SUPPLY OR DEMAND?

THE POLITICAL ECONOMY OF THE AMERICAN OVERDOSE EPIDEMIC

America is in the midst of an ongoing overdose epidemic. Existing scholarship tends to view addiction either as a biological response to repeated exposure to a chemical – suggesting a policy solution that is focused on keeping drugs out of the hands of the population – or as a rational choice to self-medicate in the face of "despair" or stressors emanating from factors like economic decline – implying that forces of "demand" are more important than of "supply" and recommends policy solutions that lie in social and industrial spheres. We bridge these two liteartures by understanding addiction as a learned behaviour that leads to the loss of self-control, developing in response to social adversity. We connect theory and data by relating spatial variation in overdose mortality rates to the joint effects of drug supply and social conditions, notably in the form of income dynamics, labour force participation, and incarceration rates.

Key words: drug supply, economic decline, incarceration, overdose mortality.

INTRODUCTION

America is in the midst of an ongoing overdose epidemic claiming tens of thousands of life every year. Much effort, both public and scientific, has been devoted to understanding its root causes and to arresting its continued upward-spiralling trajectory. Yet the existing literature is largely divided between two seemingly competing explanatory accounts. On the one hand, within the field of epidemiology and public health, emphasis has been placed on the role of pharmaceutical corporations in propagating higher prescription rates of opioid pain medication, leading to addiction, substitution with illicit opioids, and opioid-related deaths amongst vulnerable patients. The policy implication of this narrative seems largely focused on keeping drugs out of the hands of the population at all cost. On the other hand, within the field of economics, a strand of literature advances a social model of addiction, in which addiction is a

rational choice to self-medicate in the face of "despair" or stressor emanating from economic decline. Such a theory posits an alternative source of the epidemic and recommends policy solutions that lie in social and industrial spheres. The purpose of this paper is to make a sociological contribution to this debate by wedding these seemingly competing explanations within a unifying framework and by providing a novel empirical analysis that connects theory and evidence. We demonstrate that both the supply of opioids by pharmaceutical companies, as well as political and economic adversity stemming from economic decline, high rates of incarceration, and attendant social problems are jointly driving this epidemic.

Existing population-level studies of the drivers of the overdose epidemic are scarce, and those that exist typically come to inconsistent conclusions. We use previously unavailable empirical data and an alternative methodological strategy to isolate county-level variation in overdose mortality and generate key counterfactuals of interest, finding support for both opioid prescription rates and social conditions in the form of income decline and mobility, incarceration rates, and labour market conditions. Our analysis sheds novel light on the social dynamics underpinning a major health crisis and can inform future policy-making. Moreover, we showcase the contribution, both empirical and conceptual, that sociology can make to the understanding of population health.

EXPLAINING AMERICA'S POPULATION HEALTH CRISIS

In 2017, over 72,000 individuals, disproportionately from the bottom of the class structure, died from drug overdoses in the United States (National Institute on Drug Abuse, 2018), joining the ranks of over half a million people who have suffered the same fate since 1980 (Dwyer-Lindgren et al., 2018). A panoply of media portrayals has conveyed the corrosive impact of opioid addiction and mounting mortality burdens from drug use disorders across a rapidly

growing number of local communities. The overdose epidemic has been declared a national public health emergency, while researchers and policy makers alike are searching for effective means of curbing the exponentially growing death toll. Such interventions are predicated, of course, on identifying the underlying aetiology and relevant mechanisms at work. One causal narrative is concerned with the role of "supply-side" factors, and most notably the commercially driven promotion of prescription opioids. On the website of the National Institute on Drug Abuse,[1] the root cause of the epidemic is attributed to the diffusion of opioid pain relievers through the health care system, while the Centers for Disease Control and Prevention similarly focuses on efforts to swiftly curtail opioid prescribing and improve addiction treatment.[2] According to this account, aggressive marketing strategies pursued by pharmaceutical companies resulted in physicians increasing prescriptions of opioids for long-term pain relief (Keefe, 2017; Hoffman, 2019).

While public opinion has been shaped by extensive media focus on individual corporate actors, such as Purdue Pharmaceutical owned by the Sackler family, the most influential social scientific analysis has emphasised the social conditions, or "demand-side" variables driving so-called "deaths of despair". In this literature, spearheaded by Anne Case and Angus Deaton, economic decline, unemployment, and fractured social ties are put to the fore (Case and Deaton, 2015; 2017; 2020). In particular, the key ingredients of what they dub "cumulative disadvantage" are long-run patterns of deteriorating employment conditions, notably declining labour force participation rates amongst individuals with low levels of formal education, as well as unstable social bonds resulting from economic hardship, especially in the form of dwindling marriage rates and disintegrating family units.

Existing scholarship on the epidemic is thus structured around two putative causal forces – the *supply of* drugs and the *demand for* drugs (cf. Dasgupta et al., 2018). In the social science literature, Ruhm's study (2018) offers evidence in support of the "supply-side" account, finding weak and non-robust effects of economic conditions (combining local measures of unemployment, income, prices, and import competition) compared to a major effect of drug cost and availability on opioid deaths. His analysis of medium-run changes in drug-related mortality at the county level suggests that such mortality is higher in areas having experienced economic decline, whether relative or absolute, but that this correlation appears to be spurious once other county-level characteristics are taken into account. Similar arguments are made by Currie and Schwandt (2020), who posit that the experience of poor labor market conditions only account for a modest portion of the total variation in overdose mortality, whereas specific facets of the US health care market are more likely to have fostered the current crisis.

These findings, however, are at odds with other studies in which macroeconomic shocks are significantly associated with opioid deaths. For example, Venkataramani and colleagues (2020) find that opioid mortality rates have increased by 85% since the turn of the century in counties experiencing waves of automotive assembly plant closures compared to unexposed counties, equivalent to 8.6 excess deaths per 100,000 population relative to a baseline mortality rate of 12 deaths per 100,000. Contrary to Ruhm's analysis, this finding suggests a major role played by deindustrialisation in the making of the epidemic. This is corroborated by Monnat (2018, 2019) and Monnat et al. (2019), who discover the clustering of drug-related mortality in economically distressed areas.

In the present paper, we take our cue from this literature and further probe the interface between "supply" and "demand" frameworks. Our contribution is to connect theory and data such that

better sense can be made of these (at times) contradictory findings in the extant literature. While acknowledging the strengths of these recent papers, we believe that a more distinctly sociological approach may help address their limitations. As we describe below, the notion (whether implicit or explicit) according to which drug supply alone can account for the geographical patterning of overdose mortality relies on what we consider too narrow a conception of what causes substance use and addiction. On the other hand, while some studies discard or ignore the demand side, others – including Case and Deaton – ignore the supply side. On an empirical level, none of the mentioned studies explicitly examine the interplay between supply and demand, nor do they quantify the relevant magnitudes with respect to key counterfactuals of interest. The present paper seeks to examine these two dimensions in tandem rather than in isolation by using previously unavailable data and to quantify their relative import in the making of between-county inequality in drug-related mortality rates.

ADDICTION, DESPAIR, AND THE MAKING OF AN EPIDEMIC

Both the "supply" and the "demand" strands of existing scholarship on the American overdose epidemic rely on a particular conception of what drives addiction and substance use to begin with. On the one hand, the notion according to which the supply of drugs alone is sufficient to cause an epidemic is anchored in a conceptual framework in which addiction itself is a brain disease wrought by exposure to a chemical, typically materialised in changing neural systems that encode the experienced anticipation of reward. The physiological manifestation of addiction is linked to changes in dopamine metabolism through which synaptic[3] networks are subject to structural modification. Such rewiring of the brain's architecture is said to permanently alter the neurological basis for goal-seeking behaviour and self-control in the form of multiple distinct neurocognitive changes. However, as noted by Lewis (2018), durable neurocognitive changes are not exclusive to addictive behaviour. Rather, they can be revelatory

of an underlying neuroplasticity that unfolds as a result of the lived experience of one's social and material environment. Every human brain changes throughout the life course, sometimes in dramatic fashion, fostered by continuous feedback loops between experience and synaptic configurations. As such, the morphological discrepancies between "addicted brains" and "non-addicted brains" do not in themselves vindicate a simple brain-disease theory of addiction.

