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THICK MARKET EXTERNALITIES AND THE PERSISTENCE OF THE OPIOID EPIDEMIC

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ABSTRACT

Opioid overdose death rates in the United States have risen continuously for over three decades, increasing 2,142 percent in total from 1990 to 2020. This is surprising. One might expect drug epidemics to be self-limiting, as policy and individual behavior reacts to observed deaths. We study why opioid deaths have risen so greatly and for so long. We consider three reasons for a prolonged epidemic: exogenous and continuing changes in demand or supply, and spillovers in demand for opioids across users, which we term "thick market externalities." We show there is no evidence of sufficiently large exogenous changes in the demand or supply of opioids that could explain such a prolonged increase in death rates. We test for spillovers using county-level data on opioid deaths from 1991–2018 and opioid shipments from 2006–2009, combined with data on friendships and distance between counties. Estimating a model with addiction and spatial spillovers, we find large spillovers in opioid use and deaths across areas. A shock that increases opioid death rates by 1 in an index county causes 0.38 to 0.76 more deaths in other counties because of spillovers. Because opioids are addictive, this leads to even more deaths and spillovers in future years. In some specifications, these effects are large enough to generate a continuously increasing epidemic without any ongoing changes in demand or supply. We estimate spillovers explain 84 to 92 percent of opioid deaths from 1990 to 2018 and are the main reason deaths have increased for so long.

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The continuous rise in opioid overdose deaths in the past few decades is one of the worst health crises in history. **Figure 1** shows total drug overdose deaths, opioid overdose deaths, and the composition of opioid deaths by type of substance from 1990 through 2020. Total opioid deaths rose in all years but one over this time. In total, more than 1 million people died of a drug overdose since 1990, 58 percent of which involved opioids.[1] The situation is, if anything, getting worse. 2020 saw the largest percentage increase in opioid mortality (38 percent) since 1990, even as the baseline rate was the highest.

One might expect that drug overdose epidemics would be self-limiting. As people that become addicted are seen to suffer, fewer people will initiate addictive substances. Policy may also respond, disrupting drug markets and providing treatment and harm reduction services to people experiencing harm. Each of these would be expected to lower death rates. So far, the opioid experience belies this expectation.

In this paper, we examine why opioid death rates have increased so greatly and for so long. We consider three theories that could explain the continual increase in deaths. The first two are that demand or supply was steadily and exogenously increasing. With respect to demand, people may demand more opioids because they experience more physical or mental pain and desire relief from it. However, while people do report increased pain over time, this increase has been far less than the increase in opioid use and death rates (Cutler and Glaeser 2021). Mental distress, another possible contributor to demand for opioids, has also been relatively constant over time. Opioid use has increased mostly because people use more opioids given their level of pain, not because they experience more of it (Cutler and Glaeser 2021).

On the supply side, there have been changes in the types of opioids responsible for increased deaths, from misuse of legal opioids in the 1990s and early 2000s to use of illegal ones starting in the 2010s—first heroin and then illicitly made fentanyl (see **Figure 1**). However, these illegal opioids were not new to this time. Heroin has been circulating in the U.S. for over a century, and fentanyl has been available for decades. Greater use of these drugs involved ramping up existing supply to meet higher demand, rather than creating new narcotics. Consistent with this, we show that heroin prices rose as people switched from legal opioids to heroin and then reset to baseline levels as heroin deaths increased further – consistent with relatively inelastic short-run

[1] This is likely an undercount, as 20-25 percent of overdose deaths are not tested for the specific cause (Ruhm 2017). Ruhm (2017) estimates that opioids were involved in 77 percent of drug deaths since 1990.

supply and more elastic long-run supply.

Accordingly, we focus the bulk of our explanation on a third theory: that demand increases endogenously, due to spillovers in demand for opioids across people. Spillovers in demand for illegal opioids, or illegal uses of legal opioids, may result from several factors: spread of information about the substances, and ease and safety in ability to acquire the substances with more users. We collectively refer to these spillovers as "thick market externalities" (Caulkins and Kleiman 2018; Cook et al. 2007; Jacobson 2004). While spillovers are common for many goods and behaviors,[2] they have even stronger effects for addictive products. Spillovers and addiction reinforce each other, generating dynamic and spatial feedback (Becker 1992; Reif 2019). The result can be unstable equilibria that feature perpetual increases in use and harms.

We first show how spillovers can affect the dynamics of an epidemic theoretically. While there are several models of use of addictive drugs in the literature (Becker and Murphy 1988; Gruber and Köszegi 2001; Orphanides and Zervos 1995), these models consider isolated individuals and focus on steady-state substance use, not deaths. We expand the theoretical models to include spillovers in use and to add in death rates. We show that use and deaths can trend very differently. We further show that spillovers can turn even temporary shocks to drug demand or supply into long-term epidemics by amplifying the shocks across time and space.

We consider the importance of spillovers empirically by combining data on county opioid death rates and opioid shipments with data on physical distance and friendships across counties, taken from the universe of Facebook friend linkages in 2016 (Bailey et al. 2018). We ask whether opioid deaths in county A increase if deaths increase in counties that are geographically close to A or in other counties where people in county A have more friends.

We start with a case study of the first areas to have significant 'pill mills,' Southern Ohio and Northern Kentucky (Quinones 2015). We show that deaths increased in areas with pill mills and in two additional areas: areas that were geographically close to the pill mill counties and areas where more people had friends living in pill mill counties. Even as early as 2010 (before the transition to heroin and illicit fentanyl), opioid deaths in areas with the most friends in the pill mill

[2] See, for example: alcohol (Eisenberg, Golberstein, and Whitlock 2014; Kremer and Levy 2008), cigarettes (Cutler and Glaeser 2010; Fletcher 2010; Powell, Tauras, and Ross 2005), crime (Glaeser, Sacerdote, and Scheinkman 1996), illegal guns (Cook et al. 2007), social welfare enrollment (Dahl, Kostøl, and Mogstad 2014), new technologies (Agha and Zeltzer 2022; Bailey et al. 2022), retirement plan enrollment (Duflo and Saez 2003), and youth risky behaviors (Case and Katz 1991).

counties were higher than in the typical county in 2020.

The main empirical challenge to estimating spillovers empirically is the well-known reflection problem (Manski, 1993): it is difficult to separate out spillovers from correlated demand or supply shocks across areas. We overcome this challenge by using exogenous factors affecting opioid usage and lagged opioid deaths in peer and neighboring counties as instruments for opioid deaths in peer and neighboring counties. We divide our empirical analysis of spillovers into two time periods. The first is from 1996 through 2010, a time where deaths mostly involved excessive use of prescription opioids. The variables that identify exogenous increases in opioid utilization include presence of state triplicate prescription programs (Alpert et al. 2021); mid-1990s cancer mortality rates (Arteaga and Barone 2021); and 1990 disability rates (Cutler and Glaeser 2021). All three of these instruments are highly predictive of county-level death rates. To smooth out short-term fluctuations in death rates and ease computation of our models, we estimate persistence and spillover effects using county-level data grouped into three- to five-year intervals.

We find that opioid deaths are highly persistent over time and that there are large spillovers across areas. Due to persistence of opioid use, each death at time t is followed by 0.5 to 0.9 deaths in the same county at time $t + 1$. Further, when opioid death rates increase in one county, they increase in neighboring counties and in counties where people in the index county have more friends. Averaging across county pairs, we find that shocks that increase opioid death rates by 1 in one county increase opioid deaths in other counties by 0.38 to 0.76 deaths in the same period.

Beginning around 2010, there was a transition from deaths resulting primarily from excess use of legal opioids to deaths resulting from excess use of illegal opioids. Most studies attribute this transition to prescription opioid users substituting to heroin as prescription opioids became more difficult for them to obtain and abuse (Alpert, Powell, and Pacula 2018; Compton, Jones, and Baldwin 2016; Evans, Lieber, and Power 2019). Our focus is not on this transition. Rather, we study whether there were also thick market spillovers in the period after 2010.

To do so, we estimate models for opioid deaths in three-year intervals from 2010 to 2018. To instrument for demand for illicit opioid deaths in a county, we use each area's historical rate of prescription opioid shipments prior to 2010 (as in Cutler and Glaeser 2021); heroin death rates prior to 2010 interacted with geographic variation in the share of heroin that was white powder (thus, more substitutable for fentanyl) (Pardo et al. 2019); and lagged death rates.[3] As in the earlier period, we find that opioid death rates are persistent and that there are large spillovers in illicit opioid deaths across areas. The coefficients are of similar magnitude to those in the prescription opioid period, with autocorrelation in mortality of 0.5 to 0.7 and spillovers of 0.40 to 0.75 deaths in other counties for each death in the index county.

Using these estimates, we simulate the path of opioid deaths under an assumption of no spatial spillovers and compare that to actual trends in mortality. While we find persistence in opioid deaths over time, the persistence coefficients are all significantly less than 1. Thus, in the absence of spatial spillovers, the opioid epidemic would die out reasonably quickly over time. However, spatial spillovers change that calculation. Using our preferred models, counterfactual calculations indicate that opioid deaths would have been 84 to 92 percent lower without spatial spillovers and would have peaked by 2006. We conclude that thick market spillovers, not exogenous increases in demand or supply, are the most important reason why the opioid epidemic has lasted for so long.

In addition to helping to understand the opioid epidemic, our paper contributes to the literature on models of addictive goods. The Becker and Murphy (1988) rational addiction model has been widely studied in the literature, including a variety of evidence consistent with at least some forward-looking behavior (Cawley and Ruhm 2011). However, we show the model has difficulty explaining persistent epidemics and high death rates. We extend this literature to consider deaths as well as substance use and to endogenize spatial spillovers. Each of these extensions is vital to understanding observed epidemics.

The remainder of the paper is structured as follows. Section I presents a summary of the opioid epidemic. Section II develops a theoretical model of opioid death rates and discusses reasons opioid epidemics may last for a longer or shorter period. Section III describes our data. Section IV presents a case study of opioid deaths spilling over into other geographic areas from areas where the first pill mills in the U.S. were established. Section V presents the empirical methodology, and Section VI presents the main results. Section VII presents several robustness checks. Section VIII concludes.

[3] If we instead use the exogenous factors that predict prescription opioid utilization (state triplicate prescription programs; mid-1990s cancer mortality rates; and 1990 disability rates), results are similar.

I. The Opioid Epidemic

Opium and its derivatives (called opioids) have been used for millennia.[4] Because opioids are addictive, there is almost always a low level of chronic use, and epidemics are common (Courtwright 2009). Prior to the current epidemic, the most recent widescale epidemic in the U.S. took place in the mid- to late-1800s (Courtwright 1983; Musto 1999). Like today's epidemic, it was ignited by the widespread medical use of opium and morphine to alleviate pain.[5] The epidemic peaked in 1890, when the medical profession became reluctant to prescribe opioids outside isolated and acute clinical settings (Musto 1999), and states implemented policies to stem prescribing (Kolb and Du Mez 1924). Still, opioid use remained a concern in the U.S. until the 1910s.

Since that time, epidemics involving illicit opioids have taken place in some urban areas, but these have been smaller on a national scale and lasted less long. For example, there was a good deal of press and public concern about heroin entering in the US in the 1960s and 1970s through the French Connection, which ultimately led to President Nixon's famous declaration of the "War on Drugs".[6] Death rates during these epidemics peaked in around a decade (DuPont and Greene 1973). It was around this time that drug policy in the U.S. shifted from opioids to other drugs such as marijuana and cocaine, for which use was increasing (Musto 1999). Like the 1960s heroin epidemics, the crack cocaine epidemic that subsequently took place in the 1980s was also comparatively short, peaking and declining in around a decade (Fryer Jr et al. 2013).

The current opioid epidemic has lasted much longer. The epidemic is generally dated to the mid-1990s, with the FDA approval and subsequent heavy marketing of *OxyContin* in 1995 (Humphreys et al. 2022; Van Zee 2009). *OxyContin*, which released opioids more slowly than previous prescription opioids, was promoted as less addictive. This perceived innovation in safety, and subsequent marketing of such, convinced the medical profession to again prescribe opioids more liberally for common and chronic pain. This was accompanied by broader changes, with industry-funded advocacy groups persuading clinicians to treat pain more aggressively and

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[4] The earliest reference to opium poppies comes from the third millennium B.C., where it was cultivated in Mesopotamia and referred to by Sumerians as Hul Gil, the "joy plant" (Brownstein 1993). Lengthy discussions of various opioid epidemics that have occurred throughout history can be found in Courtwright (2009).

