conservative.

B Signing unobservable differences in mortality risk

Section 2.2.2 and Section 2.2.3 use our data to place conservative and informative bounds on the pre-release and post-release effects of incarceration. For this analysis, they rely on the assumption that the unobserved characteristics of the incarcerated predispose them to higher mortality risk than the comparison group of non-incarcerated defendants. While this is relatively intuitive, this appendix section provides direct empirical evidence for this claim.

For this analysis, we use the 1997 wave of the National Longitudinal Survey of Youth (NLSY). This is a panel data set that contains two sets of variables useful for our analysis.

First, for each year of the data, it records whether the respondent was either incarcerated or charged with a crime but not incarcerated. As a result, we can construct sets of individuals within the NLSY data following the same sample criteria as the incarcerated and non-incarcerated in the main Ohio analysis data. Second, the NLSY contains information on many individual behaviors and characteristics related to mortality risk such as use of hard drugs or belonging to a gang. These factors are clearly related to mortality risk, but are not observable in our main analysis data. Putting these variables together, we can use the NLSY to test for differences between the incarcerated/non-incarcerated on the types of mortality risk factors that are unobservable in our main analysis data.

To be more specific, our analysis takes the sample of individuals who will be criminal defendants in year $(t+1)$ in the NLSY and regresses characteristics in year t on whether the individual is incarcerated in year $(t+1)$. We look at factors including hard drug use (related to overdose deaths), measures of depression (related to suicide), self-rated health (related to natural causes of death), binge-drinking (related to drunk driving and death due to accident), and belonging to a gang (related to homicide). We test the relative mortality risk of the incarcerated for these factors. For all the variables aside from general health, positive coefficients indicate that those who end up incarcerated indeed have higher "unobservable" mortality risk for that factor than the charged but not incarcerated; for general health, higher values indicate better health, so this is indicated by a negative coefficient.

Table A3 runs this test. Panel A includes controls for age-race-gender, while Panel B includes the same types of controls as in our data (income decile, criminal history, type of crime charged with). The incarcerated are slightly more likely to be smokers when we do not control for characteristics observed in our main analysis data (Panel A). When we include those controls, this difference is no longer statistically significant (Panel B), i.e., controlling for observables marginally reduces the difference in "unobservable" characteristics and potential omitted variable bias. In general, the incarcerated and non-incarcerated are quite similar in terms of these "unobservable" mortality risks, although the incarcerated

typically have slightly higher risk profiles.

While it is not possible to test all factors that are related to mortality risk and are unobservable in our data, the ones investigated here are highly related to all of the major categories of death for the criminally involved sample. As a result, we view this as fairly compelling evidence that mortality risks related to unobservable characteristics are either higher for the incarcerated or similar between the two groups. This implies that the estimated differences in pre-release and post-release mortality will be conservative bounds on the extent to which incarceration causally reduces pre-release and post-release mortality.

The NLSY data is also informative as to why the comparison group that we use in the paper may provide more credible estimates than existing work, which primarily compares the incarcerated to the general population. Table A4 runs a similar regression, but using the full NLSY sample, including those who were not charged with a criminal offense. We then regress the aforementioned mortality risk factors on dummy variables for being incarcerated in the next year or charged but not incarcerated in the next year. The incarcerated and charged/non-incarcerated have significantly higher risk profiles than the general population for nearly every category.[1] This suggests that papers relying on comparisons with the general population will overestimate the extent to which incarceration increases mortality risk. In contrast, our approach, using a more comparable control population and a research design to estimate the direct effect, will likely produce more accurate estimates.

The NLSY data also helps explain why our estimates for white defendants are more discrepant from the existing literature than estimates for black defendants. In Table A5, we run the same analysis as in Table A4, but split the sample into white and black defendants. This analysis finds that for determinants of mortality risk that are unobservable in our sample, the general population is more comparable to the incarcerated defendant population for blacks than for whites. For example, the average health score (self-reported, runs from

[1]The estimated differences between the incarcerated and charged but not incarcerated differ slightly from those in Table A3. This is because the sample in Table A3 is restricted to years between 2003 and 2016 since the full set of control variables are only available for those years.

1 to 5) is 0.40 points lower for incarcerated relative to non-criminal justice involved white respondents. However, the difference is statistically indistinguishable among black respondents.[2] A similar pattern presents itself across most of the other measures. We take this as one possible reason why previous work has found similar results as ours for black defendants, but not for white defendants.

C Unobserved heterogeneity

In this section, we discuss the effects of unobserved heterogeneity through the lens of a simple parametric model and use the results to develop tests for unobserved heterogeneity.

We first discuss how in a proportional hazards model, time-invariant, gamma-distributed unobserved heterogeneity: (1) causes the hazard rate to decrease more quickly; and (2) attenuates the effects of exogenous variables over time. These results are standard and similar results can be found in Lancaster (1990) and Cameron and Trivedi (2005). The second result implies that the difference in mortality rates between those who are incarcerated and those who are not incarcerated will become increasingly understated over time during incarceration. This is because the higher mortality risk in the non-incarcerated population causes people with high time-invariant unobserved heterogeneity to die and consequently exit the sample earlier. The compositional differences result in higher unobserved mortality risk in the incarcerated population after release, leading to inflated estimated mortality of the post-incarcerated group relative to the non-incarcerated group.

More formally, let $\lambda^*(t|\mu)$ be the hazard rate for the population without unobserved heterogeneity. Assume it takes a proportional hazards form $\lambda^*(t|\mu) = \mu\lambda_0(t)$, with $\mu = \exp\{x'\beta\}$ being differences arising from observed covariates x and $\lambda_0(t)$ is the baseline hazard rate. We compare this hazard rate to a population hazard rate with unobserved heterogeneity. For individual i, the hazard rate takes the form $\lambda_i(t|\mu) = \mu\lambda_0(t)\theta_i$, where θ_i is time-invariant

[2]Note that though the coefficient on charged is statistically significant in column (1), we cannot reject equality between that coefficient and the coefficient for incarcerated.

individual-specific mortality risk drawn from a distribution with mean 1 and variance σ^2. We write the corresponding conditional population hazard as $\lambda(t|\mu)$. We further denote $I_0(t)$ as the cumulative baseline hazard and $S(t|\mu)$ as the survivor function.

The population hazard rate with unobserved heterogeneity is lower than the population hazard rate without unobserved heterogeneity. Our assumption of a proportional hazards model with multiplicative frailty allows us to write the hazard rate in the form

$$\lambda(t|\mu) = \lambda_0(t)\mu E[\theta|T \geq t]$$

With θ following the gamma distribution, we can further write the population hazard rate as

$$\begin{aligned}\lambda(t|\mu) &= \lambda_0(t)\mu/[1+\sigma^2\mu I_0(t)] \\ &= \lambda_0(t)\mu[S(t|\mu)]^{\sigma^2} \\ &= \lambda^*(t|\mu)[S(t|\mu)]^{\sigma^2}\end{aligned}$$

Since the survivor function is non-increasing, $\partial[S(t|\mu)]/\partial t \leq 0$ and hence, the population hazard rate decreases more quickly with unobserved heterogeneity. Increasing unobserved heterogeneity exacerbates this effect.

The effects of covariates are increasingly attenuated over time in the context of unobserved heterogeneity. The proportional hazards assumption and form of μ imply that

$$\frac{\partial \log \lambda^*(t|\mu)}{\partial x_j} = \beta_j$$

With gamma-distributed unobserved heterogeneity, we instead have

$$\frac{\partial \log \lambda(t|\mu)}{\partial x_j} = \beta_j[S(t|\mu)]^{\sigma^2}$$

which is increasingly attenuated in t as compared to the no unobserved heterogeneity benchmark. Also of note, the effects of covariates are non-constant in the context of unobserved heterogeneity.