On the other hand, there is a sociological literature that views addiction as a process of *learning*. Addiction is here seen as a cognitively grounded behavioural response to exogenous environmental shocks that induce various forms of self-medication to mitigate physical and psychological suffering. It is construed as a form of learning anchored in the cognitive, symbolic, and emotional fabric of social life which subsequently fosters distinct physiological sensations. A landmark sociological contribution to this line of argument is Alfred Lindesmith's (1938) theory according to which drug addiction is not a unilinear function of substance use but emerges out of a subject's conscious effort to alleviate withdrawal distress. Addiction is said to be anchored not only in biochemically transformative interactions between some substance and the human body but in psychologically distinct and symbolically mediated experiences of meaning and interpretation (Lindesmith, 1968). Other learning approaches, located along a similar line of thought, emphasise the social settings through which addiction emerges, as well as the motives and dispositions by which learning unfolds (cf. Becker, 1953). As noted by Weinberg (1997), however, the Lindesmithian approach is rooted in a seemingly irreducible antinomy between meaningful experiences and biological sensations, between subjective cognitions and physical events, and ultimately between voluntaristic psychologism and biological reductionism. Arguably, such a dualism derives from "the overwhelming tendency to conceptualise human biology and human social life dichotomously as two, and only two, wholly discrete and independently integrated ontological domains" (Weinberg, 2013:

173). As such, the learning theory of addiction, at least in its predominant form, underplays the material basis of addiction and the neuronal substrate through which it manifests itself. It also tends to conceptualise the social dimension of addiction as nothing other than a deliberative and self-transparent act, a symbolically invested and meaningful form of "utility maximisation" on the part of the addicted person (cf. Weinberg, 2019). As such, it skips the pre-reflective layers of human practice and ignores that such practice is ecologically nested within broader structural configurations through which environmental shocks are generated. Although learning theory acknowledges that drug use is socially situated – and much of the existing literature charts such settings through ethnographic means (e.g. Becker, 1953; Ray, 1961; Stephens, 1991; Densin, 1993) – it rarely pays much heed to the macroscopic political and economic forces that shape micro-environments conducive to psychosocial distress.

In this paper, we emphasise the social nature of addiction while recognising its biological manifestations (Lewis, 2018; Szalavits, 2016, 2017). We propose a conceptual bridge between seemingly irreconcilable frameworks by viewing addictions as neuronally inscribed configurations of attraction, desire, pursuit, and reward, mediated by emergent synaptic clusters located in the motivational core of the brain (Lewis, 2015). This approach rests on a distinct phenomenology of perception in which addiction is not merely something that "happens in the brain" in response to exogenous stimuli (cf. Merleau-Ponty, 2013; Noë, 2009). Rather it is a process that unfolds in the subject's dynamic engagement with a socially structured and symbolically invested environment which, in turn, becomes durably imprinted in the neural underpinnings of the self. Such an ecological understanding of addiction weds the neuroscientific and the sociological within a single coherent framework wherein learning is indeed construed as the "selective, durable transformation of the body through the reinforcement or weakening of synaptic connections" (Bourdieu, 2000: 136).

It is against this conceptual backdrop that we articulate our empirical hypotheses. Our proposed framework helps us eschew a setup that divorces the study of "supply"- and "demand"-side factors by analytically privileging one over the other. We hypothesise that geographical variation in overdose mortality burdens can, at least in part, be explained by the interplay of local drug supply and socioeconomic disruptions. To operationalise this interplay, we study county-level opioid prescription rates on the one hand and a set of economic and political variables on the other. More specifically, on the "demand" side, we focus on two principal factors. First, following previous research linking social disruption to addictive behaviour (Alexander, 2008), we are interested in the effects of rapid economic change or decline in the form of falling household income and (un)employment relations. Second, we are interested in the historically unprecedented rise in incarceration rates and the impact this rise may have had on the overdose epidemic. We expect that economic decline in areas of concentrated disadvantage can account for the socially skewed distribution of psychosocial distress, while the historically unprecedented expansion of the penal apparatus is seen as part of a broader public policy repertoire through which attendant social divisions are curbed and controlled, managed and magnified. This dynamic, inscribed in the very political economy of American capitalism, forms an upstream determinant of intergenerationally transmitted adversity and trauma – or, to use Case and Deaton's term, "despair" – which in turn fosters addictive behaviour through psychological suffering and the loss of self-control.

This framework helps explain the geographical expression of the epidemic. One of the key features of American industrial decline, namely the reallocation of investment across economic sectors and the selective reterritorialisation of economic activity, is related to the geographical logic of capital accumulation, allowing corporations to combine the spatial dispersal of

production with the economic concentration of ownership in their pursuit of profit (Bluestone and Harrons, 1982). Subsequent changes in the criminal justice system have also followed a distinct sociospatial logic whereby area of poverty, such as Chicago's aforementioned territories of urban relegation, experience incarceration rates more than forty times higher than privileged White communities and three times higher than communities with a similar crime rate (Sampson and Loeffler, 2010: 27–28). For instance, in the wake of economic desolation, Detroit's deindustrialised wasteland has undergone a social transformation so profound that the number of its inhabitants under correctional supervision outweighs the number holding union jobs in the city's manufacturing plants (Thompson, 2010: 708).

The sociospatial dynamics of the American carceral state strongly suggests the need to study economic decline and penal expansion – and their potential role in the overdose epidemic – in tandem rather than in isolation from one another. A rich body of evidence ties incarceration to a variety of factors that are associated with drug overdoses and, more broadly, with inequalities in health (Link and Phelan, 1995), such as stigma, joblessness, family disruption, and neighbourhood decline (for a recent study, see Western [2018]). Nonetheless, very few existing studies have sought to link the over half a million drug-related deaths that have occurred over the past three and a half decades to the gargantuan expansion of the penal state since the early 1970s. The penal state, despite its unique place in American society, has remained conspicuously absent from research and policy debates surrounding "deaths of despair" and health inequality.

Our argument, in short, is twofold. On the one hand, we discard the binary division between "supply" and "demand" by spotlighting the joint effects of increasing opioid prescription rates and sociospatially concentrated material disadvantage. On the other hand, we extend current

debates by highlighting not only economic decline but also political responses to such decline. The literature on the health effects of resource shocks associated with deindustrialisation (Hopper et al., 1985; Hamilton et al., 1990; Wagner, 1991; Byrne, 1995; Aghion et al., 2016), trade liberalisation (Pierce and Schott, 2016; Barlow et al., 2017; McNamara, 2017), or broader labour market conditions is vast (see Avendano and Berkman [2014] for an overview). However, there are various possible welfare state responses to such resource shocks (e.g. Iversen and Cusack, 2000) that can moderate or magnify deleterious impacts (Stuckler and Basu, 2013). A distinctive feature of American social policy is its punitive mode of poverty regulation in the wake of economic decline, epitomised by high rates of incarceration (see Muller and Schrage, 2019; Wacquant, 2009). Thus, like Case and Deaton, we leverage insights from previous research to investigate the role of resource shocks – but such resource shocks must be viewed in tandem with the set of institutions that abet or abate broader structures of inequality. As demonstrated by previous sociological scholarship, the penal state has emerged as a key institution in this regard (Wacquant, 2009) and thus merits special analytic attention in the study of a population health crisis and its distributional effects.

DATA AND METHODS

We test our hypotheses using a two-pronged methodological approach involving both panel data and cross-sectional models. Our outcome variable is the natural logarithm of the age-standardised mortality rate from drug use disorders per 100,000 population for 2,925 US counties. We posit that $Y \sim \mathcal{N}(\mu_i, \sigma^2)$, where $\mu_i = \mathbb{E}(Y|X) = X\beta$, with *i* indexing individual counties. In this expression, *Y* is the outcome variable, assumed to be Normally distributed, its expectation being a function of the matrix of covariates *X* containing information on county-level retail opioid prescription rates dispensed per 100 persons, variation in median household income, rates of jail and prison incarceration, and a number of other variables, all of which are

listed and defined in Appendix Table A. Descriptive statistics are given in Appendix Tables B and B, and a correlation matrix is displayed in Appendix Table C.