[5] Records from physicians at the time indicate opioids were used to treat virtually any type of pain or mental unrest, including conditions as diverse as nausea, asthma, bronchitis, cholera, colic, diarrhea, dysentery, hemorrhoids, and intermittent fevers (Courtwright, 1983)

[6] The French Connection describes a heroin trade route that originated in Indochina, with heroin being smuggled from there through Turkey and France before arriving in Canada and the U.S.

prescribe all kinds of opioids more liberally (Humphreys et al. 2022). This included promotion of other opioids that had already been in circulation for some time, such as *Percocet*, *Roxicodone*, *Vicodin*, as well as generic opioid products.

Unfortunately, just as it had a century prior, widespread opioid prescribing resulted in massive collateral damage. Prescription opioid use soared in the late 1990s and 2000s. Along with it came the more than three-fold increase in opioid death rates from 1995 to 2010, mostly involving prescription opioids, as shown in **Figure 1**.

Consistent with *OxyContin* playing an important role in this, Alpert and colleagues (2021) show harms were experienced most acutely in areas where documents indicate that *OxyContin's* manufacturer (Purdue Pharmaceuticals) initially targeted promotions: states without triplicate prescription programs. Arteaga and Barone (2021) provide further support, showing deaths increased more in counties with higher mid-1990s cancer mortality. This is consistent with internal documents from Purdue showing a desire to target marketing at physicians treating cancer patients. Lastly, consistent with demand for pain relief playing an important role, Cutler and Glaeser (2021) show that opioid shipments and deaths increased the most in areas with high levels of pain, as measured by the percent of adults that received disability benefits in 1990.

By the mid-2000s, public and private policies started to act against prescribing believed to be excessive. Early efforts predominantly involved litigation against *OxyContin*'s manufacturer (Meier 2007) and enforcement interventions that targeted prescribers, dispensers, and distributors that supplied opioids recklessly (Donahoe 2023; Kennedy-Hendricks et al. 2016). Around mid-2010, *OxyContin* was also reformulated to be abuse-deterrent (Alpert et al. 2018; Evans et al. 2019). Other substantial policy interventions, such as state programs that require prescribers to query each patient's opioid prescribing history before issuing a prescription (Horwitz et al. 2021), came later, starting around 2013. While these policies reduced use of prescription opioids (Compton et al. 2016) and some even reduced mortality (Donahoe 2023; Kennedy-Hendricks et al. 2016), they did not stem aggregate death rates. Rather, deaths transitioned to illicitly made opioids, heroin and then non-prescription fentanyl. The increase in opioid deaths since 2010 has been almost entirely among illegal opioids.

II. Theories About The Extended Opioid Epidemic

In this section, we develop a theoretical model of addictive drug consumption and deaths to frame explanations for why drug epidemics may last for an extended period (like the current opioid epidemic) or be over relatively quickly (like the 1980s crack cocaine epidemic). We model consumer behavior allowing for addiction and spillovers, as in Reif (2019).[7]

A. *Consumption with addiction and spillovers*

Consumers experience temporal utility $V(a_{it}, S_{it}, x_{it}, c_{it}, E[\bar{a}_t])$, where a_{it} is i's consumption of addictive goods (e.g., opioids) in period t. S_{it} is the consumer's consumption stock, with evolution $S_{it+1} = (1-d)(S_{it} + a_{it})$ and depreciation rate d. x_{it} are 'taste' parameters that increase utility from drug use (e.g., pain, exposure to drug marketing, etc.) and c_{it} is a composite of all other goods. Lastly, $E[\bar{a}_t]$ denotes expected addictive drug consumption by a person's peers. This may affect utility because there is adjacent complementary in consumption, or because of information spillovers across people.[8] As in Reif (2019), we assume quasi-linear utility in the private and social components (i.e., that $V = U(a_{it}, S_{it}, x_{it}, c_{it}) + G(a_t, E[\bar{a}_t])$) and linear spillovers ($G(a_t, E[\bar{a}_t]) = b_g a_t E_t[\bar{a}_t]$) for group g. Taking U as concave and quadratic and choice of c_{it}^* as optimal, the consumer's optimization problem for use of the addictive product can be written as follows:

$$\max_{a_{it}} \sum_{t=1}^{\infty} \beta^{t-1} V^*(a_{it}, S_{it}, x_{it}, E[\bar{a}_t])\,, \tag{1}$$

with discount rate β. The consumer faces a budget constraint:

$$A_{i0} = \sum_{t=1}^{\infty} (1+r)^{-(t-1)} (c_{it}^* + p_t a_{it})\,, \tag{2}$$

with interest rate r and the price of addictive drugs p_t.

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[7] There is another set of models that simulate drug epidemic dynamics using contact models of infectious disease (MacKintosh and Stewart 1979). While these may be parameterized to fit a drug epidemic's dynamics, drug epidemics are fundamentally different from infectious disease epidemics. Thus, these models are ill-suited for explaining why people behave in a particular way.

[8] There may also be spillovers because the product is easier to obtain from friends or relatives instead of in the black market. This could be modeled a variety of different ways, e.g., through lower search costs or prices associated with thicker markets. Quantitatively, these alternative representations would have similar impacts.

For ease of illustration, first consider the case where consumers are fully myopic (i.e., they ignore the effect that consuming drugs today will have on their utility in the future). Assume also that V(.) is quadratic in its elements.[9] Maximizing equations 1 and 2 with $\beta = 0$ yields a demand equation of the following form,

$$a_{it}^* = \alpha S_{it} + \gamma \bar{a}_t + \pi p_t + \delta x_{it} + k. \tag{3}$$

In equation (3), $\alpha = \frac{b_{aS}}{b_{aa}} > 0$ captures the addictiveness of drugs, $\gamma = \frac{b_g}{b_{aa}}$ denotes spillovers, which may be positive or negative, $\pi = -\frac{\lambda}{b_{aa}} < 0$ denotes downward sloping demand (λ is the marginal utility of wealth), $\delta = \frac{b_{ax}}{b_{aa}} > 0$ denotes the demand response to the tastes parameter, and $k = \frac{b_a}{b_{aa}} > 0$ is a constant term.

The fully dynamic specification, where consumers have perfect foresight, is similar, with additional terms for future individual consumption ($\alpha_2 a_{it+1}$), future group consumption ($\gamma_2 \bar{a}_{t+1}$), future prices ($\pi_2 p_{t+1}$), and future tastes ($\delta_2 x_{it+1}$):

$$a_{it}^* = \alpha_1 S_{it} + \alpha_2 a_{it+1} + \gamma_1 \bar{a}_t + \gamma_2 \bar{a}_{t+1} + \pi_1 p_t + \pi_2 p_{t+1} + \delta_1 x_{it} + \delta_2 x_{it+1} + k'. \tag{4}$$

The details of each term in equation (4) are in **Appendix A**.

Some people that use addictive substances become poisoned and die. Even with optimally chosen consumption, poisoning deaths are stochastic, for two reasons. First, the line between the quantity of opioids that will get a person high or prevent withdrawal, and the quantity that will kill them, is very thin (Gable 2004). Small variations in quality can cause a person to take more than they intended to and overdose. Second, even if a person takes the same dose that they always have, death can occur due to random fluctuations in a person's metabolism or interactions with other drugs or health problems the person has at the time (Humphreys, 2023). We model the probability

[9] The quadratic parameterization of $V^*(a_{it}, S_{it}, x_{it})$ is as follows: $b_a a_{it} + b_S S_{it} + b_x x_{it} + b_{as} a_{it} S_{it} + b_{ax} a_{it} x_{it} + b_{Sx} S_{it} x_{it} - \frac{1}{2}(b_{aa} a_{it}^2 + b_{SS} S_{it}^2 + b_{xx} x_{it}^2)$.

that a person dies as a hazard function $H(a_{it}, S_{it})$, where $H_a > 0$; $H_{aa} > 0$; and $H_{aS} < 0$.[10]

The time series behavior of deaths would be expected to differ from that of use. Deaths will increase when use rises in relation to past use and fall when use stabilizes or falls.

B. Inciting an epidemic

We discuss factors in the model that could cause drug use and deaths to increase, inciting an epidemic, and to continue to rise. We temporarily ignore spillovers (i.e., assume that $\gamma = 0$) and focus on the myopic model; the model with dynamics and spillovers is presented below.

The condition for an epidemic to continue indefinitely (i.e., without any exogenous changes to the taste or price parameters) is:

$$\frac{b_{as}(1-d)}{d} > b_{aa}. \tag{5}$$

The left-hand side reflects addictiveness, how much utility from current use rises with past use. The right-hand side reflects the slope of marginal utility of consumption, defined as positive in the model.[11] Assuming the left-hand side is sufficiently small relative to b_{aa} and thus this equation is not satisfied, equation 3 will lead to an internal equilibrium: ($a_i^*(1-d) = S_i^* d$) (Reif 2019).

Two factors can disrupt this equilibrium. The first is exogenous changes in tastes (x_{it}): e.g., rising pain or mental distress, or more favorable perceptions about drugs due to marketing. The second is an exogenous expansion of supply (resulting in lower prices p_t) or development of a new drug.[12] In the static model, only current x_{it} and p_t affect demand. In a dynamic model (equation 4), expected changes to prices and tastes in the future can also cause more drug use. If people expect that their tastes for drugs will be higher in the future (x_{it+1}) or that prices will decline (p_{t+1}), they will increase current use in response to higher expected future marginal utility.

In response to a positive demand or supply change, average consumption of addictive drugs will converge to a new, higher level, as shown in the solid blue line in **Figure 2(A)**. Along the adjustment path, consumption increases over what people have developed a tolerance for, and

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[10] To formally model deaths below, we use a logistic model: $H(a_{it}, S_{it}) = \frac{\exp(\psi_0 + \psi_a a_{it} + \psi_{aa} a_{it}^2 + \psi_{aS} a_{it} S_{it})}{1+\exp(\psi_0 + \psi_a a_{it} + \psi_{aa} a_{it}^2 + \psi_{aS} a_{it} S_{it})}$, with $\psi_0 < 0$; $\psi_a > 0$; $\psi_{aa} > 0$; and $\psi_{aS} < 0$. While it is natural to assume H_{aa} is convex around the dose a particular person uses, it will clearly become concave at some point as the overall hazard approaches 1.

[11] Note in footnote 8, b_{aa} is pre-multiplied by -1/2.

[12] A new drug can also be conceptualized through lower prices (as prices for drugs that are unavailable are infinite). If the new drug is perceived to be safer than other drugs, demand could shift out even if it is sold at the same price.

deaths rates will rise. When the new steady state consumption is reached, deaths should peak and then decline. This yields the characteristic "epidemic" curve in the solid line (without spillovers) in **Figure 2(B)**.

This inverse U-shaped epidemic curve may be enhanced if public policies are enacted in response to the increase in deaths that aim to reduce utilization. Policymakers observing increases in drug deaths might increase the price of legal drugs or make illegal versions of the drug more difficult to obtain, for example by disrupting drug markets. Further, if demand for the good increased because people believed a new drug to be non-addictive and effective, that belief may reset as people learn they have been deceived or that the prior science was wrong.[13] This would lower death rates below pre-change levels until the new equilibrium is reached.

C. Difficulty with Exogenous Demand and Supply Changes

The initial increase in opioid use in the 1990s, described in section I, has features that match the model above. A new formulation of opioids was introduced, which was promoted as safer and more effective than existing formulations. In response to this, demand shifted out, and use rose. Deaths increased along with it. The subsequent pattern does not fit this pattern, however. As described above, deaths should have plateaued and returned to initial levels once use reached a new steady state. Further, one would expect the reaction to increased deaths and the variety of restrictive policies that were implemented in response to the epidemic (see section I) would have caused demand to fall and for opioids to become more costly, leading to further declines in use and a temporary reduction in deaths below the steady-state level. Instead, **Figure 1** shows death rates kept climbing.