To understand the implications of these results, we first consider the early periods where some individuals are incarcerated and some are not. Assume everyone is incarcerated for at least a few periods, x_j is incarceration and β_j is negative, as we find. As t increases, the negative effect of incarceration on mortality risk is attenuated due to a relative decrease in the average unobserved mortality risk of the non-incarcerated population relative to the incarcerated population. That is, when incarceration reduces the likelihood of mortality, it creates a compositional effect that leaves more high risk individuals alive in this group. This result increases in t, so the cross-group difference in mortality during incarceration becomes increasingly attenuated.

The changing sample composition during the time of incarceration implies that the incarcerated group has higher average mortality risk post-incarceration in the context of unobserved heterogeneity. As those with highest unobserved mortality risk are most likely to die, the elevated post-release effect should decay in time. The combined effects of a positive compositional effect between the incarcerated and non-incarcerated groups during incarceration (i.e., less negative) and a positive compositional effect post-incarceration implies that the bias for our prospective DiD estimate of β is ambiguous.

In Section 2.2.3, we estimate the degree of unobserved heterogeneity in a parametric model similar to the one above. We find evidence consistent with a negligible variance of unobserved heterogeneity: our estimates of the variance of unobserved heterogeneity from a mixed proportional hazards model are very small ($< 1 \times 10^{-5}$), and the other parameter estimates with and without unobserved heterogeneity are indistinguishable. The next subsection also uses simulations of increasing dispersion of unobserved heterogeneity to show that the likely effects are small. Put together, this indicates that unobserved heterogeneity should not generate serious bias in our estimates.

C1 Simulations of unobserved heterogeneity

As an alternative approach to the problem of unobserved heterogeneity, this section runs simulations to place bounds on the extent to which it could bias our estimates. Note that in the paper, we control for a number of observable determinants of mortality: as a result, if incarceration induces compositional changes related to those observable determinants of mortality, this will not bias our estimator. However, if there is significant unobserved heterogeneity, then compositional changes can bias our estimates.

A bias from unobserved heterogeneity will make both the pre-release and post-release estimates more positive. The rationale for both coefficients is the same. Throughout the pre-release period, the unobservably more mortality-prone non-incarcerated individuals are more likely to die relative to those who are incarcerated. This generates a compositional difference between the samples. This first biases the pre-release coefficient positively, since the control group becomes unobservably less risky relative to the incarcerated group. This same bias continues in the post-release period as the non-incarcerated group is still compositionally less risky although this difference could dissipate over time.

To assess the extent of this issue, we run three sets of simple simulations—one with no unobserved heterogeneity (NOH) and two with varying degrees of unobserved heterogeneity. In each of these simulations, we generate simulated mortality data, run our benchmark specification from Equation 3, and save the pre- and post-release coefficients. We then compare the distribution of estimates to measure the extent to which increased unobserved heterogeneity might affect our estimates of pre- and post- release effects.

First, we take our data and estimate the mortality probability for each defendant in each quarter based on observable characteristics of defendants (e.g. age, race, gender, previous criminal history). In the NOH simulation, this is their predicted mortality probability in the quarter. In the simulations with unobserved heterogeneity, we assume individual-specific, time-invariant, and gamma-distributed unobserved heterogeneity. We take a draw for each individual from the relevant gamma distribution and multiply that by the estimated mor-

tality probability in each quarter to get predicted mortality probability in that quarter.

Next, to approximate the treatment effect of incarceration, we set the predicted probability of mortality equal to zero in periods while incarcerated. While this is more extreme than actually observed in our data, it is a helpful simplifying assumption and is an upper bound since it will lead to the most extreme possible form of bias. Finally, we simulate death as occurring in a period if a draw from the random uniform distribution is below the individual's predicted mortality probability in that period (and they have not already died in a prior period). We run 500 simulations under each of the three cases and then plot the distribution of parameter estimates in Figure A7.

For the unobserved heterogeneity simulations, we select the parameters of the gamma distribution to preserve the mean mortality likelihood and vary the variance (i.e., the product of the shape and scale parameters is equal to one). In the first simulation, we select parameters that double the standard deviation in mortality risk, while the second quadruples the standard deviation of mortality risk. Note that a doubling of the standard deviation of mortality risk is roughly the same as the proportional increase in the standard deviation of predicted mortality risk due to age. Since age is by far the most important predictor of mortality risk, this seems like a good benchmark for the increase in variance that unobserved heterogeneity may produce. This is also far larger than the estimated variance parameter estimated in Table 3, so will be a clear upper bound on how unobserved heterogeneity affects results in our context.

While it is the case that an increased variance in the unobserved heterogeneity parameter shifts the distribution, Figure A7 shows that this effect is quite small. For example, even under the more extreme version of unobserved heterogeneity, the distributions of both pre- and post-release coefficients are only biased upwards by approximately 15 deaths per 100,000 relative to the NOH distribution (panels A and B respectively). This is quite small relative to the estimated pre-release effect of incarceration of -357 deaths per 100,000. Furthermore, in both cases, it makes our estimates more conservative as to the extent to which incarcer-

ation reduces mortality, so this has little impact on the high-level take-aways of the paper. Intuitively, the economically small bias reflects the fact that the probability of mortality in any given period is not very high for even the riskiest set of individuals—and as a result, the compositional changes in the sample induced by stints of incarceration are not very large.[3] Given that we observe minimal bias generated by even substantially more severe unobserved heterogeneity than we estimated to be present in our sample, we conclude that this is not a major issue for our estimates.

Panel C of Figure A7 looks at the difference-in-differences estimator within these simulations. Recall that the difference-in-differences estimator is the difference between the post-release and pre-release estimates. Since those estimates are biased upwards by similar amounts, the two biases approximately cancel each other out. As a result, the absolute magnitude of the bias in the DID estimates is small. However, since the bias on the pre-release coefficient is slightly smaller than that on the post-release coefficient, the difference-in-differences estimate is not conservative like the pre/post release estimates are; instead it is slightly biased downwards. Thus, while both approaches perform well, the DiD (relative to the cross-group comparison) has a smaller degree of bias but in the direction of the effect we are estimating.[4]

[3]There could also be individual-specific, time-varying unobserved heterogeneity—e.g. shocks that occur due to an outbreak of violence in the individual's community and dissipate over time such as a gang conflict. Note that this represents less of a problem for our estimates since the shock can dissipate over the period of incarceration, and so the compositional effect should be reduced; this is exactly what we find when we run simulations with time-varying unobserved heterogeneity. We focus on time-invariant unobserved heterogeneity here since that will provide an upper bound on the bias generated by time-varying unobserved heterogeneity.

[4]The extent of bias in the difference-in-differences estimates also depends on the exact form of unobserved heterogeneity. For example, in simulations with time-varying, positively correlated unobserved heterogeneity that we have examined, we typically find that the magnitude of the bias is larger for pre-release than the post-release estimates (though both are still conservative as to the extent incarceration reduces mortality). As a result, the DiD estimator is conservative as to the extent that incarceration reduces mortality risk, and the absolute magnitude of the bias is smaller than in the time-invariant case.

D Prospective difference-in-differences

In Section 2.3, we conduct a *prospective DiD* around the *removal* of treatment, i.e., exit from incarceration. Using only periods after sentencing, we define the prospective DiD as the change in mortality risk between incarceration and the post-release period for those sentenced to incarceration relative to the changes over time in mortality risk observed in the comparable non-incarcerated group. This appendix shows that this identifies the ATT with a few additional assumptions beyond standard DiD parallel trends.