In all our analyses, we use multiple imputation to account for missing data (Honaker, King, and Blackwell, 2011). Multiple imputation avoids the pitfalls of the default approach to dealing with missing data, namely listwise deletion, which typically leads to bias, inefficiency, or both (Blackwell, Honaker, and King, 2017). As compared to using listwise deletion, the use of multiple imputation expands our sample sises from 17,103 to 26,274 county-years in the panel-data analysis and from 1,891 to 2,925 counties in the cross-sectional analysis. Various diagnostics checks, including density comparisons and overimputation, reveal a plausible fit of the imputation model. We run each analysis separately on 10 multiply imputed data sets. We pool the resulting estimates and standard errors into a single set of parameters using Rubin's method (Rubin, 1987). For the sake of comparison, we present our panel model results with and without multiple imputation in the Appendix (Table E).

The key quantity of interest is the vector of parameters, β, associated with our predictors ("supply" and "demand"). We first exploit the temporal dimension of our data by estimating a two-way fixed-effects panel regression, the virtue of which is to eliminate both time-invariant confounders and secular trends that affect all places simultaneously. Within-county variation, over time in both independent and dependent variables, is typically much smaller than between-county variation. We therefore assess the set of plausible counterfactuals in relation to which our fixed-effects estimates might be interpreted (see Mummolo and Petersen, 2018). Despite the fact that fixed effects panel models better isolate variation for identification, we are nevertheless interested in the between-county variation, for several reasons. First of all, a sole focus on within-county variation over time prevents us from examining one of the principal

phenomena of sociological interest, namely the spatial expression of the overdose epidemic and the magnitude of disparity in mortality burdens across counties. Moreover, a cross-sectional analysis allows us to simulate key counterfactuals of interest and examine relative mortality differences in ways that are more intuitive than a fixed-effects approach. Calculating a variance partition coefficient shows that over 90% of the variation in prescription rates ("supply") is located between rather than within counties. Hence a time-series, fixed-effects approach effectively eliminates most of the information in the data and leads to potentially underpowered results. This further motivates the use of a cross-sectional design to isolate spatially embedded inequalities and not merely temporal variation in mortality rates. Finally, only a handful of covariates are observed for all years in our panel data. For the year 2014, however, we are not only in a position to adjust for important control variables but also to examine a significant number of associations of interest to social scientists using previously unavailable data (see Table A).

In the cross-sectional analysis, we adopt a simulation-based approach proposed by King and colleagues (2000). Simulation can be used to obtain useful information about a chosen probability distribution by drawing random numbers from it. Since we are interested in examining a number of counterfactual scenarios, simulating numbers from the sampling distribution of our parameter estimates allows us to integrate model uncertainty into our predicted values for each counterfactual. We note, in other words, that prediction intervals are not calculated the same way as standard confidence intervals, which motivates the use of simulation. Let Y designate the outcome variable of interest (drug-related mortality), let $T \in \{0, 1\}$ designate a "treatment" variable, and let X designate a series of control variables (education, crime, county demographics, etc.). By simulating from our model estimates, we obtain expected values of Y (drug-related mortality) for counties with different values of the

treatment *T* (which might denote "demand" or "supply"). These expected values are used to quantify the uncertainty surrounding the model parameters and to compare the distributions of $\mathbb{E}(Y \mid T = 0, X)$ and $\mathbb{E}(Y \mid T = 1, X)$. Throughout, the model is run with a continuous predictor, comparing counties with treatment values at one standard deviation below the mean ($T = 0$) to those at one standard deviation above the mean ($T = 1$). Further details of this method are described in the Appendix and in King et al. (2000).

We conduct a simple sensitivity analysis in which we assess the amount of unobserved confounding that would theoretically be required to explain away our estimated effect sises. Let $\hat{\beta} = \mathbb{E}(Y \mid T = 1, X) - \mathbb{E}(Y \mid T = 0, X)$ denote the expected difference in the outcome variable *Y* for $T = 1$ and $T = 0$, respectively, net of a matrix of controls *X*, and let *U* denote an unmeasured confounder. Then the *bias factor*, $\mathcal{B}$, is defined as the difference between $\hat{\beta}$ and what $\hat{\beta}$ *would have been* had we controlled for *U* as well. We make two simplifying assumptions: that *U* is binary and that the effect of *U* on *Y* is the same across both treatment states (i.e. no *U*-by-*T* interaction). Now define $\gamma = \mathbb{E}(Y \mid U = 1, T, X) - \mathbb{E}(Y \mid U = 0, T, X)$ as the effect of the unmeasured confounder on the outcome, net of the treatment and control variables, and define $\delta = \mathbb{P}(U = 1 \mid T = 1, X) - \mathbb{P}(U = 1 \mid T = 0, X)$ as the difference in the prevalence of the unmeasured confounder between the treatment and control groups. Under the two simplifying assumptions given above, the bias factor is readily obtained as the product of these two parameters: $\mathcal{B} = \gamma \cdot \delta$ (VanderWeele, 2015: 68–69). In assessing the sensitivity of our model coefficients to such unmeasured confounding, we vary these two parameters across a range of possible values to obtain a plausible range of corrected estimates.

FINDINGS

We begin to test our hypotheses by specifying the regression model displayed in Table 1. To identify plausible shifts in our key predictors wherefrom relevant counterfactual scenarios are derived, we isolate within-county variation in each variable by residualising that variable with respect to the fixed effects (Mummolo and Petersen, 2018). We then calculate the distribution of the within-county range in each "treatment" and use the average of this range to produce the presented coefficients. The latter are thus interpretable as semi-elasticities, i.e. the percentage change in drug-related mortality rates associated with the average within-county variation for a given predictor. Since high school graduation rates and the ethno-racial composition of counties are nearly time-invariant and only marginally contribute to the parameter estimation, we omit the corresponding coefficients from Table 1. Histograms of within-county variation for each predictor and its corresponding mean are plotted in Appendix Figure A for one randomly chosen multiply imputed data set. Pooled regression results for the imputed as well as non-imputed data using the original untransformed variables are presented in Appendix Table E.

The results suggest that opioid prescription rates are significantly associated with mortality rates from drug use disorders. Substantively, the average within-county variation in "supply" is associated with a 0.287% increase in drug deaths (95% CI: 0.0935, 0.480; $P < 0.01$). This is only about one-third of the effect of prison incarceration ($\hat{\beta}$ = 1.01%; 95% CI: 0.734, 1.29; $P < 0.001$), and less than one-tenth of the effect of variation in household income ($\hat{\beta}$ = -4.00%; 95% CI: -4.29, -3.71; $P < 0.001$). We note that the effect of unemployment is statistically insignificant, as is that of jail incarceration (once we impute for missing data).

[Table 1 about here.]

We proceed to the cross-sectional analysis, displayed in Table 2, in which we specify a baseline regression model based on the following covariates: opioid prescription rates, decline in median household income between 2006 and 2014, the labour force participation rate, jail and prison incarceration rates, the violent crime rate, absolute income mobility, racial segregation, the high school graduation rate, the ethno-racial composition of counties, as well as urbanicity status. We also include state fixed effects, which help account for state-specific confounders, including legislation pertaining to drug "supply" or long-run institutional configurations. We derive this preferred baseline specification on the basis of model fit and a number of sensitivity checks. In Appendix Figure B, we show that this baseline model is robust to additional adjustments for a range of covariates, including income levels and inequality, import competition, and the population percentage without health insurance. These additional controls have no noticeable effect on our baseline parameter estimates. However, due to what we suspect to be multicollinearity bias, especially in the case of our economic variables, a number of our control variable coefficients have unexpected signs, notably those for poverty rates and income inequality. We therefore also present single-variable regressions in Appendix Table F, in which the outcome variable is regressed on each of the aforementioned regressors, one at a time, while only controlling for state fixed effects. This serves the purpose of avoiding over-specification and seems to confirm our suspicion of collinearity problems in the "long" regressions. We therefore proceed with the more parsimonious but robust baseline specification.