One theory about why opioid deaths have kept increasing is that tastes for opioids are continuing to rise (i.e., x_{it} is still increasing) due to rising physical or mental pain in the population. The latter is commonly referred to as the "deaths of despair" theory (Case and Deaton 2015, 2017, 2020, 2021). However, data show that increases in pain (physical and mental) have not been large enough to explain these trends. Between 1999 and 2018, opioid overdose death rates increased four-fold while population prevalence of physical pain increased by at most around 20 percent (Cutler and Glaeser 2021). Trends in mental pain and life dissatisfaction were flat or falling over the same time. Thus, trends in pain are simply not quantitatively large enough to explain why

[13] Demand may come from individuals, their physicians, or their health insurance company.

opioid deaths rose so continuously or for so long.

A second factor that could cause an extended epidemic is further technological innovations (e.g., development of new opioids) or exogenously expanding supply (causing further reductions in prices). The expansion in use of illegal opioids after 2010 is potentially such a supply shock. However, neither of the products commonly used in this era (heroin and fentanyl) were new. As noted, heroin has been circulating in the U.S. since the early 1900s (Courtwright 2009; Musto 1999). Fentanyl was synthetized in 1959 and there were even been small-scale epidemics of illicit fentanyl deaths in the mid-2000s in several areas heavily affected by fentanyl today (e.g., Chicago, Detroit, and Philadelphia) (Centers for Disease Control and Prevention 2008; Westhoff 2019).

The idea of using fentanyl to augment heroin—and later to incorporate it in counterfeit pills—is newer to this era, suggesting innovations in the production process if not the specific drug. Fentanyl is significantly cheaper to produce than heroin.[14] Thus, fentanyl (which is a white powder) was used to cut and replace white powder heroin in areas where white power heroin is common.[15] Because the potency of fentanyl-based products varies significantly from batch to batch,[16] this adulteration of heroin would be expected to increase deaths, though it would explain a one-time change and not a continual increase. How long it takes a one-time shock to play out is unknown, though a decade of ever greater increases seems unusual historically.

The importance of exogenous expansions of supply can be assessed by matching trends in illicit opioids prices with death rates. If exogenous supply shifts are the driving factor in increased deaths, we should see reductions in prices coincident with or just prior to increased use and death rates and an increase in use consistent with demand elasticities for that substance. In contrast, if illegal drug supply was responding to an increase in demand from users, prices should increase at times of heavy demand and then decline over time, as long-run supply expands.

Heroin price data are more readily available than fentanyl prices. **Figure 3** presents real (in 2020 dollars) street prices of heroin based on drug seizures data and death rates from 1999 to 2020

[14] The cost is roughly 1/300 to 1/400 as much per effective dose (Mars, Rosenblum, and Ciccarone 2019). Further, fentanyl can be manufactured anywhere and with limited resources.
[15] Qualitative research obtained from interviews with opioid users (Mars, Rosenblum, and Ciccarone 2019) suggests that initially, users they did not know they were being sold fentanyl instead of heroin, suggesting a supplier- rather than user-driven shift from heroin to fentanyl.
[16] To divide wholesale fentanyl into retail products, many suppliers mixed it with other additives using magic bullet blenders (Quinones 2021). This is clearly not ideal for quality control and led to a lot of people using more fentanyl than they intended to and overdosing.

(UNODC 2020).[17] Heroin death rates started increasing in 2005, rising eight percent annually from 2005 to 2010. Over that same time, heroin prices were also increasing—rising 60 percent from 2005 to 2010. The confluence of rising death rates and rising prices is consistent with an increase in demand, not supply. Heroin prices only started declining after 2010, as supply expanded.[18]

Accounting for the increases and subsequent decreases, the price of heroin has changed little from prior to the major increase in heroin deaths up through recent years – in real terms, heroin prices were approximately the same in 2020 as they were in 2005. Even still, deaths were over five times higher. The data are thus strongly consistent with an exogenous increase in demand that was satisfied by elastic supply rather than an exogenous increase in illegal drug supply.

Data on fentanyl prices are somewhat more nuanced. Illicit fentanyl enters the US drug supply in two primary ways: directly shipped from China to individuals in the US, who further refine it; and via Mexican drug trafficking organizations, who distribute refined fentanyl alongside heroin – and often in combination.[19] The share from each route is not generally known, though both are believed to be significant (Dudley et al. 2019). Dark web prices for Chinese fentanyl fell by roughly 50 percent from 2014 to 2016 (Miller, 2020). Similarly, bulk purchases prices of fentanyl in the US fell roughly 63 percent between 2016 and 2020 (Kilmer et al., 2022).

Retail prices, in contrast, have been far less affected. Street prices of heroin containing fentanyl are typically sold at the same price as heroin without fentanyl (Mars, Rosenblum, and Ciccarone 2019). Further, as noted above, heroin prices were rising in the mid-2010s, even as the heroin was increasingly cut or replaced with fentanyl.

The net impact of these fentanyl price changes on expected utilization is unclear. By retail prices, there is no reason for illegal fentanyl use to increase. However, even if the 63 percent price decline in wholesale fentanyl prices were passed through fully to retail prices, estimates of the demand elasticity for heroin (-0.8) (Olmstead et al. 2015) suggest that the decline in fentanyl prices can explain only a 50 percent increase in fentanyl deaths from 2016 to 2020. In contrast, fentanyl deaths rose 188 percent over the same time.

Overall, therefore, the data do not suggest that exogenous changes in demand for pain

[17] This is a price per pure gram. Over this time period, the DEA data show that purity was reasonably constant; the increase in price was because nominal prices were increasing.

[18] There was another spike in heroin prices from 2016 to 2018. The cause of this price increase is not known, but it may reflect declining production of heroin in Columbia and the shift of production to Mexico around this time, combined with continued high demand (Drug Enforcement Administration 2019, p. 24, Figure 14)

[19] The precursor chemicals may come from China or India, or be made in Mexico.

relief, expansions of supply, or new modes of production are a major reason why deaths have risen as much and for as long as they have. We thus turn to other explanations.

D. The impact and importance of thick markets

An alternative theory about the long epidemic is that increased opioid use by some people causes other people to use more opioids as well. Returning to our model and equation 3, we model this through spillovers (through $b_g \bar{a}_t$). While spillovers are common for many goods, they have particularly large impacts for addictive goods. Consider a shock that leads to higher use at time t. That shock will lead to an increase in consumption capital in $t+1$, which will lead to higher opioid use among those who used in the first period, as well as everyone else who interacts with first period users, due to the spillover effects. This has potential to generate a self-perpetuating cycle of continuously increasing opioid use and deaths. Specifically, the model of use in equation 3 does not have a steady-state equilibrium if:

$$\frac{b_{as}(1-d)}{d} + b_g > b_{aa}. \tag{6}$$

The first term on the left-hand side of equation 6 is the same as in equation 5. The second term, (b_g), is new, reflecting the importance of social spillovers. Equation (6) is more likely to be true than is equation (4).[20] Because spillovers lead opioid use in subsequent periods to continue to exceed the stock of past consumption, they will likely cause deaths to continue increasing as well. This is illustrated in **Figure 2**, for a scenario where spillovers are 80 percent as large as the effect of past use on current use, close to empirical estimates that we present in section VI. In this scenario, use continues to increase and deaths along with it.

There are several reasons that one would expect spillovers to be important for opioids. The first is information conveyance. In illegal markets, information does not flow as freely as it does in legal markets. Users and dealers cannot openly advertise their intent to use or sell opioids without incurring risk, and informal methods of communication (such as through social networks) are more important (Cook et al. 2007). Having more people using opioids in one's social network eases this friction. One of the earliest reports about *OxyContin* misuse in the U.S. describes the

[20] See Reif (2019) for the similar stability condition for the fully dynamic model in equation 4.

epidemic spreading in this way:

"The earliest reported cases of OxyContin abuse were in rural Maine, rust-belt counties in western Pennsylvania and eastern Ohio, and the Appalachian areas of Virginia, West Virginia, and Kentucky. The problem travelled through these regions, as friends told friends and the word spread from town to town, county to county, up and down the Appalachians… Part of what makes the spread of OxyContin abuse so difficult to track, let alone to stop, is that the drug moves not physically but conceptually… a recovering OxyContin addict and former small-time dealer offered an explanation for OxyContin's sudden geographical shifts. `It's the idea that passes on,' he told me. `That's how it spreads… It's dealt by word of mouth. I call a friend in Colorado and explain it to him: Hey I've got this crazy pill, an OC 80 [OxyContin 80 mg]… You've got to go to the doctor and get it. Tell him your back hurts." (Tough 2001)

Second, the costs of obtaining opioids may fall when there are more opioid users. On the monetary side, the risk of interdiction of drug sales creates high fixed costs that must be spread across many users. This can lead to lower per-user prices when there are more users (Caulkins and Reuter 2006). Jacobson (2004) demonstrates evidence of this phenomenon for marijuana, exploiting plausibly exogenous variation in the size of youth cohorts. Thicker markets for opioids may also lower non-monetary costs associated with obtaining opioids, such as time spent searching for a seller, an idea that dates back to search models of macroeconomics (Diamond 1982).

Third, when there are many users, one can more readily obtain the product from friends or relatives. Drug markets are often characterized by participants being both buyers and sellers of the good. When familiar individuals are also sellers, the costs and risks from entering an illegal market can be reduced. Data from the National Survey on Drug Use and Health show that half of people who first misused prescription opioids obtained them from a friend or relative (Lipari and Hughes 2017).

Finally, other non-monetary costs of using opioids (such as health harms and social penalties) may also fall when there are more users. When addiction is more widespread, policy may shy away from the harsher legal responses that may be pursued when drug use is concentrated among smaller and stigmatized groups (Courtwright 2009; Kim, Morgan, and Nyhan 2020). Greater opioid use may also contribute to use becoming normalized and thereby associated with lower social penalties.

Understanding whether these spillovers can explain why opioid deaths have been increasing for so long is ultimately an empirical question. We turn to that next, starting with an explanation of our data.

III. Sources of Data

A. County opioid poisoning death rates

We obtained counts of county drug overdose deaths from 1990 to 2018 from restricted-access vital statistics data from the National Centers for Health Statistics (NCHS 2018).[21] We used these data to construct county-specific overdose death counts from any drug, opioids, and (since 1999) opioids by type (prescription opioids, heroin, and synthetic opioids other than methadone [which includes fentanyl]). Coding conventions for these deaths followed prior literature.[22] Deaths are reported by area of residence. Thus, a person who travels to obtain opioids and dies in another county will be attributed to the county in which they live. Data on county population sizes, which we use to construct death rates, are from the National Institutes of Health Surveillance, Epidemiology, and End Results Program (N.I.H. and Vilhuber 2021). We age- and sex-adjust all death rates to the U.S. 2010 population.

Figure 4 shows a map of average annual opioid deaths per 100,000 people (average from 1990 to 2018) across 3,117 counties for which we have data. Death rates are censored when fewer than 10 total deaths occurred, per our data use agreement. From the figure, death rates are clearly highly spatially correlated: counties that have high opioid death rates tend to be near others with high death rates and vice versa. The clustering also tends to bleed across state boundaries, making it clear that this is not purely the result of differences in state policies across areas. The largest clusters include Appalachia (particularly southeastern Ohio, eastern Kentucky, West Virginia, and western Virginia); several states in the northeast (from Connecticut and upwards through to Maine); and several clusters of counties out west in Nevada, Utah, and New Mexico. We formally

[21] Counts of national opioid deaths for 2019 and 2020, used in **Figure 1**, were obtained using publicly available NCHS data for those years.

[22] Drug deaths after 1999 were identified based on the International Classification of Diseases (ICD), 10th edition underlying cause-of-death codes X40–X44, X60–X64, X85, and Y10–Y14. Overdoses by category were identified by multiple-cause-of-death codes T40.1 (heroin), T40.2 (prescription opioids = natural and semisynthetic opioids), T40.3 (methadone), and T40.4 (fentanyl/tramadol = synthetic opioids other than methadone). Total opioid deaths also included code T40.6 (other/unspecified narcotics). Drug deaths before 1999 were identified based on ICD, 9th edition underlying cause-of-death codes E850-E858, E950.0-E950.5, E9620, and E980.0-E980.5. Opioid deaths before 1999 were identified from underlying cause-of-death codes E850.1-E850.2 and 305.5, as well as multiple-cause-of-death codes 965.00-965.09. Deaths involving more than one opioid category are counted in both. See Fingerhut and Cox (1998) and Hedegaard, Miniño, and Warner (2020) for more details. To account for the change from ICD-9 codes (1990–1998) to ICD-10 codes (1999–2017), the following comparability ratios were applied to ICD-9 codes E850-E858, E950-E950.5, E9620, and E980.0-E980.5 (respectively) in the calculation of total drug deaths: 1.0365, 1.0013, 0.9870, and 1.0417 (Miniño et al. 2006). Total opioid deaths were adjusted upward by about 20 percent (comparability ratio = 1.195) (Hoyert et al. 2001).

taste for spatial correlation in section VI.