The research design can be described in a potential outcomes framework. For simplicity, suppose there are two periods. In period 0, only the treatment group ($D = 1$) is incarcerated, while neither group is incarcerated in period 1. Potential outcomes are denoted $Y_t^d(D)$, where $t \in 0, 1$ indexes time, d indexes potential treatment, and D indexes actual treatment (e.g. $Y_0^1(1)$ is the potential outcome observed in period 0 for a treatment group individual who is treated in period 0, while $Y_0^0(1)$ is for a treatment individual who is untreated in period 0). For outcomes in period 1, we express the history of treatment in the superscript as (d_0, d_1), representing treatment in periods 0 and 1 (e.g. $Y_1^{1,0}(1)$ is the outcome in period 1 for a treatment individual who was incarcerated in period 0 but not period 1). We are interested in the ATT of how mortality differs in the state of incarceration versus non-incarceration, $\beta^{ATT} \equiv E[Y_0^1(1) - Y_0^0(1)]$.

The prospective DiD is the regular DiD run "backwards" in time: the difference in mortality risk between period 0 and period 1 for the treatment group (incarcerated in period 0 but not period 1) minus the difference in mortality risk between the periods for the non-incarcerated. The estimator is equal to

$$\widehat{\beta} = \left[\bar{Y}_0^1(1) - \bar{Y}_1^{1,0}(1)\right] - \left[\bar{Y}_0^0(0) - \bar{Y}_1^{0,0}(0)\right] \tag{A1}$$

where $\bar{Y}_t^{d_0,d_1}(D)$ is the mean value of the outcome for group D in period t experiencing treatment history (d_0, d_1). We examine the expected value of $\widehat{\beta}$ and reformulate it in terms of

β^{ATT} and two additional quantities. With two substitutions—the addition and subtraction of $Y_0^0(1)$ and $Y_1^{0,0}(1)$—we find:

$$E[\widehat{\beta}] = \beta^{ATT} + \underbrace{E\left[Y_1^{0,0}(0) - Y_0^0(0)\right] - E\left[Y_1^{0,0}(1) - Y_0^0(1)\right]}_{\text{Parallel trends assumption}} - \underbrace{E\left[Y_1^{1,0}(1) - Y_1^{0,0}(1)\right]}_{\text{Post-release ATT}} \quad \text{(A2)}$$

The second term represents the difference between the treated and untreated groups in how much their outcomes would change between period 0 and period 1 if untreated in both periods. This is equal to zero under the standard parallel trends assumption. Section 3.2 presents evidence against violations of parallel trends biasing our results.

The third term is the post-release effect, i.e., the effect of experiencing the treatment in period 0 on mortality in period 1. Thus, the prospective DiD returns the direct effect of incarceration under the parallel trends assumption and with no post-release effects. If there are post-release effects but their sign is the same as β^{ATT}, then $\widehat{\beta}$ will be a conservative estimate of β^{ATT}.

The preceding discussion of the prospective DiD omits the possible effects of persistent, individual-specific unobserved heterogeneity in mortality risk. The effects of unobserved heterogeneity could be analyzed using the potential outcomes framework above by explicitly conditioning outcomes on past survival. If so, the prospective DiD expression can be rewritten with an additional bias term. The implications of this bias, and the possible ways to test for it, are most easily understood in a hazard framework. For clarity, this is the approach we take.

Appendix C uses a simple proportional hazard model to show that the presence of unobserved heterogeneity could generate compositional effects that would impact our estimates—for example, if incarceration reduced the risk of death during the time of incarceration, as we will find, the incarcerated population would develop a progressively higher average unobserved mortality risk relative to the non-incarcerated group. This would affect our estimate of $\widehat{\beta}$ because after release, the formerly incarcerated population would have more individuals

with higher mortality risks than the surviving non-incarcerated population. Section 2.2.3 presents both a reduced form test for unobserved heterogeneity and estimates from a parametric hazard model. We find evidence for negligible effects of unobserved heterogeneity in practice, supporting the use of this estimator to measure the causal effect of incarceration on mortality.

E Decomposing the prospective diff-in-diff estimates

Our identification strategy compares offender mortality before and after pre-scheduled releases from incarceration, where we condition on demographic characteristics and time-since-decision using non-incarcerated defendants to estimate the controls. Figure A1 shows how conditioning on these factors affects our results. For reference, the black line represents the main estimates from Panel A of Figure 3. To understand the effect of using the non-incarcerated sample, the blue line estimates mortality among initially-incarcerated defendants in each half-year relative to the mortality rate of only the initially-incarcerated defendants 7 years following release, conditional only on age. As in our main results, there is a dramatic increase in mortality at the time of release, followed by no subsequent change. The purple line adds in initially-non-incarcerated defendants and quarter since court decision fixed effects, and so the coefficients reflect the difference in mortality between the incarcerated and non-incarcerated, before and after release. While we see a similar increase in mortality upon release, the post-release mortality is higher for the initially incarcerated defendants without the controls. Comparing the three sets of results, we conclude (1) that the main results almost entirely reflect variation within the set of initially-incarcerated defendants, and (2) that the degree of selection along the demographic and crime type margins is small relative to the effect size.

F Accounting for incarceration on later charges

Our main DiD approach estimates the effect of incarceration on mortality using the change in incarceration status at the time of release. We measure the time of release based on the defendant's pre-determined sentence in court. One concern with this approach is that we implicitly assume that those who are not initially sentenced to incarceration were not incarcerated on later charges and that after release, individuals are not reincarcerated. Both would cause our estimates to understate the direct effect of incarceration on mortality. In practice, around 18.6% of individuals who are not initially sentenced to incarceration are incarcerated on different charges within 3 years, and 33.8% of the incarcerated are reincarcerated within three years of release. We estimate a modified version of the DiD regression in which we instrument for incarceration in each quarter using the following 2SLS setup:[5]

$$death_{ict} = \theta_1 Sentenced_{ic} + \theta_2 IncarAnyCase_{ict} + X_{iact}\phi + \eta_t + \gamma_a + \psi_c + \varepsilon_{iact} \tag{A3}$$

$$IncarAnyCase_{ict} = \pi_1 Sentenced_{ic} + \pi_2 Sentenced_{ic} \times PreRelease_{ict} + X_{iact}\Delta + \rho_t + \omega_a + \tau_c + u_{iact}$$

In this specification, $IncarAnyCase_{ict}$ is a dummy variable equal to one in every quarter individual i was incarcerated for any reason. We instrument for $IncarAnyCase_{ict}$ with $Sentenced_{ic} \times PreRelease_{ict}$, whether the individual was sentenced to be incarcerated in that quarter in the initial case c. Panel A of Table A2 estimates this over the full sample, while Panel B estimates it for only those who were initially sentenced to incarceration. The estimates are approximately twenty percent larger than our baseline results, but the conclusions are qualitatively the same.

[5] The IV-DiD identifies the LATE for those who switch treatment status under two non-standard assumptions: constant mean treatment rates over time among the control group (non-incarcerated), and common trends in mean untreated potential outcomes and treated potential outcomes (De Chaisemartin and d'Haultfoeuille, 2018). The first assumption is implied by the equality of the first stage with and without the control group (column 1 of panels A and B). The second assumption is also likely satisfied—the period-by-period comparison on overall mortality in Figure 3 shows parallel trends in the post-release period (indicative of parallel trends in untreated outcomes) and parallel trends in the pre-release period (indicative of parallel trends in treated outcomes).

G Power calculations for use of a judge instrument

A standard approach to estimating the incarceration-mortality relationship would be to instrument for incarceration using the random assignment of cases to judges, as in other work with this data (Norris et al., 2021). In Ohio, criminal cases are randomly assigned to judges who differ in their propensity to incarcerate defendants, which we can use as a source of exogenous variation in incarceration probability. Differences between judges in sentencing behavior are significant; assignment to the most severe judge increases the likelihood of incarceration by 34 percentage points relative to the least severe judge. We construct the instrument as the judge's leave-out propensity to incarcerate and find that this has a strong first stage relationship with incarceration ($F = 5,605$).