We see that nearly all coefficients are somewhat attenuated after adjusting for covariates, with the notable exception of the labour force participation rate, which becomes more strongly associated with the outcome in Table 2 as compared to Appendix Table F. This suggests that its link to drug mortality might be moderated by one of the remaining predictors. In both

models, there is a strong association between opioid prescription rates and drug-related mortality. However, on the "demand" side, a number of predictors also exhibit strong associations, including economic decline and incarceration rates, but also labour force participation, absolute income mobility, or racial segregation. Somewhat interestingly, we find that urban areas experience significantly higher rates of mortality from drug use disorders than rural areas, although there is reason to believe that such associations shroud significant heterogeneity with respect with to different kinds of drug use disorders (see Monnat et al., 2019). We also find that the associations between unemployment and import competition with the outcome disappear once other covariates are included, although increasing import competition between 1999 and 2011 retains significance at $P = 0.027$. The interaction term, though statistically significant in Appendix Table F and indicative of an important dynamic, is likely to be underpowered and becomes insignificant in the multivariable regression. We therefore leave it out of the subsequent analysis. Finally, as the variables measuring the ethno-racial composition of counties are log-transformed, corresponding coefficients are interpreted as elasticities, i.e. as the percentage change in the outcome associated with a 1% change in the predictor. As such, we find that counties with higher proportions of African American and Hispanic populations have lower mortality rates from drug use disorders, whereas areas with a higher proportion of other ethnic minorities appear to experience higher mortality rates. However, given the aggregated nature of our data, the interpretation of such estimates warrants great caution.

[Table 2 about here.]

By drawing simulated values from the parameter estimates in Table 2, we proceed to estimating expected mortality differences between counties based on a number of key counterfactuals, as

described in Table 3. Based on the findings of Table 2, we combine income decline, mobility, labour force participation, and incarceration rates to assess the effects of "demand". To isolate the "supply" effect, we first compare counties that have low levels of "demand" – characterised by high rates of labour force participation, high income mobility, low rates of incarceration, and low levels of income decline between 2006 and 2014 – and low versus high rates of opioid prescription (all other covariates in our model held constant at their mean). As shown in the first row of Table 3, we find that a standard-deviation shift in prescription rates alone is expected to increase the drug-related mortality rate by 18.4% (95% CI: 16.1, 20.7; $P < 0.001$). In the second scenario, we hold prescription rates constant at a low level and assess the effect of a shift from low to high levels of "demand," finding, as shown in the second row, an increase in the log-mortality rate of 48.5% (95% CI: 41.6, 55.4; $P < 0.001$). To assess the combination of high "supply" and high "demand," we compare low to high levels of both "supply" and "demand". This yields an expected increase in drug deaths of 66.9% (95% CI: 59.3, 74.5; $P < 0.001$).

[Table 3 about here.]

QUANTIFYING ENDOGENEITY BIAS: A SENSITIVITY ANALYSIS

Let *U* denote a binary measure of drug environment, say "toxic" drug environment versus "healthy" drug environment, that is not captured by our data on opioid prescription rates. The scenario of greatest interest would be where *U* designates environments with a high supply of synthetic opioids, such as fentanyl, or other illicit drugs such as heroin. Given that *U* is unmeasured, we know neither the distribution of *U* across various strata of our data nor the potential effect of *U* on our outcome variable. However, we may visually inspect the variation

of the bias factor $\mathcal{B}$, as defined earlier, across a range of possible values of the two sensitivity parameters δ and γ.

Using the estimated effect of "demand" on drug-related mortality rates as per Table 3, Figure 1 shows a plot of these two sensitivity parameters, with δ denoting the degree of selection on the unmeasured confounder across the two treatment states (ranging from 0 to 1, with higher values indicating a higher prevalence of the confounder in the treatment group, i.e. in counties with high levels of "demand"), and γ denoting the magnitude of the effect of U on the outcome, above and beyond that of the treatment and other controls, that would be required to completely eliminate the effect of "demand" on mortality rates from drug use disorders. The reader will note that even for unusually high levels of selection on the unmeasured confounder, the effect of U on the outcome would have to be substantially larger than any of the effects derived from our own model to nullify that of "demand". For instance, even when the difference in the prevalence of the confounder between the treatment and control groups is as high as 0.9 – a highly unlikely scenario – γ would have to exceed 50% to eliminate our model estimate. A more plausible value of δ would be at the lower end of the X-axis in Figure 1. At, say, $\delta = 0.2$, the effect of U on Y would have to be nearly 250%, which seems highly improbable (if not impossible). For the sake of argument, assume that $\mathcal{B} = 20\%$. Then the adjusted effect of "demand" would still be 28.5% (95% CI: 21.6, 35.4; $P < 0.001$), which is substantially higher than that of "supply" alone.

CONCLUSION

The political economy of industrial decline and of subsequent changes to the criminal justice system provides an important addition to current debates surrounding America's overdose epidemic. Our statistical analysis supports the view that differences in opioid prescription rates

play an important role in accounting for substantial spatial variation in overdose mortality rates – but it also spotlights the potentially major impact of downward economic mobility and punitive policy responses thereto. Our analyses provide a series of parsimonious model specifications that suggest a robust set of associations and our sensitivity checks suggest that an inordinate amount of unmeasured confounding would be needed nullify our findings, which indicate that ending the overdose epidemic requires not just a crack-down on unethical and illegal activities by corporations to increase the sale of opioid pain medications: areas with serious declines in household incomes, low labour force participation rates, and elevated incarceration rates need policies that shift their focus from (punitive) correction to (social) protection.

We do, of course, refrain from making any causal claims. As in any observational study, our results may in part suffer from issues of endogeneity related to omitted variable bias. Our analysis relies on imperfect measures of the variables of interest. For instance, we use a number of proxy variables – e.g. changes in median household income – to capture economic decline, and although we argue this is a robust and meaningful measure of the phenomenon at hand, we are unable to measure other facets of the economic processes outlined in our framework, chief amongst them job destruction rates in manufacturing or rates of unionisation. In investigating punitive policy responses to economic decline, we are only able to examine incarceration rates which, although a prominent feature of the policy landscape, constitute but one facet of a broader public policy repertoire that impacts on the social and spatial patterning of overdose mortality. Our ecological approach precludes any microscopic investigation of individuals or local communities, and our focus on opioid prescription rates precludes any broader account of drug environment or availability. Nonetheless, our data remain of high quality, including a number of previously unavailable measures at the county level such local incarceration rates

and absolute income mobility. Our analysis allows us to explicitly quantify a number of key counterfactuals in which both researchers and policy-makers are interested, but which are rarely estimated and quantified. In doing so, we deploy a sociological toolkit to shed light on a phenomenon that typically falls outside the analytic purview of sociologists, namely inequalities in health and wellbeing. Our approach thus showcases the contribution that sociology can make to tackling an ongoing population health crisis.

ENDNOTES

1. https://www.drugabuse.gov/drugs-abuse/opioids/opioid-overdose-crisis

2. https://www.cdc.gov/drugoverdose/prevention/index.html

3. A synapse is a specialised gap region between two apposing cell membranes that permits a nerve cell to transmit an electrical or chemical signal.