B. County opioid shipments

Data on opioid shipments are from publicly available summary reports of the Automation of Reports and Consolidated Orders System (ARCOS) and include all shipments of codeine, oxycodone, fentanyl base, hydrocodone, hydromorphone, and morphine. The ARCOS reports shipments at the 3-digit zip-code level, which we convert to counties using a 2010 zip-to-county crosswalk from the U.S. Census Bureau. These data include all shipments to the area and thus likely dispensed in the area, including to people who live in other counties. We use the data to construct each county's average rate of opioid shipments from 1997 to 2010, standardized in terms of 50 morphine milligram equivalents (MMEs) per capita. This is equivalent to roughly one high dose of opioids (Dowell, Haegerich, and Chou 2016).

We also obtained exact MME shipments at the county level from 2006 to 2014 from a special extract of the ARCOS which was unsealed as part of multi-district litigation against opioid manufacturers, wholesalers, and pharmacies, and is only available for those specific years.[23] We term this extract the detailed ARCOS data. For the detailed data, we calculate total MMEs along with *OxyContin* MMEs (including generic *OxyContin* during years that it was available) and other non-*OxyContin* opioid MMEs (including non-*OxyContin* oxycodone). These detailed ARCOS data are our dependent variable in modeling spillovers in opioid shipments. The longer-term ARCOS data are used to instrument for opioid death in our models of deaths during the illegal opioid era.[24]

C. Exogenous factors related to initial increases in opioid deaths

We obtained data on factors that have been cited in the literature as exogenously leading to greater opioid use. We first focus on three factors which were related to where prescription opioid use increased through 2010: areas without state triplicate prescription programs (Alpert et al. 2021); areas with higher 1994–96 cancer mortality rates (Arteaga and Barone 2021); and areas with higher shares of adults (age 25 to 64) that received disability benefits (Cutler and Glaeser 2021). We define triplicate programs as in Alpert and colleagues (2021) and crude rates of cancer

[23] Data are available at: https://www.slcg.com/opioid-data/.
[24] Because three-digit zip codes often cross county borders, data on county shipments formed from three-digit zip codes will induce some spatial correlation in opioid shipments. The detailed ARCOS shipment data (available at the exact county-level) do not have this problem.

deaths per 1,000 people from 1994-1996,[25] following Arteaga and Barone (2021), using our NCHS data. Data on the number of adults receiving disability benefits in 1990 comes from an Old-Age, Survivors, and Disability Insurance 1990 report (Cutler and Glaeser 2021); we divide these counts by the number of adults aged 25 to 64 using SEER population counts.

For the analysis of illicit opioids (after 2010), we focus on instruments that have been related to illicit opioid use. These include pre-2010 (1997 to 2010) opioid shipments per capita and each county's pre-2010 heroin death rate, interacted with the share of heroin seizures that were white powder in eastern vs. western states. This latter instrument is included because fentanyl is a more natural substitute for white powder heroin than black tar heroin. Data on heroin seizures come from the Drug Enforcement Administration's Heroin Domestic Monitoring Program, which analyzes undercover purchases of retail heroin in several major U.S. cities (Drug Enforcement Administration 2004–2010). The dividing line for white powder heroin is the Mississippi River (east is almost exclusively white powder; west is almost exclusively black tar) (Ciccarone 2009). We do not have seizures for all counties, so we estimate each county's share of heroin that is white powder using the average share of seizures that is white powder in each county's geographic region (east or west of the Mississippi).

In supplementary analyses, we also use several other variables that have been identified in the literature as leading to more opioid deaths. For initial prescription opioid increases, we use the percent of people aged 25 to 64 without a college degree (from the 2005 to 2009 American Community Survey); and predicted increases in import competition from China based on industry shares in 1990 from Autor, Dorn, and Hanson (2013).[26] For increases in illicit opioid use, we examine effects separately for *OxyContin* use in the area and other drugs. Because *OxyContin* was reformulated to be abuse deterrent in 2010 (Alpert et al. 2018; Evans et al. 2019), pre-2010 *OxyContin* use may be more related to post-2010 illegal drug use than use of other opioids is.[27]

D. Measures of social interactions across areas

We obtained two measures of interactions between two counties. The first is the geographic

[25] We include all deaths with an ICD10 underlying cause of death code for a malignant neoplasm (C00-C97).

[26] Case and Deaton (2021) show that people without college education were particularly vulnerable to opioids as the epidemic expanded, due to higher levels of pain and despair. Autor, Dorn, and Hanson (2013) show that opioid deaths increased more in areas where rising imports from China reduced wages and displaced people from jobs.

[27] Several studies argue *OxyContin* reformulation was the cause of increased heroin deaths immediately after 2010, due to users substituting from *OxyContin* to heroin (Alpert, Powell, and Pacula 2018; Evans, Lieber, and Power 2019).

distance (in miles) between county centroids (denote $d_{i,j}$ for counties i and j).[28] This measures how physically close two counties are to one another and picks up, among other things, how easily one can move from one county to the other to obtain opioids that are being supplied there. The second is based on the amount of Facebook friendships between people in different counties, from Bailey and colleagues (2018). Specifically, it is the normalized total number of Facebook friendship links between two counties i and j as of April 2016.[29] We refer to this measure as Social Connectedness Index (denote $SCI_{i,j}$ for counties i and j). We translate this to a measure of relative probability by dividing the SCI for counties i and j by the sum of the SCI for county i and all other counties $j \neq i$, as in Kuchler, Russel, and Stroebel (2021). This measure is likely to be particularly valuable in picking up information flows about opioids and their availability across counties.

As described in Bailey and colleagues (2018), social connectedness is inversely correlated with distance. Averaging across counties, the elasticity of social connectedness with respect to distance is -1.2 (Bailey et al. 2018). Still, the relationship is non-monotonic and may differ across areas, as we discuss below.

IV. Case Study of Spillovers from Initial Pill Mill Counties

In this section, we analyze a case study of exposure to an area that was one of the first areas where prescription opioid addiction and overdose deaths greatly increased—Greenup County, Kentucky, and neighboring Portsmouth County, Ohio. David Procter, considered the founder of the first pill mill in the U.S. (and who later was sentenced to jail), practiced in these two counties and prescribed large amounts of opioids to patients who did not have legitimate medical need for them in exchange for cash payments, starting in the 1990s (Quinones 2015). Journalistic accounts frequently cite this initial pill mill as foundational in the trajectory of the overall epidemic, often describing the epidemic as spreading outward from this very first pill mill:

"In Ohio, pain-pill addiction and its consequences got bad first in Portsmouth. After that, the state's public health maps each year showed the red stain spreading north, as if the dope had captured an outpost and from there it conquered more of Ohio every year." (Quinones 2021)

[28] Data are from the Census Gazetteer files.
[29] Note that this stock measure reflects current friendships in April 2016 as well as friendships in a person's past.

We empirically examine whether opioid deaths increased more in areas where people more frequently interacted with people in the counties where Procter initially practiced, based on physical distance to these counties and having more friends in them. **Figure 5** maps where people have more friends in these two counties. Distance is clearly an important predictor of friendships. However, friend relationships do not decline as rapidly as distance increases. The elasticity of social connectedness to Greenup and Scioto counties with respect to distance between them is -0.81. Thus, there is significant interaction between Greenup and Scioto counties and other areas of the U.S. that are not immediately nearby, as shown in the map.

Figure 6 shows trends in opioid overdose death rates from 1990 to 2018 for four cohorts of counties: Greenup and Scioto counties (where the initial pill mills were established); counties with above 95th percentile social connectedness to Greenup or Scioto County; counties with a 5th to 95th percentile social connectedness to them; and counties with less than 5th percentile connectedness. We censor the rates when fewer than ten total opioid deaths occur, per stipulations from the National Center for Health Statistics.

Starting in the early 1990s, the counties with the highest rates of opioid deaths were those with the lowest degree of social connectedness to where the pill mills were initially established. Many of these areas are urban, such as New York and San Francisco, where there was a longstanding rate of heroin use. However, as time progressed, the relationship reversed. Opioid death rates started increasing in Greenup and Scioto County in the late-1990s (initial years not shown, due to data censoring). Opioid death rates in the counties with the most friends in Greenup and Scioto counties quickly followed, surpassing death rates in other areas by 2001 and converging with the death rates in Greenup and Scioto counties in 2011. Though opioid death rates increased in all areas, they increased much sooner and by far more in the counties where pill mills got their start and counties where people had more friends in Greenup and Scioto counties. Even after the transition from prescription opioids to heroin and fentanyl in the 2010s, death rates were twice as high in areas where early pill mills were established and those that were connected to them.

Figure 7 shows a binned scatter plot of the change in opioid deaths per 100,000 people from 1992–1995 to 2008–2011 (the peak of overall U.S. prescription opioid shipments and death rates) against log miles to Greenup or Scioto County, whichever is closer (**Panel A**) and the log social connectedness to Greenup and Scioto County (**Panel B**). Distance and social connectedness to the two initial pill mill counties are both strongly related to changes in opioid deaths. The coefficients imply that halving distance to Greenup or Scioto County is associated with a 0.70 (10 percent) greater increase in the rate of opioid deaths per 100,000 people. Doubling social connectedness to Greenup and Scioto County is associated with a 2.27 (33 percent) greater increase in the opioid death rate.

V. Empirical Framework

In this section, we discuss how we test for spatial spillovers in opioid use and show how thick market externalities can cause extended opioid epidemics. As noted in Section III, the data on county-level opioid deaths are available for a longer period (1990 to 2018) than the detailed ARCOS data on county-level opioid shipments (2006 to 2014). Thus, our primary models focus on death rates. The methodology extends to opioid shipments as well.

Building on our theoretical framework, our model takes the following form:

$$y_{c,t} = \lambda \sum_{j \neq c}^{I} w_{j,c} y_{j,t} + \gamma x_c + \rho y_{c,t-1} + \tau_t + \epsilon_{c,t}. \tag{7}$$

$y_{c,t}$ denotes county c's opioid death rate at time t. This is regressed on weighted averages of death rates in other counties at time t ($\sum_{j \neq c}^{I} w_{j,c} y_{j,t}$), lagged opioid death rates in county c ($y_{c,t-1}$), time fixed effects (τ_t), and an error term ($\epsilon_{c,t}$). Relative to the theoretical model, we do not have price in the model, as we do not observe it.

We use two alternative forms of weighting for $w_{j,c}$. The first is the inverse geographic distance (in miles) between county j and c ($1/d_{j,c}$). The second is based on the relative probability of a Facebook friendship between county j and c. We follow the recommendations of Kelejian and Prucha (2010) and normalize the weighting matrices by the largest eigenvalue to facilitate interpretation and comparisons between the two different forms of weighting.

The primary concern with estimating equation (7) with ordinary least squares is the reflection problem. Common shocks in neighboring counties will appear as spillovers in deaths when that is not really the case. To account for these sources of bias, we use the exogenous determinants of opioid use (x_c) and lagged death rates ($y_{c,t-1}$) in other areas (denote $z_{c,t}$) as instruments for identifying spillovers. Specifically, we implement a generalized spatial two-stage least squares (GS2SLS) procedure that instruments for weighted averages of opioid deaths in other areas ($\sum_{j\neq c}^{I} w_{j,c}y_{j,t}$) with weighted averages of the instruments ($\sum_{j\neq c}^{I} w_{j,c}z_c$) (Arraiz et al. 2010; Drukker, Egger, and Prucha 2013; Kelejian and Prucha 1998, 1999, 2004, 2004).[30] The estimating equation is in **Appendix A**. Identification relies on exogeneity of determinants of current use in other counties (including lagged death rates), conditional on determinants of use in one's own county.