Despite the large number of randomly assigned cases in our data (809,347), we find that the IV estimates are imprecise due to the low likelihood of mortality. Panels A and B of Figure A6 plot coefficients from a regression of incarceration of whether the defendant died in the t^{th} quarter since the original charge (panel A) or has died within t quarters of the original charge (panel B), instrumenting for incarceration using judge severity. The confidence intervals contain our point estimates, but are too wide to be informative.

A natural question is whether it would ever be possible to gather enough data to use this approach. We conduct Monte Carlo simulations to estimate the sample sizes required to detect an effect of the magnitude found using the prospective difference-in-differences design. For these simulations, we take the same set of court data in this paper and Norris et al. (2021). This contains the identity of the judge to whom the case is assigned, which we use to construct a leave-out measure of the judge's propensity to incarcerate. We then estimate the mortality probability for each defendant in a given quarter based on a regression of observable characteristics of defendants (age, race, gender, previous criminal history) on mortality in our death records and applying the estimated treatment effect of incarceration on mortality from our main analysis to adjust downward the mortality probability in each quarter in which the inmate is sentenced to incarceration. Based on these quarterly mortality

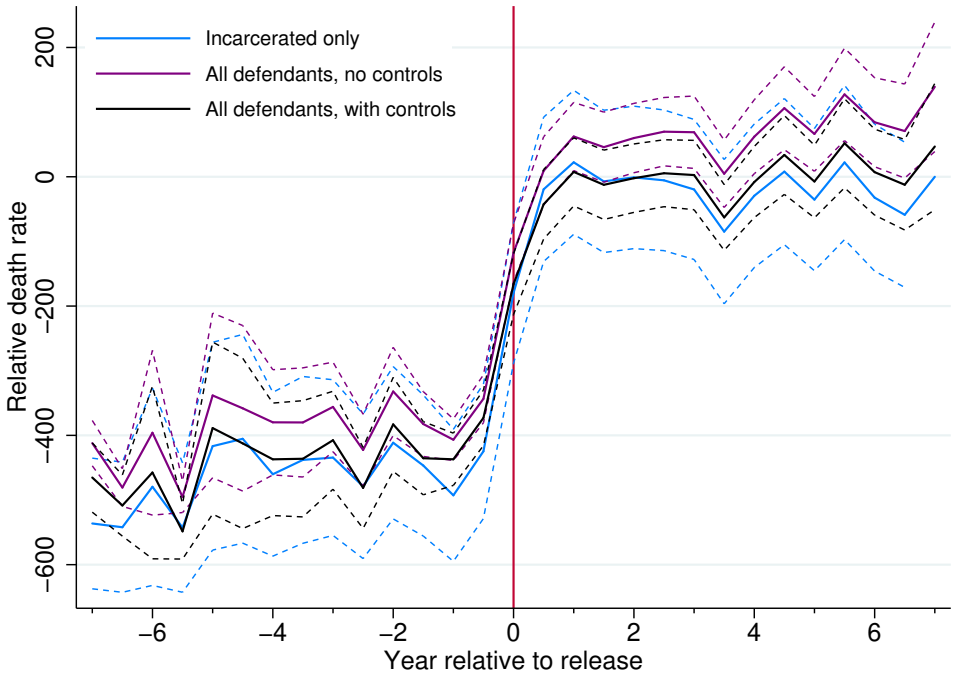
probabilities, we estimate the mortality for the inmate after 1 year, 5 years and 10 years after the sentencing decision.

We then draw samples with replacement to generate simulated datasets of 1, 2, 5, 10, 20, and 30 million cases, and repeat this 200 times for each sample size. In each sample, we simulate whether the individual has died using their individual-specific estimated mortality rate. We then estimate the relationship between incarceration and mortality, instrumenting for incarceration using the severity of the assigned judge. This approach means that our simulated data replicates the distribution of sentence lengths in our actual data, as well as the characteristics of individuals assigned those sentences. We estimate power for each sample size as the fraction of samples in which we can reject a null hypothesis of zero at a 95% confidence level.

Panel C of Figure A6 plots the relevant power curves and shows that a sample size of more than 20 million observations is required to achieve power at the levels typically considered adequate in randomized controlled trials (80%) for 5 year mortality. This is over ten times as large as the largest set of randomly assigned court cases ever assembled for economics of crime research, which consists of thirty years of court records from Harris county, the third largest county in the United States (Mueller-Smith, 2015). If the number of randomly assigned cases per resident is roughly similar in other counties as in Harris county and the three counties studied here (approximately 15,000 cases per million residents annually in all four), then even with 20 years of data from all of the 20 largest counties in the United States that use random assignment of cases to judges, there would still only be around 13 million cases in which there is random assignment of cases to judges (see Thorley (2020) for details on which counties practice random assignment of cases to judges). Thus this approach is unlikely to be successful.

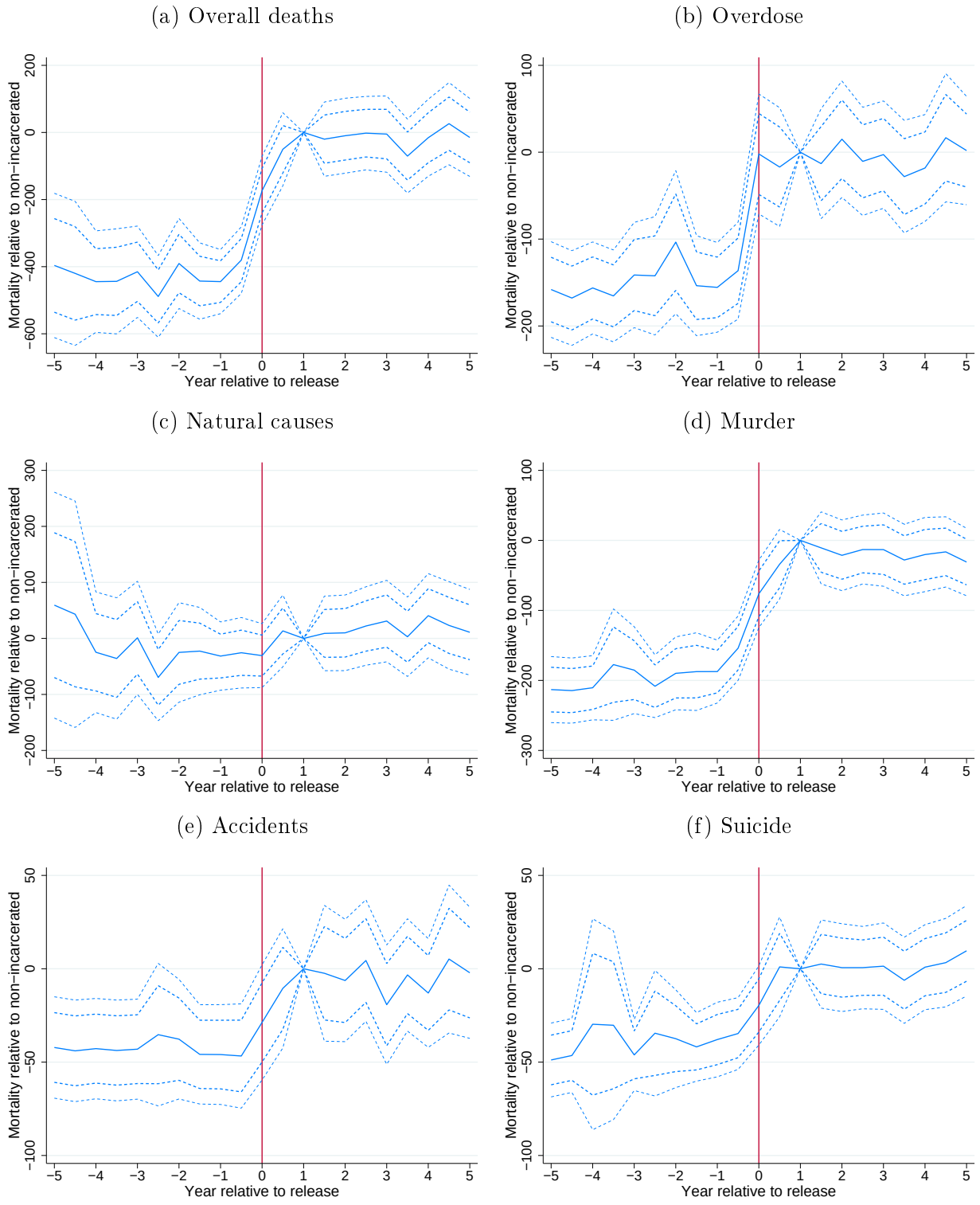
Appendix exhibits

Figure A1: Period-by-period comparison in different samples and with different controls