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TABLE 1: PANEL REGRESSION RESULTS

Predictor	Average within-county range of predictor	Parameter estimate
Opioid prescription rate	41.8 prescriptions per 100 persons	**0.287%** (0.0987) **
Household income	$5,860	**-4.00%** (0.1490) ***
Unemployment rate	2.84%	**-0.0107%** (0.1250)
Jail incarceration	5,605 admissions per 100,000 population	**0.1450%** (0.1160)
Prison incarceration	259 admissions per 100,000 population	**1.010%** (0.1410) ***
Violent crime rate	245 offenses per 100,000 population	**0.761%** (0.1250) ***

Notes: Two-way fixed-effects panel regression using ten multiply imputed data sets; the outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; the main predictors (listed in the first column) are the county opioid prescription rate, median household income, the unemployment rate, jail and prison incarceration rates, and violent crime rates; the model is adjusted for aggregate time trends using year dummies and it further controls for the county fraction of high school graduates, African Americans (log-transformed), Hispanics (log-transformed), or other non-White ethnicity (log-transformed); since the latter controls are nearly time-invariant and contribute little or nothing to estimating the model, they are omitted from this table; coefficients (listed in the final column) are interpreted as the percentage change in the drug-related mortality rate associated with the average within-county variation in each predictor (as listed in the second column); panel-corrected standard errors (robust to serial auto-correlation and heteroskedasticity) are shown in parentheses; $N = 26{,}274$; adjusted $R^2 = 71.7\%$; $^*P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$.

TABLE 2: CROSS-SECTIONAL MULTIVARIABLE REGRESSION RESULTS

Predictor	Parameter estimate
Opioid prescription rate	**14.4%** (0.835) ***
Household income decline	**5.74%** (0.799) ***
Labour force participation rate	**-13.2%** (1.03) ***
Absolute income mobility	**9.75%** (1.45) ***
Jail incarceration	**2.87%** (0.805) ***
Prison incarceration	**6.33%** (0.926) ***
Violent crime rate	**2.05%** (0.921) *
High school graduation rate	**-0.0624%** (1.06)
Racial segregation	**3.70%** (0.944) ***
Fraction African Americans	**-0.125%** (0.00951) ***
Fraction Hispanics	**-0.0359%** (0.0121) **
Fraction other ethnic minority	**0.0487%** (0.0124) ***
Small/mid (ref. rural)	**9.46%** (1.77) ***
Suburban (ref. rural)	**19.3%** (2.46) ***
Urban (ref. rural)	**33.2%** (5.01) ***

Notes: Maximum likelihood cross-sectional multivariable regression using ten multiply imputed data sets; the outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; the model includes state fixed effects (not displayed); all continuous predictors are standardised by subtracting the mean and dividing by the standard deviation, except for the demographic composition variables which are simply log-transformed; coefficients, displayed in the second column, are interpreted as semi-elasticities, except for the demographic composition variables which represent elasticities; standard errors are shown in parentheses; $N = 2{,}925$; AIC = 1,548; $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$.

TABLE 3: SIMULATED COUNTERFACTUALS

Scenario	Counterfactual	Expected change in log-mortality
Low supply, low demand	High supply, low demand	**18.4%** (1.15)
Low supply, low demand	Low supply, high demand	**48.5%** (3.54)
Low supply, low demand	High supply, high demand	**66.9%** (3.88)

Notes: The outcome variable is the natural logarithm the age-standardised mortality rate from drug use disorders; the model compares counties with "treatment" values at one standard deviation below the mean ("low") to those at one standard deviation above the mean ("high"); "supply" is measured using opioid prescription rates; "demand" is measured using household income decline, labour force participation rates, absolute income mobility, and incarceration rates; the third column shows the expected percentage change in the log-mortality rate from drug use disorders associated with each counterfactual described in the first two columns; the simulated values for mortality rates are derived from the parameter estimates displayed in Table 2; standard errors are shown in parentheses; all parameter estimates are statistically significant at $P < 0.001$.

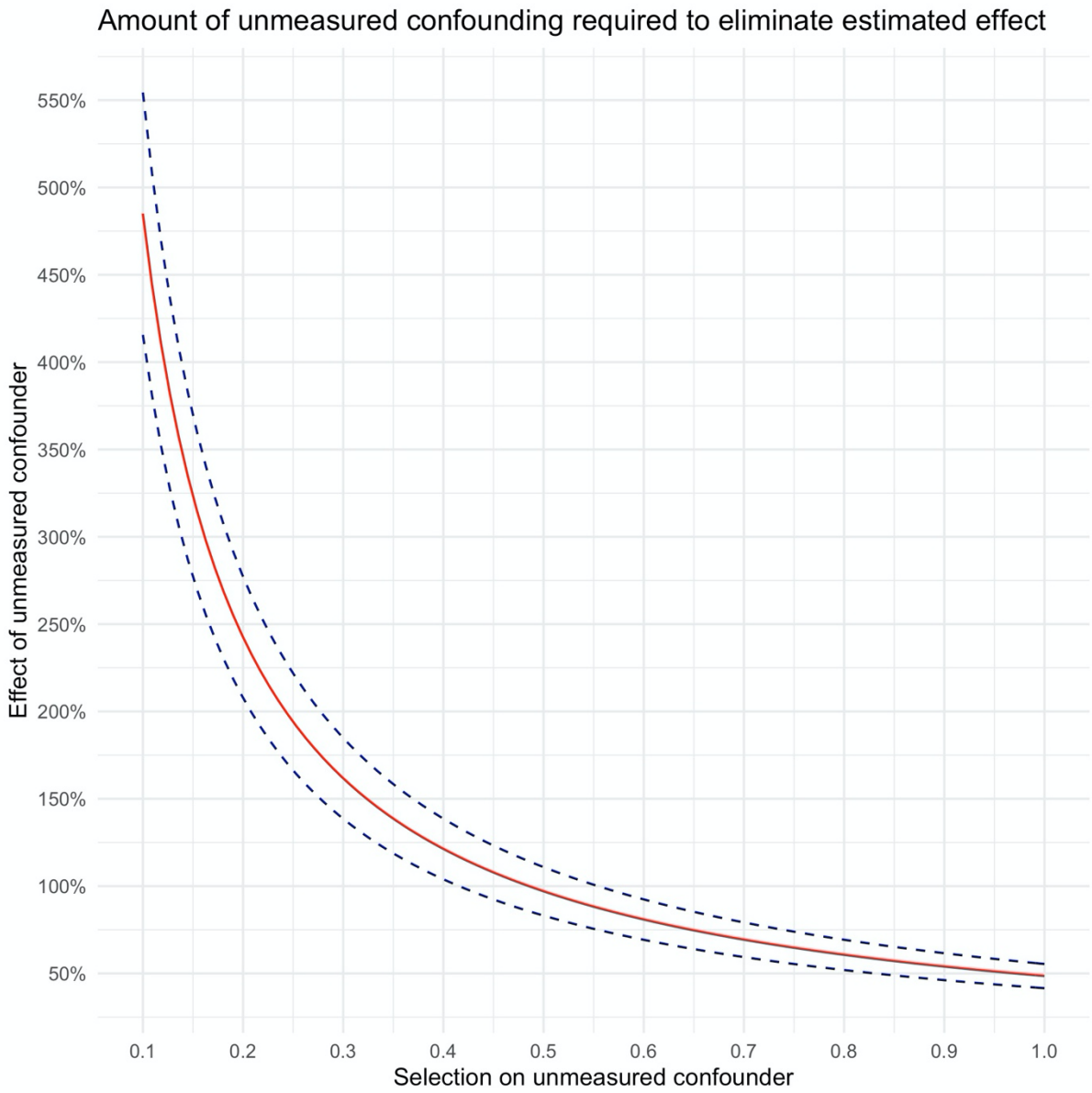

Figure 1: Sensitivity analysis plot for the estimated effect of "demand" as per Table 3; values of δ (X-axis) and γ (Y-axis) that lie on the solid red line would completely eliminate the effect of "demand"; the dotted blue lines denote the lower and upper bounds of the corresponding 95% confidence interval; values above the plotted curve would reverse the sign of the effect of "demand".