We divide estimation into two time periods: the first period, from 1996 to 2010, that was largely driven by rising prescription opioids deaths, and the second from 2010 to 2018 that saw sharply rising deaths from heroin and illicit fentanyl. We do not estimate models that span the period before and after 2010 because the nature of utilization and deaths changed in the two periods, as noted above. Estimating GS2SLS variant of equation 4 is computationally intensive. In addition, we wish to focus on average use over some interval, not necessarily the impact of one or two deaths at a specific moment.[31] Both of these considerations lead us to take averages over several years. In the models for pre-2010 deaths, we divide the interval into four five-year time windows: 1991–1995, 1996–2000, 2001–2005, and 2006–2010. In the post-2010 period, when deaths are more common, we divide the sample into four three-year time intervals: 2007–2009, 2010–2012, 2013–2015, 2016–2018. In both models, the first period of observation (1991–1995 and 2007–2009 respectively) only shows up as the lagged observation for the next period (i.e., not as dependent variables themselves).

In analyzing spillovers in opioid shipments, we use annual data from 2006 to 2009 (before any major policy efforts to curtail supply). We do this because there are fewer years of data and shipments are more stable than deaths. Again, data from the first period (2006) only enters the model as a lagged observation for 2007.

An important question is whether opioid death rates should be related to contemporaneous or lagged death rates in other areas (i.e., whether the weighting term should involve $\sum_{j\neq c}^{I} w_{j,c}y_{j,t}$ or $\sum_{j\neq c}^{I} w_{j,c}y_{j,t-1}$). Because we use large time intervals (3 to 5 years for opioid deaths), we use contemporaneous deaths, as it is more natural to assume that spillovers due to thicker markets

[30] Readers should refer to the references therein for the derivation of the estimator and its asymptotic distribution. A related estimation technique is to translate equation 4 into $Y_t = (I - \lambda W)^{-1}(\rho Y_{t-1} + X\gamma + \tau_t + \epsilon_t)$, which can be estimated by maximum likelihood (Case and Katz 1991). Using this alternative technique yields similar results.

[31] Opioid deaths are rare outcomes: e.g., the average county with 100,000 people would have experienced 5 deaths in 2010. For a small county with fewer people, there will be years where no deaths occur even with high opioid use. Averaging over a wider interval reduces such measurement error.

would occur within a few years period. If we were to use shorter intervals, it might make sense that death rates in one area would depend on lagged death rates in other areas (allowing enough time for spillovers through information or product flows to occur). Regardless, this choice does not meaningfully affect our estimates, as we discuss later.

For the estimation of opioid deaths and shipments models through 2010, the primary instruments we use are the variables related to where prescription opioid use increased: state triplicate prescription programs; mid-1990s cancer mortality rates; and 1990 disability rates. For the estimation after 2010 involving mainly illicit opioid deaths, the primary instruments we use are each county's average annual rate of prescription opioid shipments from 1997 to 2010 (denominated in MME per capita); heroin death rate (2005–2009); regional share of heroin seizures that were white powder (2004–2010); and heroin death rate interacted with the share of seizures that were white powder. In all the models, lagged opioid death rates in previous periods (which account for persistence) are also used as instruments.

VI. Estimates of Spillovers

We start by testing for spatial correlation of overall opioid death rates from 1990 to 2018. **Figure 4** shows visually a high degree of spatial correlation in opioid deaths. **Table 1** presents Moran I statistics which formally test for spatial correlation of death rates across areas, before and after conditioning on area-level factors that may influence opioid death rates and state fixed effects.[32] Across all specifications, the data show strong evidence of spatial correlation with respect to geographic distance and cross-county friendship patterns, even controlling for area characteristics and state fixed effects.

A. Determinants of the opioid epidemic's first wave

We next turn to analyzing determinants of opioid deaths in the first wave of the opioid epidemic, from 1996 to 2010. Results from estimating equation 5 are presented in **Table 2**. Models 1 to 3 use OLS and models 4 to 6 use GS2SLS to instrument for the spillover parameters. In each case, we have 9,351 observations, three for each county.

We start with the OLS results. While these may be biased by the presence of unobserved

[32]For a linear model of opioid death rates, $\text{Deaths} = \mathrm{X}\beta + \mathrm{u}$, the Moran I statistic (I) tests the null hypothesis that u_i are uncorrelated (i.e. $H_0 = \sigma^2 I$).

variables correlated across counties, comparing the OLS and GS2SLS estimates can indicate how large that bias is. Models 1 to 3 show that opioid death rates in one period are strongly related to opioid death rates in the prior period. The autocorrelation coefficient, $\hat{\rho}$, ranges from 0.52 to 0.90. In all cases, the estimate is statistically significantly less than 1. Thus, without further exogenous changes or spillovers, opioid epidemics would burn out over time.

The next row shows large spillovers in death rates across counties. In model 1, which uses distance weighting, the effect of more opioid deaths in geographically proximate areas is 70 percent as large as the effect of having more opioid deaths in the same county in the last period. In model 2, which uses friend-based weighting, the effect of more opioid deaths in socially connected areas is 70 percent larger than the effect of lagged death rates. When both measures are included in the model together, spillovers load on the friends-based term and the distance-based term becomes slightly negative. Because of the relatively high collinearity between the two measures (Pearson's correlation coefficient = 0.75), we interpret this as mild preference for the weighting measure based on friends, but this is difficult to determine firmly.

In columns (4) to (6), the exogenous variables that affect prescription opioid use are strongly related to the endogenous variable (opioid death rates). Being in a non-triplicate state and having a higher percent of adults who were disabled in 1990 are strongly related to growth in opioid overdose death rates, consistent with past research (Alpert et al. 2021; Cutler and Glaeser 2021). However, the direct effects of these variables on deaths are smaller than past research, which does not allow for these variables to have spillovers.[33] Cancer mortality rates are not significantly related to growth in mortality after we include the percent of adults who were disabled; however, as in Arteaga and Barone (2022), it is positively and significantly related to growth when it is included in the model by itself (see **Appendix Table B4**).

The coefficients on lagged death rates and deaths in other counties are very similar to the OLS estimates; the two are generally within one standard error of each other. Lagged opioid death rates are strongly related to current opioid death rates ($\hat{\rho} = 0.55$ to 0.91), depending on the specification). This estimate is again statistically significantly below 1, implying the opioid epidemic would have burned out without spillovers or further exogenous shocks. The GS2SLS estimates of spillovers are the same direction and very similar in magnitude to the OLS results—

[33] The coefficients for the non-triplicate prescription programs and disability rates are roughly double if we estimate equation 7 without the spillovers term and are closer to those in Alpert et al. (2021) and Cutler and Glaeser (2021).

implying limited bias in the OLS estimates. There are positive spillovers with both the distance and friends-based weighting metrics for deaths in other areas when included in the model separately, and spillovers load more on the friends-weighted deaths terms when both are included together. We return to what coefficients of this magnitude imply in subsection VI.C.

B. Determinants of prescription opioid shipments growth

In addition to opioid deaths, we also examine determinants of growth in opioid shipments during the first wave of the epidemic. As noted above, we analyze spillovers using the detailed ARCOS data from 2007 to 2009 (including the lag in 2006). We present spillover results for *OxyContin*, other prescription opioids, and all opioids separately, as two of our instruments relate specifically to *OxyContin* marketing. Results using the friends-based weighting are presented in **Table 3**. Starting with the first column, which reports results from OLS, we show that *OxyContin* shipments are highly persistent over time and that there are modest spillovers (22 percent as large as the direct effects of past shipments). There are also significant spillovers for other types of opioid shipments and opioid shipments overall, though the magnitudes are smaller (5 to 7 percent of lagged direct effects).

The instrumental variables results are similar. As in prior literature, non-triplicate states experienced higher growth rates of shipments of *OxyContin*, as well as other opioids (particularly non-*OxyContin* oxycodone). Impacts on other types of opioid shipments are even larger than impacts on *OxyContin*. Alpert and colleagues (2021) interpret this as spillovers of *OxyContin* marketing onto other types of oxycodone. We also find cancer mortality rates were unrelated to growth in *OxyContin* shipments and predominantly affected growth of other types of opioids (particularly non-*OxyContin* oxycodone).[34] Disability rates were generally positively related to shipments, but not significantly so with the other variables in the model.[35]

As with mortality, the spillover estimates in the instrumental variables models are very similar to the OLS estimates. The persistence effect is near 1 on an annual basis, and spillover effects are about 0.05 to 0.15 as large. Results using distance-based weighting for the spillovers

[34] It is possible this is due to *OxyContin* growth being slower over this period, ten years after it was initially approved, and spillovers to other types of opioids were more important over this time. Alternatively, the variable may be picking up factors related to more general increases in oxycodone use. Whether or not we include this variable does not affect our main estimates of spillovers in opioid deaths (see section VII).

[35] Disability rates are strongly related to opioid shipments growth over a longer time-period (e.g., 1999 to 2010), as shown in Cutler and Glaeser (2021). They are less related to growth over this shorter period after 2006.

term are shown in **Appendix Table B1** and imply spillovers of similar magnitudes.

C. Determinants of deaths after 2010

Next, we examine spillovers associated with deaths after 2010, which entailed sharply rising illicit opioid deaths. In these models, we again have three observations for each county, each with a three-year average opioid death rate.

Table 4 shows the results. As with Table 2, we first report OLS estimates (columns 1 to 3) and then equivalent GS2SLS estimates (columns 4 to 6). Starting with the OLS estimates, lagged county-level opioid death rates are highly related to current opioid death rates, but the relationship is again statistically significantly less than 1. The coefficients are a bit smaller here than in the earlier period ($\hat{\rho} = 0.44$ to 0.71), reflecting somewhat lower persistence.[36] This may be due to more variability in toxicity of opioid products in this era. Illicit opioids are more variable in potency relative to prescription opioids, which may lead to quicker deaths of the drug using population. All else equal, this would be expected to cause epidemics to burn out more quickly. We also estimate large spatial spillovers in this period. The coefficients on spillovers which rely on distance between counties are larger than in the legal era, with spillovers 88 percent as large as the effects of past opioid deaths on present opioid deaths. Spillovers that use friendship-based weighting are even larger, nearly twice the direct effect of past opioid deaths.

The results from the instrumental variables estimates are presented in the last three columns. Consistent with prior research, historical opioid shipments (including *OxyContin*) are strongly related to the increase in illicit opioid death rates (Alpert et al. 2018; Cutler and Glaeser 2021; Evans et al. 2019). Heroin death rates prior to 2010, particularly in areas where white powder heroin was more common, are also strongly related to growth in death rates after 2010. Using these variables to instrument for spillovers, we obtain similar results to the OLS models. In the models where distance and friend spillovers are separate, spillovers through friend relationships across counties are 48 percent larger than the effects of having higher opioid death rates in the past. Spillovers through geographic distance are 70 percent as large. We also confirm that spillover estimates are similar if we use the instruments that affected prescription opioid usage in the first period (1996 to 2010) rather than actual opioid shipments (see **Appendix Table B2)**.

[36] The same annual persistence would be associated with a higher correlation using three-year averages than using five-year averages.

D. The Implication of Thick Market Effects

Because the model has a dynamic component, the long-run impacts of social spillovers cannot be determined without additional analysis. Accordingly, we use the estimates in Tables 2 and 4 to simulate various equilibria.

Table 5 translates the GS2SLS estimates of equation 5 to show three effects and their 95% confidence intervals. The first is the persistence (or autocorrelation effect). This comes directly from equation 7, i.e. $\hat{\rho}$. The second is the spillover effect of a hypothetical shock that increases opioid death rates by 1 death per 100,000 in the average county. We define this effect as follows:

$$\text{Average spillover effect} = \frac{\hat{\lambda}}{N}\sum_{i=1}^{N}\sum_{j\neq i}^{N} w_{i,j}\,. \tag{8}$$

The third effect is the multiplier of the effect of current death rates on death rates in the next period, accounting for both persistence and spillover effects. This is defined as:

$$\text{Average next period multiplier} = \hat{\rho}\left(1 + \frac{\hat{\lambda}}{N}\sum_{i=1}^{N}\sum_{j\neq i}^{N} w_{i,j}\right). \tag{9}$$

Intuitively, this multiplier depreciates (by the persistence parameter) the sum of each death in the previous period plus the spillovers it had. If equation (9) is close to 1, the epidemic will be very persistent and temporary shocks will have long-term effects on death rates. If it is greater than 1, the epidemic will increase in perpetuity, even without any exogenous changes in tastes or drug supply. For both equations 8 and 9, we obtain 95% confidence intervals using the Delta method.