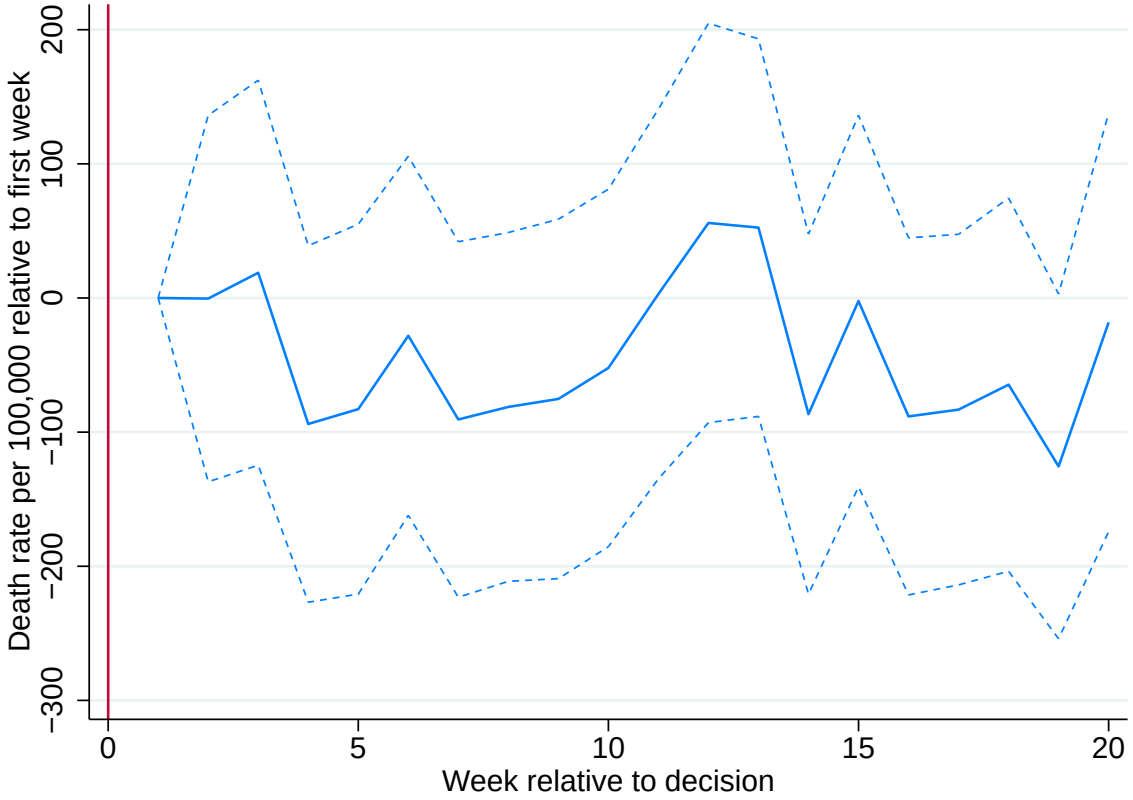
This figure shows the results from regressions of annual mortality per 100,000 on time relative to release, estimated using Equation 1. The regression represented by the blue line includes only defendants initially sentenced to incarceration, and controls only for age. As a result, coefficients represent mortality relative to the last period, the 14^{th} half-year following release. The purple line adds initially non-incarcerated defendants and quarter since decision fixed effects, so the coefficient estimates represent mortality relative to the non-incarcerated. The black line adds controls for quarter of decision, court-month, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The data consist of seven years following sentencing for each defendant. Dotted lines represent 95% confidence intervals two-way clustered at the individual and court-month level.

Figure A2: Normalized event study by time relative to exit



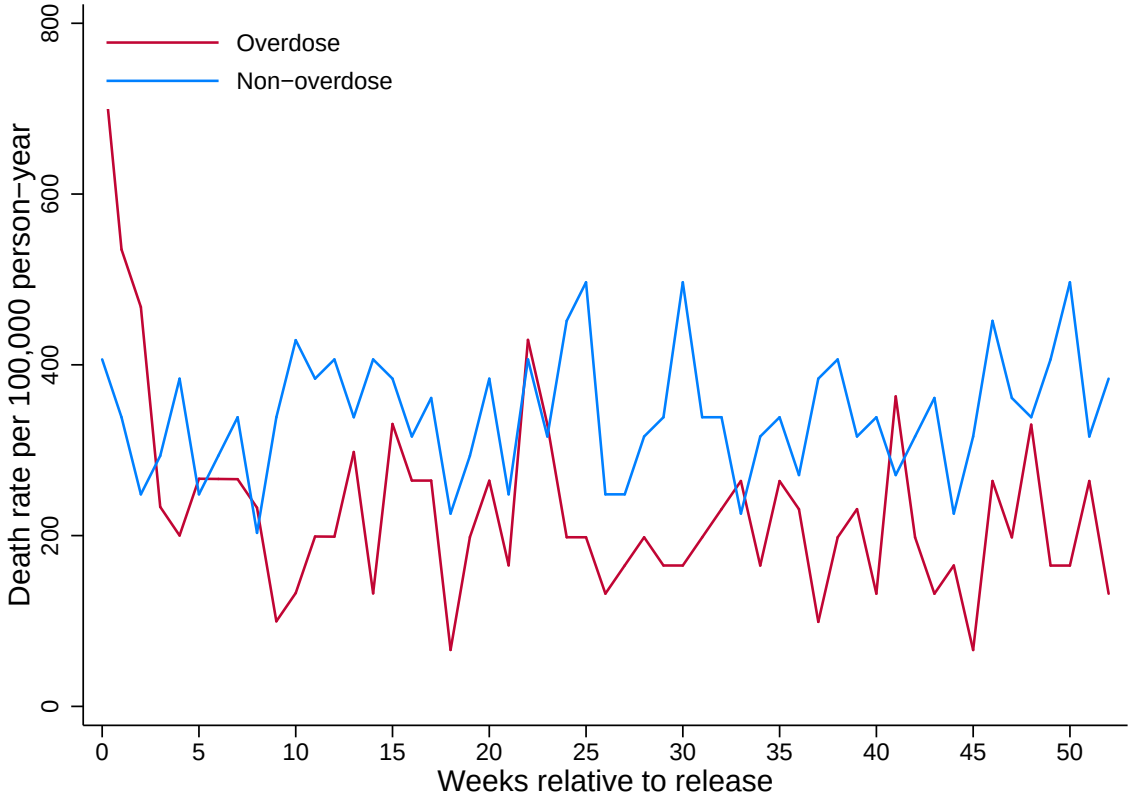
This figure displays event-study estimates of the difference in annual mortality per 100,000 between the initially incarcerated and the initially non-incarcerated group, as compared to a normalized difference in the second half-year after release. Overall mortality in Panel A, cause of death-specific results in Panels B-F. Regressions include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The data consist of seven years following sentencing for each defendant. Dotted lines represent 95% confidence intervals two-way clustered at the individual and court-month level, and thin dashed lines represent the sup-t confidence band (Montiel Olea and Plagborg-Møller, 2019).