APPENDIX

Simulating counterfactuals

We treat our model parameter estimates as features of their underlying sampling distribution. By the central limit theorem, the set of all model parameter estimates, represented by the stacked column vector $\hat{\theta} = \{\hat{\beta}, \hat{\sigma^2}\}$, follows a multivariate normal distribution with mean equal to the model estimates and variance equal to the model variance-covariance matrix, $\hat{V}(\hat{\theta})$. More formally, the distribution from which a simulated parameter value, $\tilde{\theta}$, is drawn is represented as follows:

$$\tilde{\theta} \sim N(\hat{\theta}, \hat{V}[\hat{\theta}]).$$

Now let *Y* designate the outcome variable of interest (drug-related mortality), let $T \in \{0, 1\}$ designate a "treatment" variable, and let *X* designate a series of control variables (education, crime, county demographics, etc.). To obtain expected values of *Y* (drug-related mortality) for counties with different values of the treatment, *T*, we do the following (for further details, see King et al., 2000):

1. We estimate our log-likelihood function as described above, regressing *Y* on *T* and *X*.
2. We simulate from the sampling distribution of the parameter estimates to incorporate estimation uncertainty by drawing *M* random numbers from the multivariate normal distribution.
3. For each simulated parameter value, we calculate the systematic component of the model, $\tilde{\mu}_i = T\tilde{\beta} + X\tilde{\gamma}$ (where $\hat{\gamma}$ is the vector of parameter estimates corresponding to the matrix of control variables).

4. For each simulated systematic component, we draw another *m* separate random draws of the outcome variable, $\tilde{y}_k$ $(k = 1, \ldots, m)$, from the stochastic model component (the Normal distribution *N*) to incorporate additional stochastic uncertainty.
5. For each of the *M* sets of *m* simulated values, we average over the stochastic uncertainty by computing the mean of the *m* simulations, thus obtaining one vector of *M* expected values:

$$\mathbb{E}(Y \mid T, X) = \frac{1}{m} \sum_{k=1}^{m} \tilde{y}_k.$$

These expected values are used to quantify the uncertainty surrounding the model parameters and to compare the distributions of $\mathbb{E}(Y \mid T = 0, X)$ and $\mathbb{E}(Y \mid T = 1, X)$. Throughout, the model is run with a continuous predictor, comparing counties with treatment values at one standard deviation below the mean ($T = 0$) to those at one standard deviation above the mean ($T = 1$). The above steps are done for $T = 0$ and for $T = 1$ separately before differences in the expected values of *Y* are examined. The number of simulations is set to $M = m = 100{,}000$.

TABLE A: VARIABLE DEFINITIONS AND SOURCES

Variable	Definition	Source
Mortality from drug use disorders	Age-standardised mortality rate from drug use disorders per 100,000 county population between 2006 and 2014.	US National Vital Statistics System via Institute for Health Metrics and Evaluation.
Opioid prescription rate	Retail opioid prescription rates dispensed per 100 persons between 2006 and 2014.	Centers for Disease Control and Prevention.
Median household income	Median county household income, measured in constant US dollars, between 2006 and 2014.	US Census Bureau.
Decline in median household income	Negative change in median county household income between 2006 and 2014 (derived from the previous variable).	US Census Bureau.
Unemployment rate	Unemployment rate per active county population between 2006 and 2014.	US Bureau of Labor Statistics.
Jail incarceration rate	Admissions rate to county jails per 100,000 county population aged 15–64 between 2006 and 2014.	Vera Institute of Justice.
Prison incarceration rate	Admissions rate to state prisons per 100,000 county population aged 15–64 between 2006 and 2014.	Vera Institute of Justice.
Violent crime rate	Rate of violent crime per 100,000 county population between 2006 and 2014.	Federal Bureau of Investigation.
High school graduation rate	Fraction of county population with a high school diploma between 2006 and 2014.	US Census Bureau.
Fraction African Americans	Fraction of county population who are African American between 2006 and 2014.	US Census Bureau.
Fraction Hispanics	Fraction of county population who are Hispanic between 2006 and 2014.	US Census Bureau.
Fraction other ethnicity	Fraction of county population who are other ethnic minority between 2006 and 2014.	US Census Bureau.
Poverty rate	County-level rate of poverty, as per federal poverty line, in 2014.	Opportunity Insights.
Top 1% income share	Share of total income going to the top 1% of the income distribution.	Opportunity Insights.
Income inequality	County-level Gini index amongst bottom 99% of the income distribution.	Opportunity Insights.
Income segregation	Spatial segregation of income groups at the county level in 2014.	Opportunity Insights.
Racial segregation	County-level spatial segregation by race in 2014.	Opportunity Insights.
Labour force participation rate	County-level labour force participation rate in 2014.	Opportunity Insights.
Import competition	Instrumented exposure to import competition at the commuting-sone level in 2011.	Autor, Dorn, and Hanson (2013).
Change in import competition	Change in instrumented import competition at the commuting-sone level between 1999 and 2011.	Autor, Dorn, and Hanson (2013).
Absolute income mobility	County-level fraction of children who earn more than their parents in 2014.	Opportunity Insights.
Percentage uninsured	Percentage of county population without health insurance.	Opportunity Insights.
Urbanicity	Indicator variable distinguishing rural, small/mid-sised metropolitan area, suburban, and urban counties.	Vera Institute of Justice.

TABLE B: DESCRIPTIVE STATISTICS (PANEL DATA, 2006–2014)

Statistic	N	Mean	St. Dev.	Min	Max
Mortality from drug use disorders	26,274	9.0	5.3	1.5	60.8
Prescription rate	23,352	88.9	45.8	0.0	437.2
Household income	26,274	46,666	11,402	19,946	118,335
Unemployment rate	26,267	7.2	3.1	1.1	28.9
Jail incarceration rate	20,670	7,622	3,769	20.2	18,444
Prison incarceration rate	19,907	310.0	166.1	14.1	732.1
Crime rate	23,105	252.6	215.3	0.0	2,688
High school graduation rate	26,274	0.8	0.1	0.4	1.0
Fraction African Americans	26,274	0.1	0.1	0.000	0.9
Fraction Hispanics	26,274	0.1	0.1	0.001	1.0
Fraction other ethnic minority	26,274	0.03	0.1	0.001	0.9

TABLE C: DESCRIPTIVE STATISTICS (CROSS-SECTIONAL DATA, 2014)

Statistic	N	Mean	St. Dev.	Min	Max
Mortality from drug use disorders	2,925	10.0	6.1	1.6	57.1
Prescription rate	2,775	85.2	46.3	0.0	300.1
Household income level	2,925	46,534	11,400	21,658	108,477
Decline in household income	2,925	2,020	2,471	0.0	16,752
Poverty rate	2,925	0.1	0.1	0.02	0.6
Unemployment rate	2,925	0.06	0.02	0.01	0.24
Labour force participation rate	2,925	0.6	0.1	0.3	0.9
Import competition	2,924	9.7	5.8	0.000	46.4
Change in import competition	2,925	1.7	1.5	-0.1	9.4
Jail incarceration rate	2,234	6,994	3,791	20.2	18,253
Prison incarceration rate	2,117	312.9	165.1	21.9	731.8
Crime rate	2,627	234.5	194.1	0.0	2,568
High school graduation rate	2,925	0.9	0.1	0.5	1.0
Income inequality	2,832	0.4	0.1	0.2	1.1
Income segregation	2,925	0.03	0.03	-0.01	0.4
Top 1% income share	2,832	0.1	0.1	0.02	0.7
Absolute income mobility	2,675	47.5	6.1	25.1	68.3
Percentage uninsured	2,925	18.6	5.6	3.6	41.4
Racial segregation	2,925	0.1	0.1	0.0	0.7
Fraction African Americans	2,925	0.1	0.1	0.001	0.9
Fraction Hispanics	2,925	0.1	0.1	0.002	1.0
Fraction other ethnic minority	2,925	0.04	0.1	0.001	0.9

TABLE D: CORRELATION MATRIX (CROSS-SECTIONAL DATA, 2014)