The first row of **Table 5** shows the persistence effects and their 95% confidence intervals. The second row shows the averages spillover effect of a hypothetical shock that increases death rates by 1 death per 100,000 in the average county. This causes 0.38 to 0.76 deaths per 100,000 in other counties, depending on how we measure spillovers. The next row shows how this affects the epidemic's dynamics, evaluating equation (9). In all cases, the estimate is close to 1 (ranging from 0.78 to 1.24)—implying that spillovers will cause any temporary shocks to have long-lasting effects. In some of the models, shocks will have permanent effects, as the dynamics are unstable (the next period multiplier is > 1). Thus, the combination of spillovers and addictiveness will cause deaths to increase in perpetuity.

To show this directly, we run two simulations that increase opioid death rates by 1 death per 100,000 in the average county: one using estimates from model 1 (where the impact in the next period is greater than 1 death) and one using estimates from model 3 (where the impact in the next period is just below 1). We illustrate how the simulation is done in **Figure 8**, using the numbers from model 1. We consider a hypothetical shock that initially increases deaths by 1 per 100,000. This will cause opioid use to rise in other countries due to spillovers, and thus 0.38 further deaths in those counties. The total effect on deaths initially is 1.38. Because use is addictive and death rates are persistent, both sets of areas will have a higher death rate in the next period ($1 \times 0.89 = 0.89$ in the initially affected counties and $0.38 * 0.89 = 0.34$ in the areas where the shock spilled over). Both those sets of deaths will then cause further spillover effects: $(0.89 + 0.34) \times 0.38 = 0.47$, leading deaths to rise to 1.70 (0.89 + 0.34 + 0.47), higher than they were initially. This continues to play out over time and will not stop unless there is some shock to reverse it or there are no further populations for the epidemic to spread to.

We plot this graphically in **Figure 9** panel A. The blue line shows the effects of the initial shock on death rates, and its persistence in affected counties. This is the only exogenous increase in deaths in the simulation. Due to persistence of death rates across periods, opioid deaths remain higher in areas affected by the initial shock in subsequent periods but ultimately asymptote towards zero. However, the shock also leads to spillovers. These persist and cause additional spillovers in subsequent years. Though each manifestation of shocks and spillovers fade, they fade slowly, and the combined effect of this is a continuous and exponential increase in opioid death rates. Six periods later, the vast majority (85 percent) of deaths are due to spillovers, not the initial shock.

Figure 9 panel B shows the predicted path of the shock based on model 3, which results in a declining (but slowly declining) epidemic. The same process that played out before plays out again. However, the spillovers are not large enough to override the decline in deaths in subsequent periods. Deaths asymptote back towards 1, though much more slowly than if spillovers were not occurring. Like panel A, six periods later, most deaths (96 percent) are the result of spillovers and not the initial shock.

These results show that the combination of addictiveness and spillovers are large enough to cause epidemics to persist for long periods of time after initial shocks fade away. To quantify the importance of spillovers in the actual time series of opioid deaths, we use our model estimates to decompose the share of actual deaths that can be explained by spillovers and their dynamic effects. Starting in the first time-period for each of our two estimation samples, we predict death rates setting the spillover parameter from equation (4) (λ) to 0. We then simulate death rates in the next period, using the other parameters of the model. We continue this process until the most recent year of each phase (2010 or 2018).

Results are shown graphically in **Figure 10**, for the prescription opioid period (panel A) and the more recent illicit opioid period (panel B). We use models 3 and 6 from the table, which include both friend- and distance-based spillovers. Starting with prescription opioid wave in panel A, the predicted path of the epidemic without spillovers is shown in the dashed blue line. The path increases through the 2001–2005-time interval because of the coefficients on the exogenous variables and then starts to decline. The impact of spillovers of these variables (including dynamic spillovers) is shown in the dashed red line, which grows sharply over time and accounts for the bulk of total opioid death rates. From 1996 to 2000, around a quarter of deaths are due to the direct effects of exogenous variables. By the 2006–2010 period, deaths would have been declining if not for the impacts of spillovers, and spillovers account for 93 percent of deaths. For the illicit period (shown in panel B), deaths decline even more rapidly in the scenario without spillovers—the exogenous variables driving higher death rates are not sufficiently large to sustain increases. However, as their effects are amplified by spillovers, deaths keep increasing to higher levels. In the final period, from 2016–2018, 99 percent of deaths are due to spillovers.

The overall share of opioid deaths that are explained by spillovers in each of our different models is shown in the bottom row of **Table 5**. In all cases, spillovers explain most opioid deaths since 1990. In our preferred models that include the stronger friend-based spillovers, spillovers explain essentially all of them, upwards of 84 percent.

VII. Robustness Checks

Our results are robust to alternative estimation strategies. First, rather than basing spillovers on contemporaneous deaths in other areas, we base them on lagged death rates in other areas. As mentioned in section V, this may be more appropriate if it takes a long time for information spillovers to occur. Results, which we estimate using OLS, are reported in **Appendix Table B3**. The coefficients are very similar to the models which relate opioid death rates to weighted averages of contemporaneous death rates. In several cases, they are even larger. In models 1 to 3, spillovers range from 20 to 224 percent larger than the direct effects of having higher death rates in the past. In models 4 to 6, spillovers range from 96 percent as large to 179 percent larger. In these models, even when friend and neighbor spillover terms are included in the model, both remain positive and significant: with the model still preferring the friend-based spillover terms.

Second, we study whether there are cross-substance spillovers—i.e., spillovers from heroin deaths in an index county on non-heroin deaths in other counties. While some such spillovers are to be expected, we suspect they would be smaller than spillovers of the same substance. We focus on the illicit wave of the epidemic, where the specific type of opioid is known in all years. We analyze results for heroin and fentanyl separately. We do not have separate instruments for heroin and fentanyl; however, earlier regressions indicate there is limited bias in the OLS estimates relative to the instrumental variables ones. Thus, we use OLS models for this exercise.

Results are reported in **Table 6** and show spillovers are specific to the type of opioid being used. Heroin death rates in other socially connected counties matter much more for heroin death rates in one's own county than prescription opioid or fentanyl death rates. Similarly, fentanyl death rates in other socially connected counties are more likely to cause increases in fentanyl death rates than increases in heroin or prescription opioid death rates in socially connected counties. This supports the notion of these being externalities due to opioid markets becoming thicker—when heroin use increases locally, it spills over and causes increases in heroin use elsewhere. The same is true for fentanyl.

Finally, we test whether our results are robust if we use different combinations of instruments to identify spillovers. Starting with the prescription opioid wave, we separately consider each of the instruments for initial *OxyContin* marketing (triplicate prescription and cancer death rates), and the 1990 share of workers that received disability benefits, along with two other potential instruments: the 2005 to 2007 share of adults aged 25–64 that did not have a college degree, and predicted increases in import competition from China based on 1990 industry shares. We also include all these variables in the model together.

Results are shown in **Appendix Table B4**. Four of the five variables (triplicate prescriptions, cancer death rates, the share disabled, and the share without a college degree) are positively related to opioid deaths. The last variable, predicted import competition from China, is negatively related to growth in opioid death rates. This is the opposite sign from prior literature

(Autor et al. 2013) and results from including the spatial correlation terms.[37] In all of these models, the estimates of spillovers through friend relationships are remarkably stable, varying no more than 7 percent. The last column, which includes the variables jointly, shows that triplicate prescription legislation and the share of the population that is disabled are the strongest predictors of opioid death rates.

For the illicit opioid wave, we look at results if we use each instrument separately, as well as if we split opioid shipments into *OxyContin* vs. non-*OxyContin* opioids. The opioid shipments variables are meant to pick up substitution from these drugs to heroin after 2010, and to see whether it differs for *OxyContin* or other types of prescription opioids. Our results, shown in **Appendix Table B5**, show that both pre-2010 *OxyContin* and pre-2010 non-*OxyContin* shipments were related to growth in opioid deaths during the illicit opioid wave (columns 1 and 2). Again, our estimates of spillovers are very stable regardless of which sets of instruments we use. Overall, they vary by no more than 13 percent across specifications and are large in all cases.

VIII. Conclusions

This paper studies why opioid overdose death rates have been rising nearly continuously in the U.S. for the past thirty years. Historically, there are a mix of long and short drug epidemics. Some epidemics last no more than a decade, while the opioid epidemic continues to expand even three decades later.

Our main finding is that we can explain the prolonged and substantial increase in opioid deaths by reference to thick market spillovers: as opioid markets become thicker, information flows and ease of obtaining the substances lead people to use more of them. We demonstrate theoretically and empirically that in the presence of addiction, spillovers can create unstable equilibrium that may lead to perpetual increases in epidemic death rates. Even if not perpetually increasing, death rates with spillovers and addiction can increase for a long time. In our preferred models, spillovers explain most opioid deaths since 1990 and are the main reason the epidemic has not burned out.

These findings have important implications for economics and policy. They show that even

[37] We obtain similar results to Autor, Dorn, and Hanson (2013) if we estimate equation 7 without the spillovers term. We also obtain similar results if we replicate Autor, Dorn, and Hanson's long differences specification, relating changes in opioid death rates from 1990–1995 to 2006–2010 to predicted increases in import competition from China.

temporary misconduct or mistakes on the part of regulators or suppliers can lead to long-term harms, even well after the initial mistake or misconduct has ended. It is important for policymakers to take this potential into account when regulating addictive products.

The results also show how rational models of addiction can be extended to explain drug epidemics. When goods are addictive and there are spillovers, use will persist at higher levels in society than one would predict through addiction alone. Understanding how the opioid epidemic differs from other epidemics, some of which had shorter durations, is a key issue following from our research.

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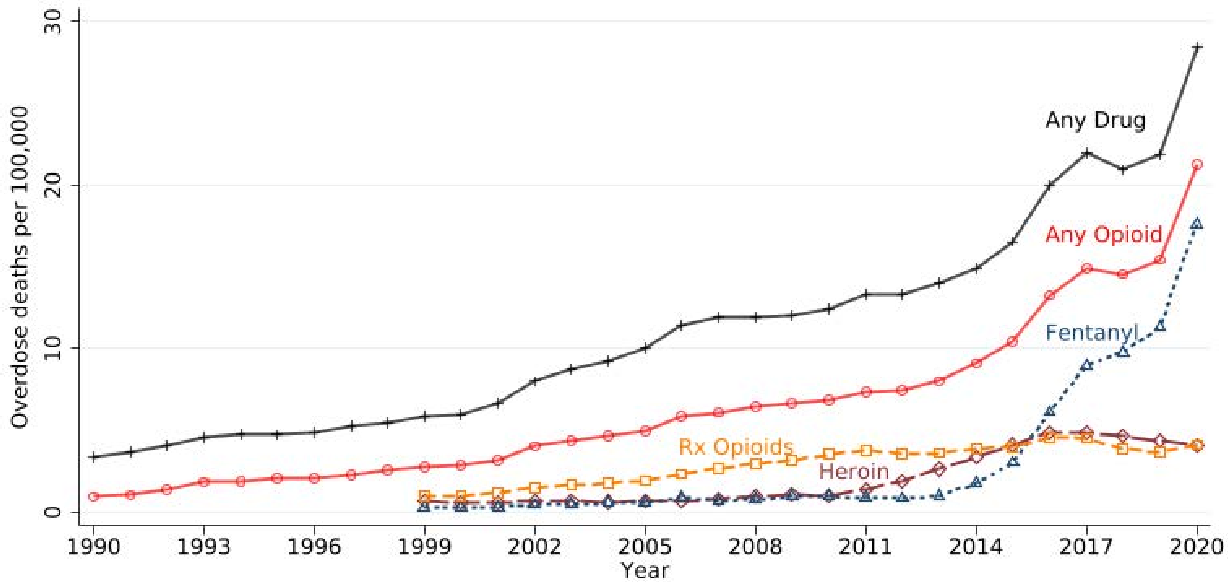
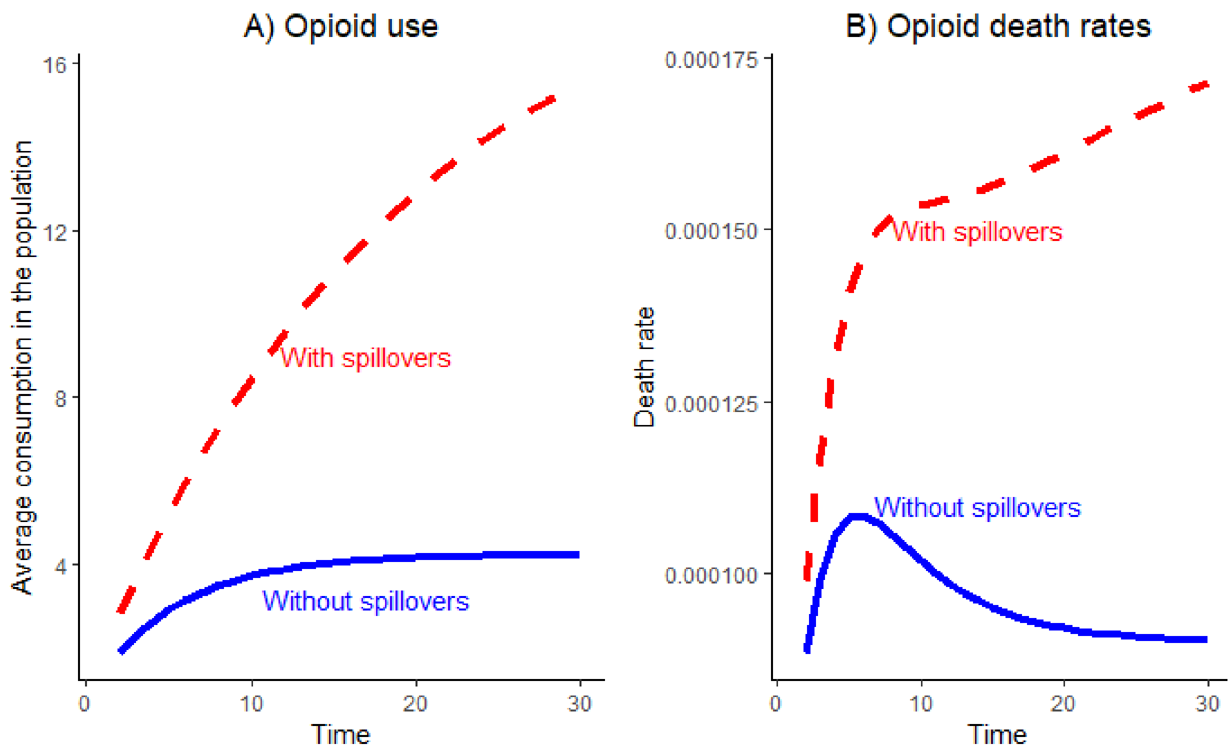
FIGURES