Figure A3: Mortality by week since decision for non-incarcerated defendants



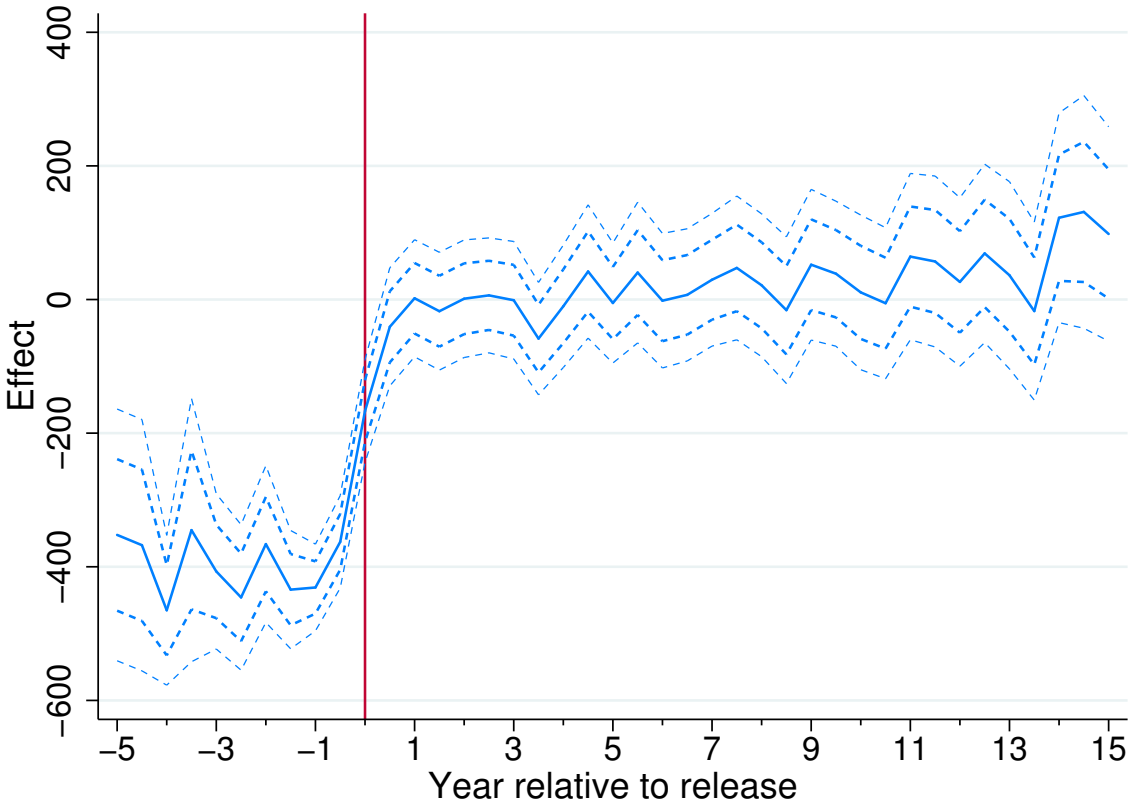
This figure displays regression estimates of annual mortality per 100,000 on indicators for week since the decision among initially non-incarcerated defendants, conditioning on quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. Dotted lines represent 95% confidence intervals two-way clustered at the individual and court-month level.

Figure A4: Death rates in the year following release, overdose vs non-overdose



This figure displays the raw mortality rate (annualized and per 100,000) for the formerly incarcerated over the year following their release (in one week intervals). It separates mortality into deaths due to overdose and non-overdose related deaths.

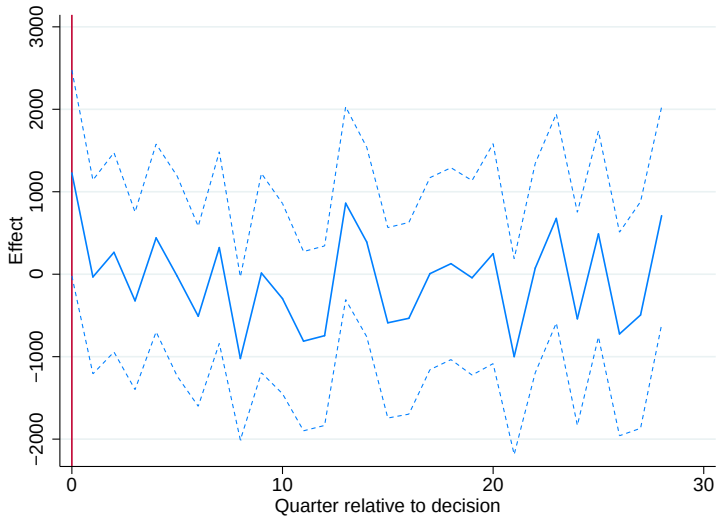
Figure A5: Death rate by time relative to exit, long-run estimates



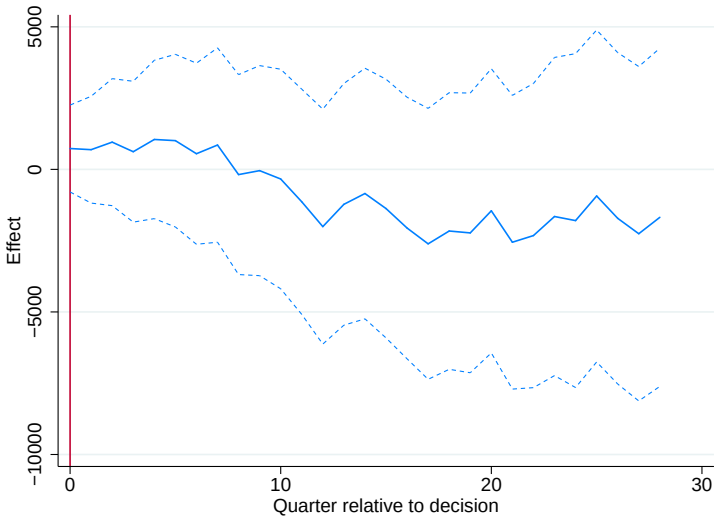
This figure displays regression estimates of the difference in annual mortality per 100,000 between the initially non-incarcerated and the initially incarcerated in each half-year relative to release. Regressions estimated according to Equation 1, and include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The data consist of 17 years following sentencing for each defendant and excludes individuals with sentences longer than 17 years. Dotted lines represent 95% confidence intervals two-way clustered at the individual and court-month level, and thin dashed lines represent the sup-t confidence band (Montiel Olea and Plagborg-Møller, 2019).