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.
1. Mortality from drug use disorders	1																					
2. Prescription rate	0.5	1																				
3. Household income level	-0.3	-0.3	1																			
4. Decline in household income	0.3	0.2	-0.1	1																		
5. Poverty rate	0.2	0.2	-0.8	-0.2	1																	
6. Unemployment rate	0.5	0.3	-0.6	0.4	0.4	1																
7. Labour force participation rate	-0.3	-0.2	0.7	0.1	-0.7	-0.5	1															
8. Import competition	0.2	0.2	-0.2	0.2	0.2	0.3	-0.1	1														
9. Change in import competition	0.2	0.2	-0.2	0.3	0.1	0.4	-0.05	0.8	1													
10. Jail incarceration rate	0.3	0.3	-0.3	0.2	0.4	0.3	-0.3	0.2	0.1	1												
11. Prison incarceration rate	0.2	0.2	0.3	0.3	-0.2	0.1	0.3	0.1	0.1	0.4	1											
12. Crime rate	0.1	0.2	0.1	0.2	-0.03	0.03	0.1	0.1	0.1	0.7	0.4	1										
13. High school graduation rate	-0.4	-0.3	0.4	-0.4	-0.4	-0.6	0.3	-0.3	-0.3	-0.6	-0.4	-0.3	1									
14. Income inequality	0.2	0.3	-0.2	0.02	0.2	0.1	-0.1	0.04	0.03	0.2	-0.1	-0.02	-0.1	1								
15. Income segregation	0.3	0.3	-0.4	-0.1	0.4	0.1	-0.3	0.1	0.1	0.2	-0.1	-0.01	-0.2	0.3	1							
16. Top 1% income share	0.3	0.3	-0.1	0.2	0.2	0.2	-0.1	0.1	0.05	0.4	0.4	0.2	-0.4	0.2	0.2	1						
17. Absolute income mobility	-0.3	-0.2	0.6	-0.1	-0.7	-0.5	0.6	-0.3	-0.2	-0.4	0.2	0.001	0.4	-0.2	-0.4	-0.2	1					
18. Percentage uninsured	0.2	0.1	-0.5	-0.1	0.6	0.2	-0.5	0.1	-0.1	0.4	-0.2	0.01	-0.3	0.3	0.4	0.2	-0.6	1				
19. Racial segregation	0.2	0.2	0.03	0.3	0.04	0.2	0.02	0.1	0.2	0.4	0.7	0.3	-0.4	-0.03	0.1	0.4	-0.1	-0.1	1			
20. Fraction African Americans	0.2	0.2	-0.2	0.3	0.3	0.3	-0.2	0.3	0.3	0.5	0.4	0.3	-0.7	0.1	0.2	0.5	-0.4	0.2	0.5	1		
21. Fraction Hispanics	-0.02	-0.1	0.2	0.05	-0.03	-0.2	0.1	-0.1	-0.2	0.2	0.2	0.2	-0.03	0.1	-0.01	0.3	-0.1	0.4	0.2	0.2	1	
22. Fraction other ethnic minority	0.01	-0.1	0.3	0.1	-0.1	-0.2	0.2	-0.1	-0.3	0.2	0.4	0.2	-0.03	0.03	-0.1	0.3	0.2	0.1	0.3	0.1	0.6	1

TABLE E: UNMODIFIED PANEL REGRESSION RESULTS

	Parameter estimate (imputed data)	**Parameter estimate** (non-imputed data)
Opioid prescription rate	**0.0069%** (0.00002, 0.00012) **	**0.000085%** (-0.000023, 0.00019)
Household income	**-0.0000068%** (-0.0000073, -0.000006.3) ***	**-0.0000061%** (-0.0000066, -0.0000056) ***
Unemployment rate	**-0.000038%** (-0.00090, 0.00083)	**0.00016%** (-0.0010, 0.0014)
Jail incarceration	**0.00000026%** (-0.00000015, 0.00000066)	**0.0000013%** (0.00000059, 0.0000020) ***
Prison incarceration	**0.000039%** (0.000029, 0.000050) ***	**0.00010%** (0.000082, 0.00012) ***
Violent crime rate	**0.000031%** (0.000021, 0.000041) ***	**0.000054%** (0.000038, 0.000069) ***
	N = 26,274; adjusted R^2 = 71.7%.	N = 17,103; adjusted R^2 = 69.2%.

Notes: Two-way fixed-effects panel regressions; the outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; the main predictors are the county opioid prescription rate, median household income, unemployment rate, jail and prison incarceration rates, and violent crime rates; the model is adjusted for aggregate time trends using year dummies and it further controls for the county fraction of high school graduates, African Americans (log-transformed), Hispanics (log-transformed), or other non-White ethnicity (log-transformed); since the latter controls are nearly time-invariant and contribute little or nothing to estimating the model, they are omitted from this table; coefficients are interpreted as the percentage change in the drug-related mortality rate associated with a one-unit increase in each predictor; the first column shows coefficients derived from the imputed data, whereas the second column shows results for the non-imputed data; 95% confidence intervals, derived from panel-corrected standard errors (robust to serial auto-correlation and heteroskedasticity), are shown in parentheses; *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

TABLE F: CROSS-SECTIONAL SINGLE-VARIABLE REGRESSION RESULTS

Predictor	Parameter estimate	AIC
Opioid prescription rate	**17.6%** (0.763) ***	2,269
Household income level	**-10.9%** (0.825) ***	2,611
Household income decline	**6.44%** (0.849) ***	2,725
Poverty rate	**5.50%** (0.878) ***	2,744
Unemployment rate	**13.0%** (0.957) ***	2,602
Labour force participation rate	**-9.54%** (0.823) ***	2,650
Import competition	**2.10%** (0.841) *	2,777
Change in import competition	**2.83%** (0.93) **	2,774
Jail incarceration	**7.67%** (0.872) ***	2,685
Prison incarceration	**10.8%** (0.891) ***	2,618
Violent crime rate	**6.88%** (0.824) ***	2,697
High school graduation rate	**-4.02%** (0.913) ***	2,763
Income inequality	**7.25%** (0.867) ***	2,709
Top 1% income share	**1.05%** (0.756)	2,781
Income segregation	**2.94%** (0.761) ***	2,768
Absolute income mobility	**-16.3%** (1.05) ***	2,548
Percentage uninsured	**1.20%** (1.29)	2,782
Racial segregation	**6.68%** (0.767) ***	2,707
Fraction African Americans	**-0.0182%** (0.00821) *	2,778
Fraction Hispanics	**-0.0271%** (0.0104) **	2,776
Fraction other ethnic minority	**0.0843%** (0.00992) ***	2,710
Small/mid (v. rural)	**0.160%** (1.96)	2,230
Suburban (v. rural)	**0.691%** (2.65)	2,230
Urban (v. rural)	**14.8%** (5.41) **	2,230
Opioid prescription rate × household income decline	**2.14%** (0.808) **	2,215

Notes: Maximum likelihood cross-sectional regressions using ten multiply imputed data sets; the outcome variable is the natural logarithm of the age-standardised county mortality rate from drug use disorders; all models include state fixed effects (not displayed); each cell represents a separate regression of the outcome variable on the listed predictor, controlling only for state fixed effects; the third column displays model fit using the Aikake information criterion (AIC); all continuous predictors are standardised by subtracting the mean and dividing by the standard deviation, except for the demographic composition variables which are simply log-transformed; coefficients, displayed in the second and third columns, are interpreted as semi-elasticities, except for the demographic

composition variables which represent elasticities; 95% confidence intervals are shown in parentheses; $N = 2{,}925$; $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$.