Figure 1: Trends in drug and opioid overdose deaths per 100,000, 1990 to 2020



Notes. Data on overdose deaths are from the National Center for Health Statistics, age and sex adjusted to the U.S. population in 2010. Cause of death codes that were used to identify overdose deaths (overall and by cause) are described in section III.A of the text.

Figure 2: A hypothetical opioid epidemic with and without spillovers

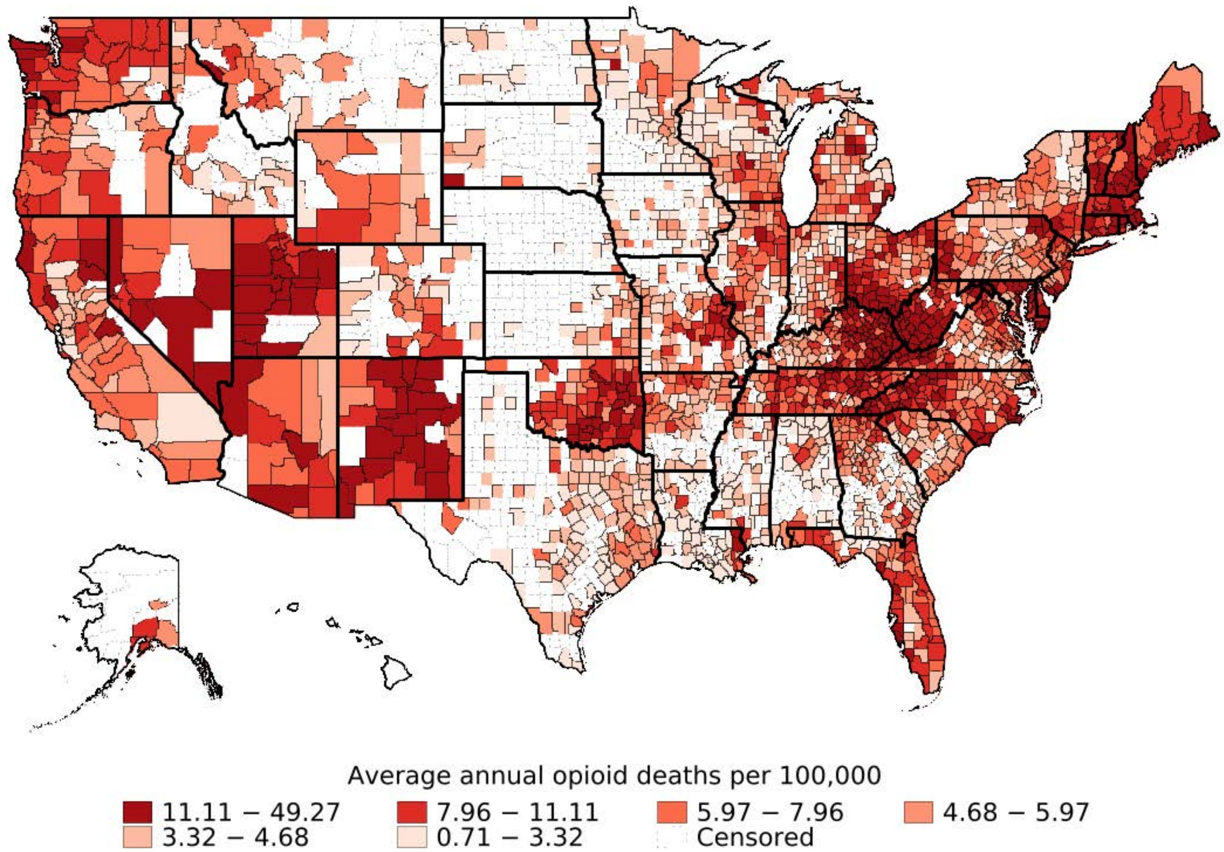


Notes. See section II for model details and **Appendix A** for specific parameters. Spillovers (b_g) are 80 percent as large as the effect of past use on current use ($b_{as}(1-d)/d$).

Figure 3: Trends in real heroin prices and heroin death rates, 1999 to 2020

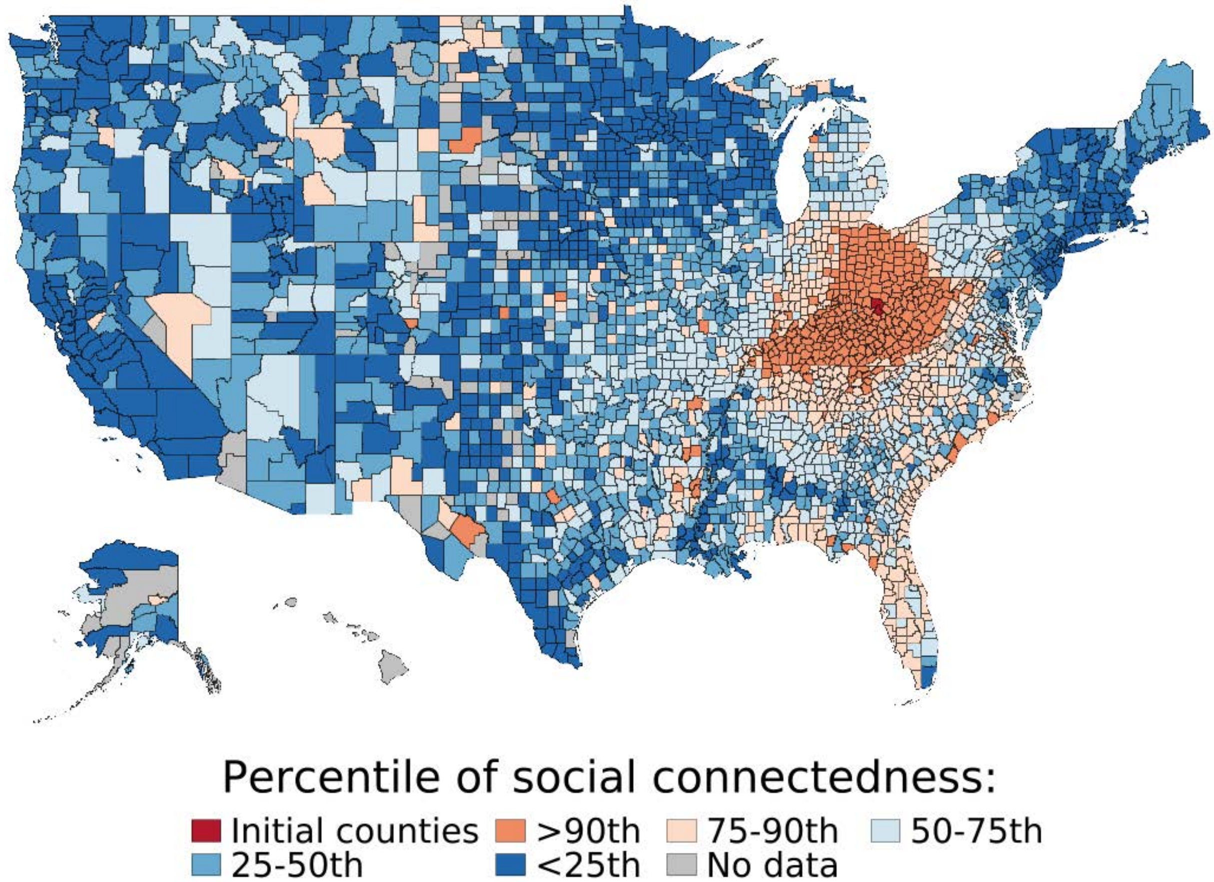
Notes. Data on heroin prices are based on drug seizures made by the US Office of Drug Control Policy (ONDCP) and are adjusted for purity, available at: https://dataunodc.un.org/dp-drug-prices-Europe-USA. Data on age- and sex-adjusted heroin death rates are from the National Vital Statistics System and described in section III.

Figure 4: Map of Average Annual Opioid Deaths Per 100,000, 1990 to 2018



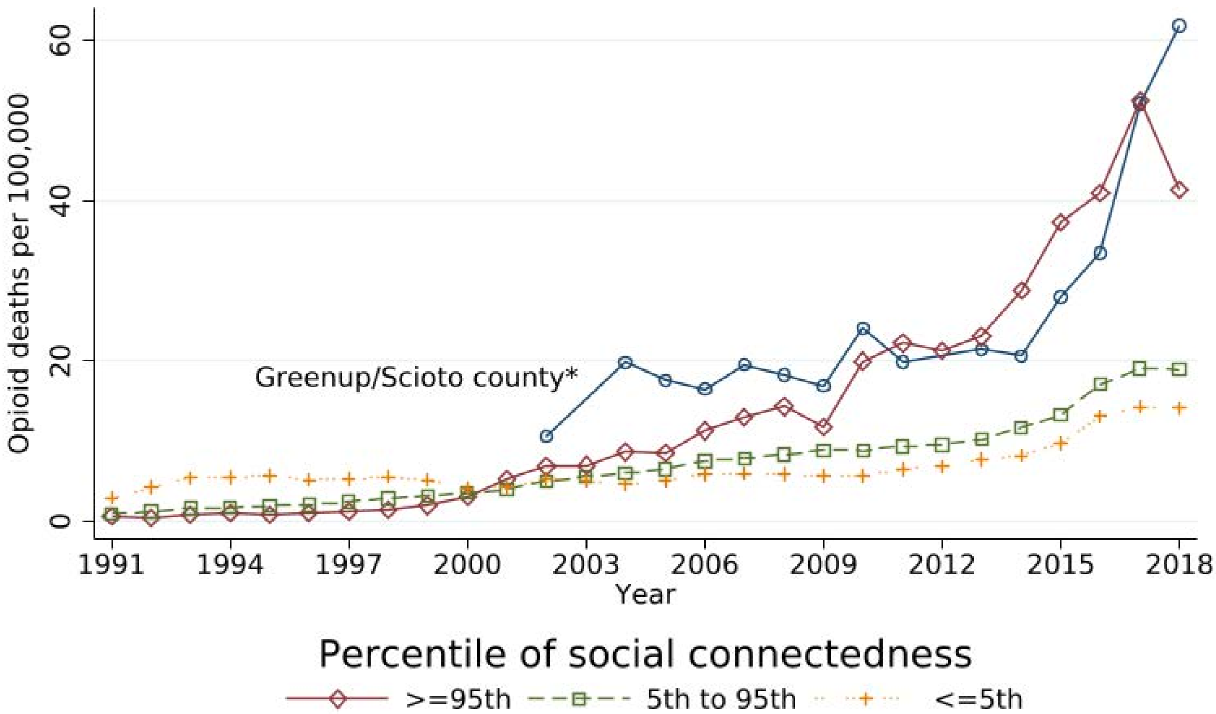
Notes. Data are from the National Vital Statistics System. Death rates in counties with fewer than ten total opioid overdose deaths over the period are censored, as per NCHS requirements.