Figure A6: IV using judge severity as instrument for incarceration

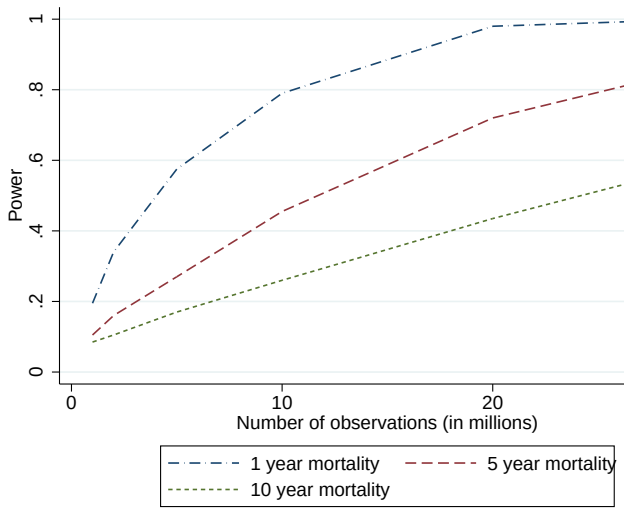
(a) Dependent variable: died in quarter



(b) Dependent variable: dead in quarter



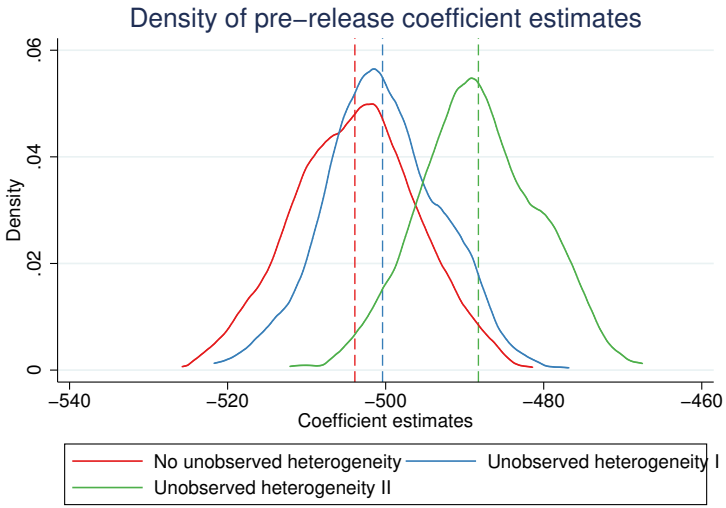
(c) Power curve for estimated effect



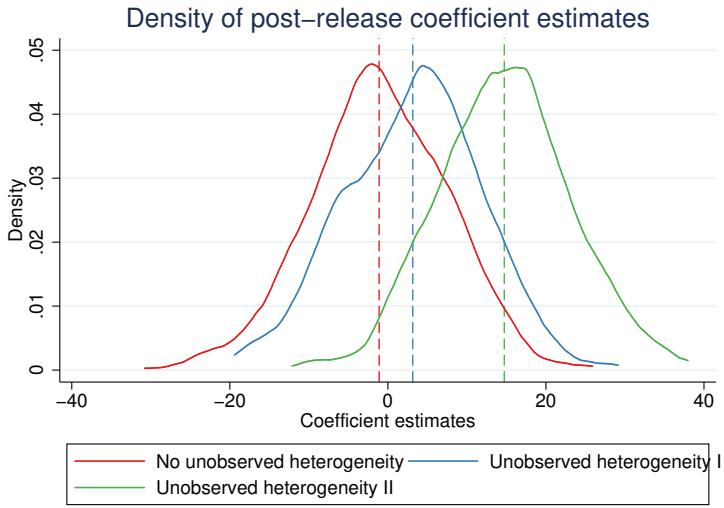
Panels (a) and (b) of this figure display coefficients from IV regressions of the outcome in panel header on initial incarceration, instrumented by judge severity and estimated separately for each quarter since judge assignment. Mortality outcomes measured per 100,000 person-years. Panel (a) examines whether the defendant died in that quarter, while panel (b) examines whether the defendant died in that quarter or any preceding quarter after sentencing. Regressions include controls for prior criminal activity and court-month fixed effects. Dotted lines represent 95% confidence intervals two-way clustered at the court-month and defendant level. Panel (c) plots power to detect the effect of incarceration on mortality using judge severity as an instrument. We run Monte Carlo simulations based on our data in order to estimate power to detect the effect on mortality after 1, 5, and 10 years (at a 95% confidence level) if incarceration reduces mortality by the magnitude estimated in the paper.

Figure A7: Simulations of unobserved heterogeneity

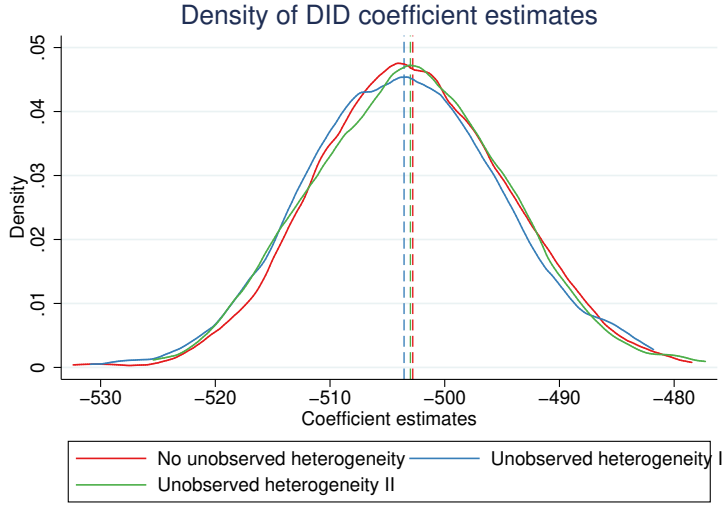
(a) Distribution of pre-release coefficient



(b) Distribution of post-release coefficient



(c) Distribution of diff-in-diff coefficient



Panels (a) and (b) of this figure display the distribution of coefficients from simulations of the extent to which the presence of unobserved heterogeneity affects our coefficient estimates. Under "no unobserved heterogeneity", simulated probability of mortality is only a function of observable characteristics. Under "unobserved heterogeneity 1" and "unobserved heterogeneity 2", we include gamma distributed unobserved heterogeneity term that increase the standard deviation of mortality risk by 100% and 300% respectively.

Table A1: Effect of incarceration on annual mortality per 100,000

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Overall		*Cause of death*					
			Overdose	Murder	Natural	Accident	Suicide	Other external
Incarcerated × pre-release	-310.52***	-311.89***	-71.72***	-121.48***	-65.57***	-26.96***	-31.53***	-4.96**
	(14.101)	(18.820)	(9.139)	(8.362)	(12.586)	(5.834)	(4.169)	(2.275)
Incarcerated × post-release	25.51**	3.11	-1.45	2.30	-6.67	6.45**	0.40	1.21
	(11.284)	(9.952)	(4.652)	(3.682)	(7.537)	(3.245)	(1.867)	(1.046)
Controls	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
DiD estimate	-336.03***	-314.99***	-70.26***	-123.78***	-58.90***	-33.41***	-31.93***	-6.17***
Post-release mean (1 year)	486	486	97.5	140.4	171.4	50.2	32.7	9
Observations	30,596,576	30,596,576	26,604,879	30,596,576	30,596,576	30,596,576	30,596,576	30,596,576
			Cause of natural death					
			Heart	Cancer	Infection	Respiratory	Liver/alcohol	Circulatory	Other
Incarcerated × pre-release			-26.94***	0.72	-11.00***	-3.22	-3.31	-1.79	-20.03***
			(6.446)	(5.463)	(4.114)	(3.473)	(3.641)	(2.642)	(6.049)
Incarcerated × post-release			-7.95**	2.43	-1.24	-3.21*	-0.34	-0.64	4.28
			(3.602)	(3.268)	(2.507)	(1.651)	(2.570)	(1.575)	(3.661)
Controls			Yes	Yes	Yes	Yes	Yes	Yes	Yes
DiD estimate			-18.99***	-1.71	-9.76**	-0.01	-2.97	-1.15	-24.32***
Post-release mean (1 year)			38.3	31.6	19.2	10.7	7.9	10.7	53
Observations			30,596,576	30,596,576	30,596,576	30,596,576	30,596,576	30,596,576	30,596,576

This table reports difference-in-differences estimates of the effect of incarceration on annual mortality per 100,000 for people whose date of decision was prior to 2010. Regressions estimated according to Equation 3, and include quarter of decision, court-month, age, race, poverty decile, female, age>30 when charges filed, previous charges and incarcerations, and offense type fixed effects. The post-release mean is the death rate for the first year after release. Standard errors in parentheses and clustered at the individual and court-month level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A2: Effect of incarceration on annual deaths: IV