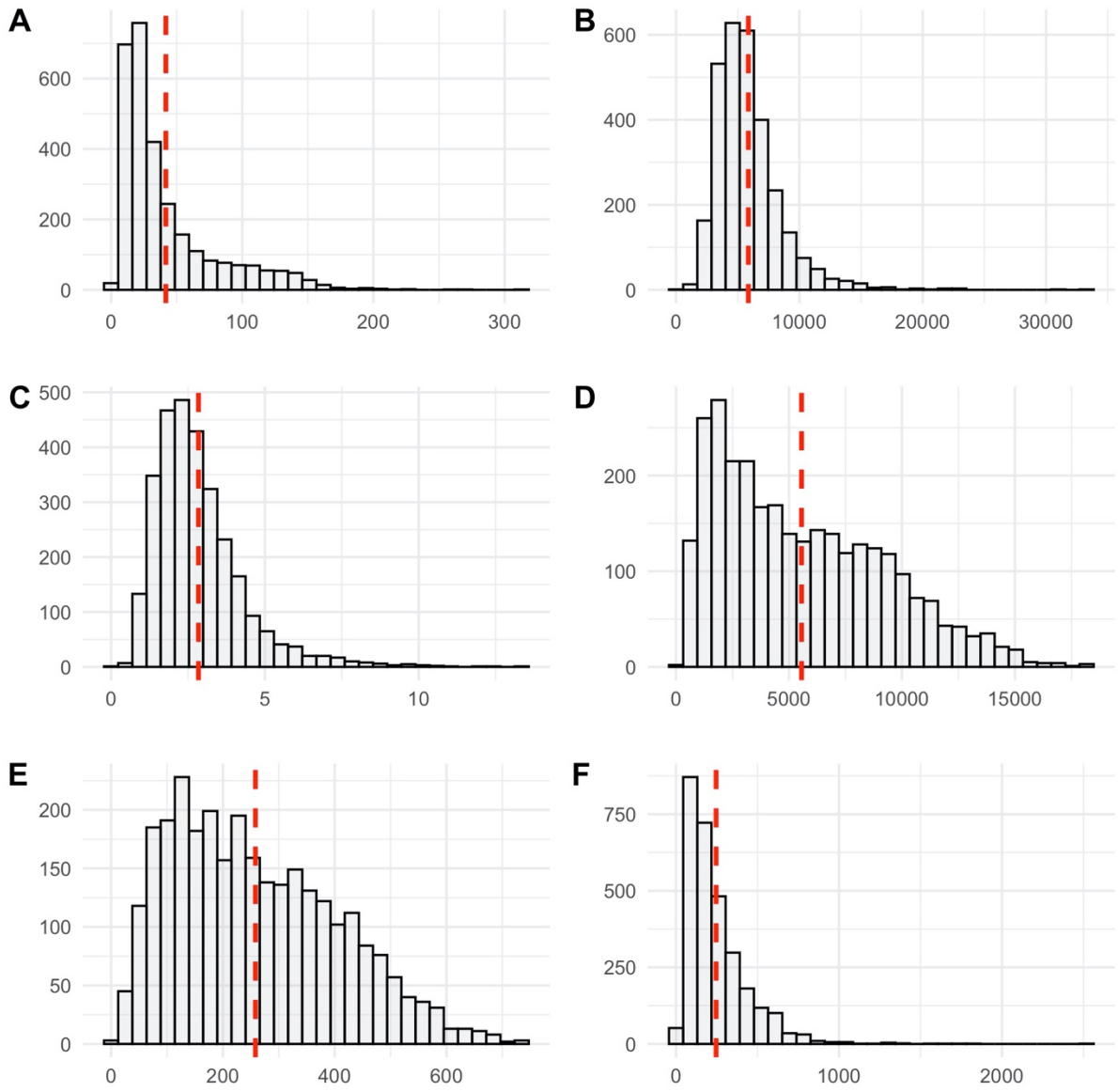

Figure A: Histograms of within-county variation in fixed-effects model predictors from one randomly chosen multiply imputed data set; the X-axis represents the within-unit range of each predictor; the Y-axis represents the total count within each bin; the dotted red line shows the mean within-unit variation for each predictor that is used to interpret regression coefficients in Table 3; A = opioid prescription rate; B = median household income; C = unemployment rate; D = jail incarceration rate; E = prison incarceration rate; F = violent crime rate.

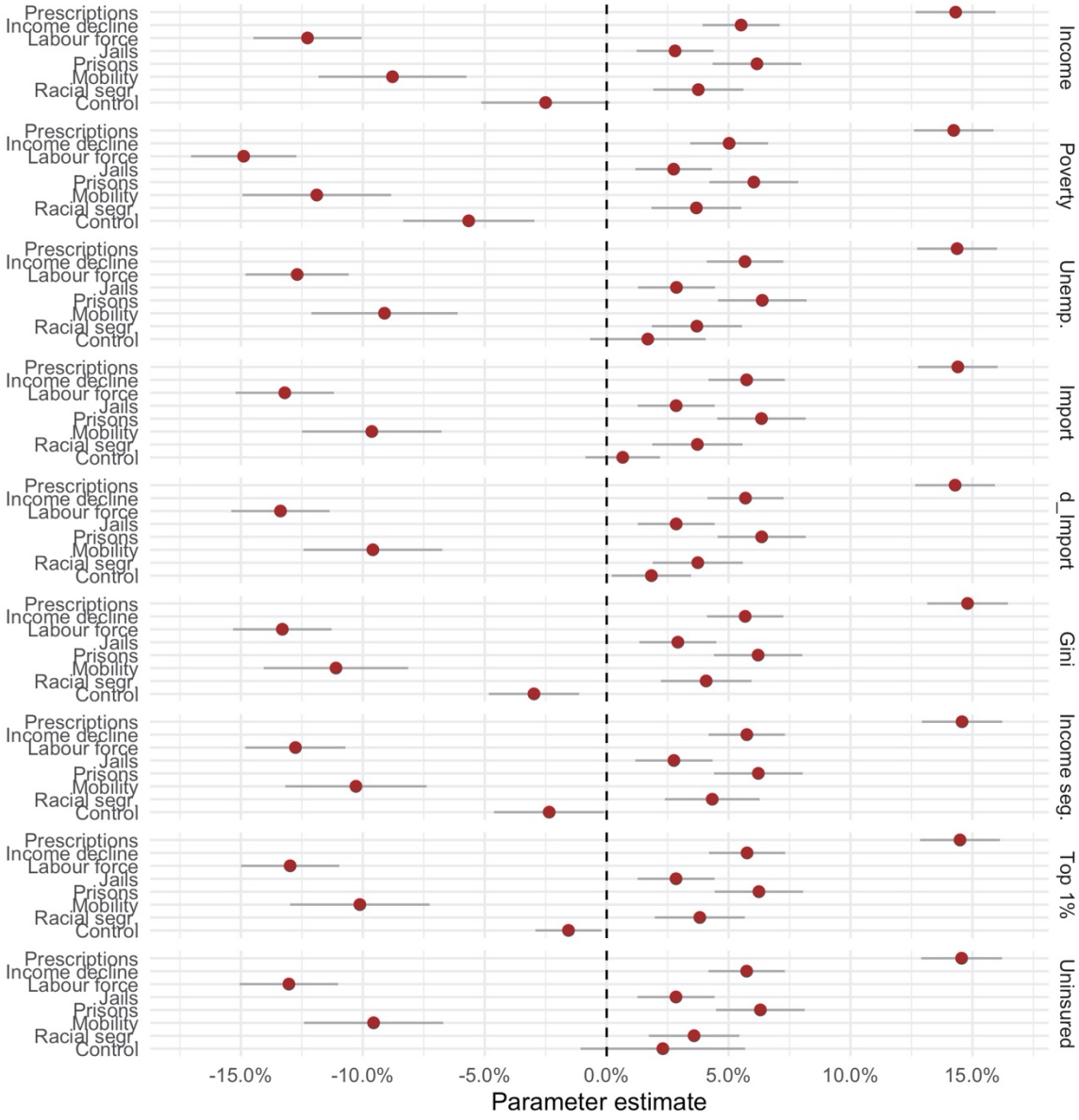

Figure B: Coefficient plot for sensitivity checks; each panel shows the parameter estimates for the baseline model (as per Table 2) adjusted for the control variable whose label appears on the right hand side; control variables are added and removed one by one; all models also control for violent crime rates, high school graduation rates, and the ethno-racial composition of counties, in addition to urbanicity status and state fixed effects (not displayed); "Income" = household income level; "Poverty" = poverty rate; "Unemp." = unemployment rate; "Import" = import competition; "d_Import" = change in import competition 1999–2011; "Gini" = Gini index in bottom 99% of income distribution; "Income seg." = income segregation; "Top 1%" = top 1% income share; "Uninsured" = percentage uninsured.