	First stage	Reduced form	IV
	(1)	(2)	(3)
Panel A: All cases			
Sentenced in quarter	0.8***	-368.5***	
	(0.001)	(17.931)	
Incarcerated for any reason			-448.1***
			(21.784)
Non-incarcerated dependent mean	.066	597	597
Incarcerated post-release mean	.16	622	622
Complier untreated mean	0	631	631
Observations	41,211,056	41,211,056	41,211,056
Panel B: Incarcerated only			
Sentenced in quarter	0.8***	-291.2***	
	(0.002)	(23.274)	
Incarcerated for any reason			-358.0***
			(28.590)
Incarcerated post-release mean	.16	622	622
Complier untreated mean	0	696	696
Observations	6,346,756	6,346,756	6,346,756

This table reports IV-related estimates. Panel (a) includes all cases, while panel (b) only includes cases for individuals sentenced to incarceration. Column (1) is the first stage of being incarcerated in any case on being sentenced to incarceration interacted with being pre-release in this case. Column (2) is a regression of overall mortality on being sentenced to incarceration interacted with being pre-release in this case, the difference-in-differences effect. Column (3) displays the IV estimate of overall mortality on being incarcerated, estimated using Equation A3. Regressions include quarter of decision, court-month, age, race, below 25^{th} percentile neighborhood SES, female, age>30 when charges filed, previous charges and incarceration, and offense type fixed effects. Standard errors in parentheses and clustered at the individual and court-month level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A3: Mortality hazards and future incarceration status (NLSY)

	General health (1)	Drug use (2)	Smoker (3)	Binge drinking (4)	Ever in gang (5)	Depression (6)
			Panel A: No controls			
Incarcerated t+1 (δ)	-0.08	0.02	0.06**	0.05	0.06	0.08
	(0.054)	(0.021)	(0.024)	(0.329)	(0.045)	(0.066)
Observations	2,065	1,882	2,054	1,880	802	903
			Panel B: With controls			
Incarcerated t+1 (δ)	-0.05	0.01	0.03	0.18	0.05	0.05
	(0.055)	(0.020)	(0.024)	(0.335)	(0.046)	(0.072)
Observations	2,061	1,878	2,058	1,876	835	897

This table reports the coefficients from regressing variables related to mortality risk on a dummy variable for whether the individual was incarcerated in the following year. The sample only includes people that have been charged with a crime in the following year and were not incarcerated in the current year, since present incarceration may reduce their access to these activities. Both panels include age-ethnicity-gender fixed effects. The second panel also includes fixed effects for income decile, charge type, arrests, and cumulative number of previous arrests. The sample in both panels is restricted to years between 2003 and 2016 since the full set of control variables are only available for those years. As a result of this sample restriction, the estimates in panel A differ slightly from those in Table A4. General health is self-reported health score from 1 to 5, where 1 is poor and 5 is excellent. Drug use indicates drug use in the last year. The variable is available for the years 1999-2013 and 2015. Smoker is a dummy for whether the respondent smoked cigarettes in the last year. Binge drinking is the number of days in which the reported consuming 5 or more drinks of alcohol in the last month. Questions regarding gang activity were only asked in years 1997-2005. Depression is self-reported frequency of feeling depressed from 1 to 4, where 1 is 'none of the time' and 4 is 'most of the time'. Questions regarding mental health were only asked in years 2000, 2002, 2004, 2006, 2008, 2010, and 2015. Standard errors in parentheses and clustered at the individual level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A4: Mortality hazards and criminal justice involvement (NLSY)

	General health (1)	Drug use (2)	Smoker (3)	Binge drinking (4)	Ever in gang (5)	Depression (6)
Charged, not incarcerated t+1 (η)	-0.21***	0.12***	0.29***	1.63***	0.11***	0.15***
	(0.022)	(0.009)	(0.010)	(0.108)	(0.011)	(0.024)
Incarcerated t+1 (δ)	-0.27***	0.13***	0.34***	1.49***	0.21***	0.27***
	(0.036)	(0.014)	(0.015)	(0.195)	(0.020)	(0.044)
$\delta = \eta$ p-value	0.083	0.344	0.001	0.501	0.000	0.015
Not in court mean	3.86	0.05	0.38	1.27	0.11	1.36
Observations	130,947	104,464	131,299	122,780	70,018	52,004

This table reports the coefficients from regressing variables related to mortality risk on a dummy variable for being in court (but not incarcerated) in the next year and a dummy variable for being incarcerated in the next year. Thus the left out group is individuals who were not in court in the next year. The sample only includes people who were not incarcerated in the current year since present incarceration may reduce their access to these activities. All regressions include age-ethnicity-gender fixed effects. General health is self-reported health score from 1 to 5, where 1 is poor and 5 is excellent. Drug use indicates drug use in the last year. The variable is available for the years 1999-2013 and 2015. Smoker is a dummy for whether the respondent smoked cigarettes in the last year. Binge drinking is the number of days in which the reported consuming 5 or more drinks of alcohol in the last month. Questions regarding gang activity were only asked in years 1997-2005. Depression is self-reported frequency of feeling depressed from 1 to 4, where 1 is 'none of the time' and 4 is 'most of the time'. Questions regarding mental health were only asked in years 2000, 2002, 2004, 2006, 2008, 2010, and 2015. Standard errors in parentheses and clustered at the individual level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table A5: Mortality hazards and criminal justice involvement (NLSY)

	General health (1)	Drug use (2)	Smoker (3)	Binge drinking (4)	Ever in gang (5)	Depression (6)
			Panel A: Black			
Charged, not incarcerated t+1 (η)	-0.09**	0.04***	0.24***	0.72***	0.13***	0.15***
	(0.040)	(0.010)	(0.020)	(0.133)	(0.024)	(0.051)
Incarcerated t+1 (δ)	-0.03	0.05***	0.36***	0.96***	0.21***	0.24***
	(0.066)	(0.016)	(0.026)	(0.251)	(0.036)	(0.079)
$\delta = \eta$ p-value	0.419	0.605	0.000	0.360	0.034	0.308
Not in court mean	3.81	0.02	0.31	0.65	0.14	1.43
Observations	34,984	27,646	34,893	32,289	18,326	13,857
			Panel B: White			
Charged, not incarcerated t+1 (η)	-0.28***	0.16***	0.32***	2.13***	0.10***	0.17***
	(0.029)	(0.014)	(0.013)	(0.164)	(0.014)	(0.031)
Incarcerated t+1 (δ)	-0.40***	0.20***	0.37***	1.81***	0.17***	0.27***
	(0.049)	(0.026)	(0.020)	(0.354)	(0.029)	(0.063)
$\delta = \eta$ p-value	0.025	0.143	0.018	0.391	0.012	0.184
Not in court mean	3.93	0.06	0.44	1.61	0.07	1.32
Observations	66,882	53,588	67,245	63,258	36,218	26,629

This table reports the coefficients from regressing variables related to mortality risk on a dummy variable for being in court (but not incarcerated) in the next year and a dummy variable for being incarcerated in the next year. Thus the left out group is individuals who were not in court in the next year. The sample only includes people who were not incarcerated in the current year since present incarceration may reduce their access to these activities. All regressions include age-ethnicity-gender fixed effects. General health is self-reported health score from 1 to 5, where 1 is poor and 5 is excellent. Drug use indicates drug use in the last year. The variable is available for the years 1999-2013 and 2015. Smoker is a dummy for whether the respondent smoked cigarettes in the last year. Binge drinking is the number of days in which the reported consuming 5 or more drinks of alcohol in the last month. Questions regarding gang activity were only asked in years 1997-2005. Depression is self-reported frequency of feeling depressed from 1 to 4, where 1 is 'none of the time' and 4 is 'most of the time'. Questions regarding mental health were only asked in years 2000, 2002, 2004, 2006, 2008, 2010, and 2015. Standard errors in parentheses and clustered at the individual level. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.