Figure 5: Distribution of social connectedness to Greenup County, Kentucky, and Scioto County, Ohio



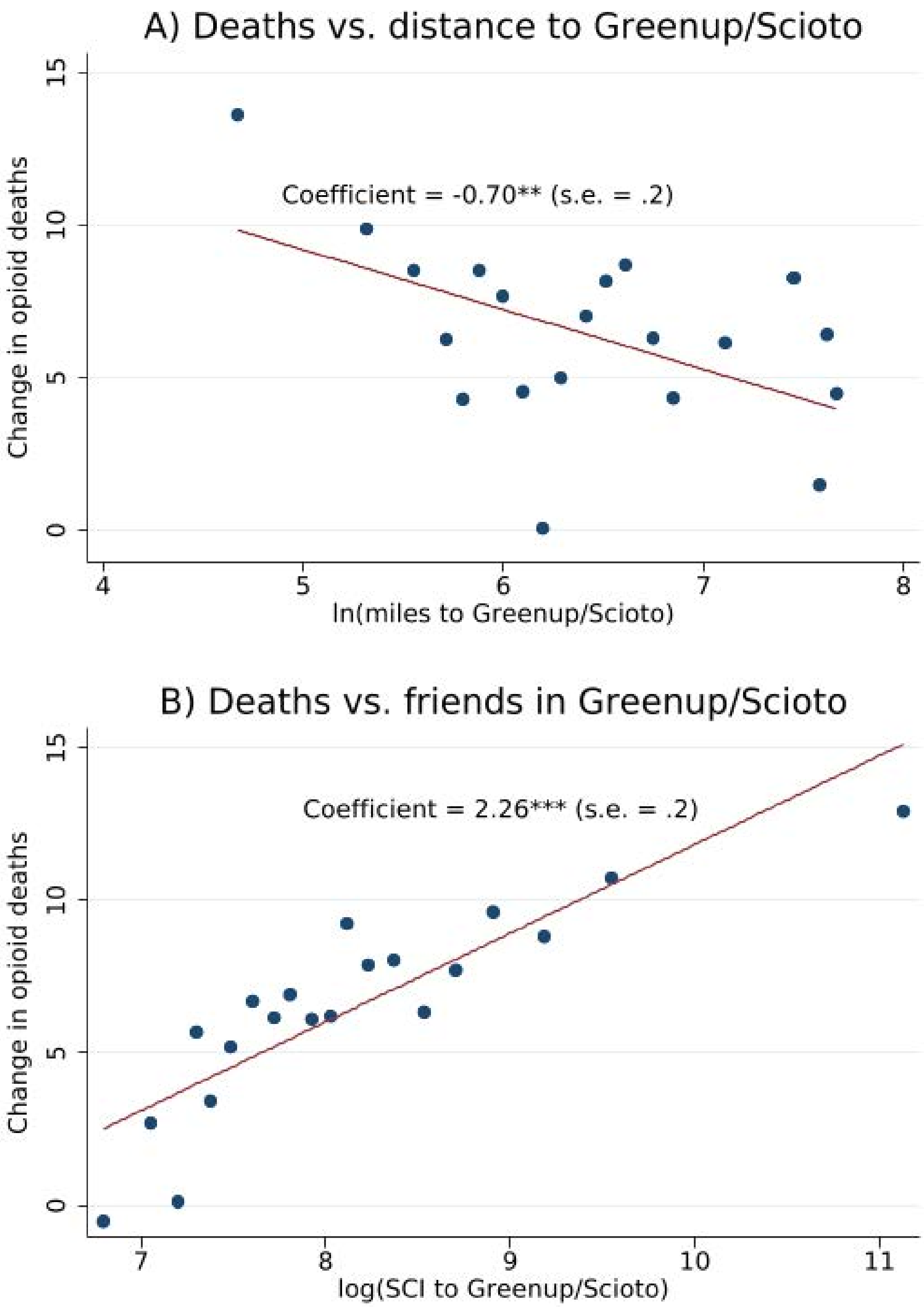
Notes. The figure plots the percentile of social connectedness between each county in the U.S. and Greenup, KY/Scioto, OH, where initial pill mills were established.

Figure 6: Trends in opioid death rates in Greenup County, Kentucky, and Scioto County, Ohio, and socially connected counties, 1990 to 2018



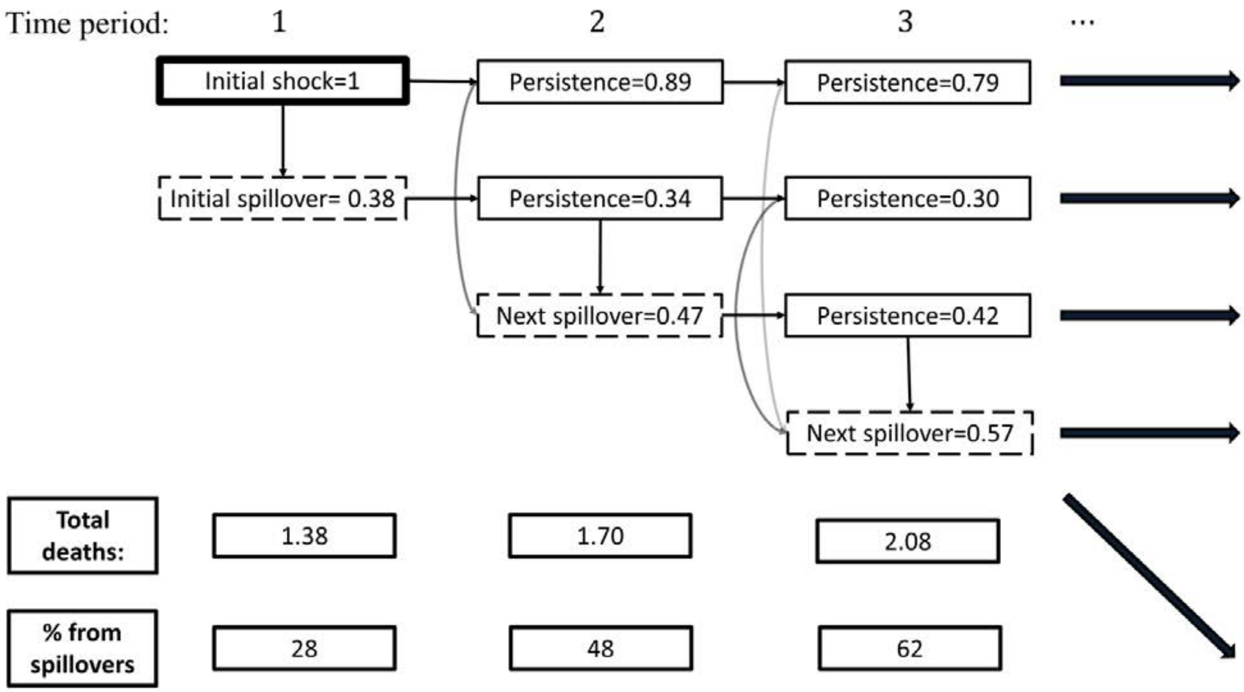
Notes. Data are from the National Vital Statistics System. Rates are censored when fewer than 10 total deaths occurred. The figure present trends in opioid deaths for Greenup County, Kentucky, and Scioto County, Ohio, where initial pill mills were established. It also presents trends for counties below the 5th, between the 5th to 95th, and above the 95th percentile of social connectedness to the counties Greenup and Scioto Counties. * Deaths are censored when fewer than 10 total deaths occurred.

Figure 7: Opioid deaths increased more in areas close to and with more friends in initial pill mill counties



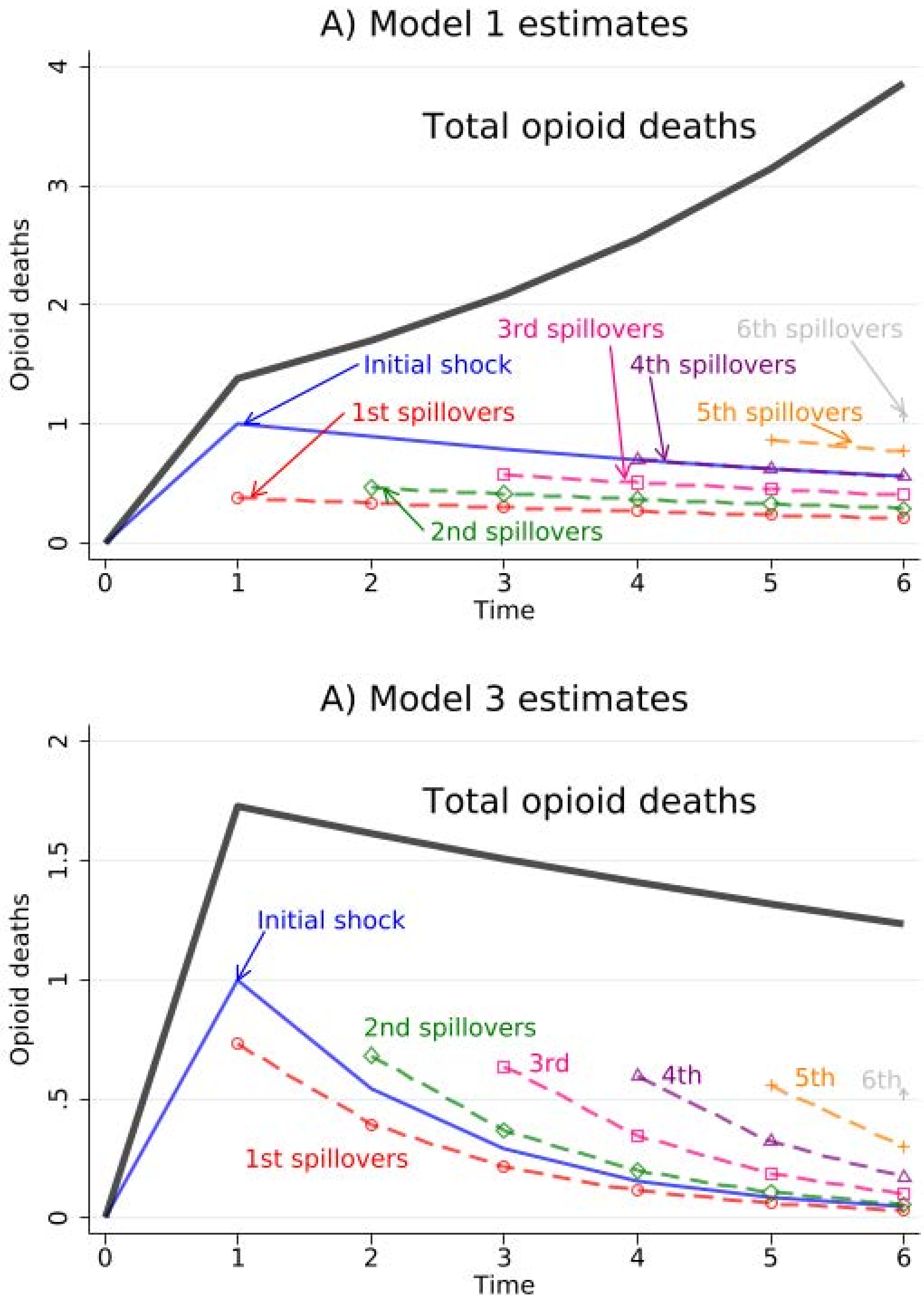
Notes. Binned scatter plots of log miles to Greenup or Scioto county (whichever is closer) (A) and log social connectedness to Greenup and Scioto counties (B) vs. the change in opioid deaths per 100,000 people from 1992-95 to 2008-11. See section IV of the text for more details.

Figure 8: Quantifying the dynamic effects of an exogenous shock to opioid use over time



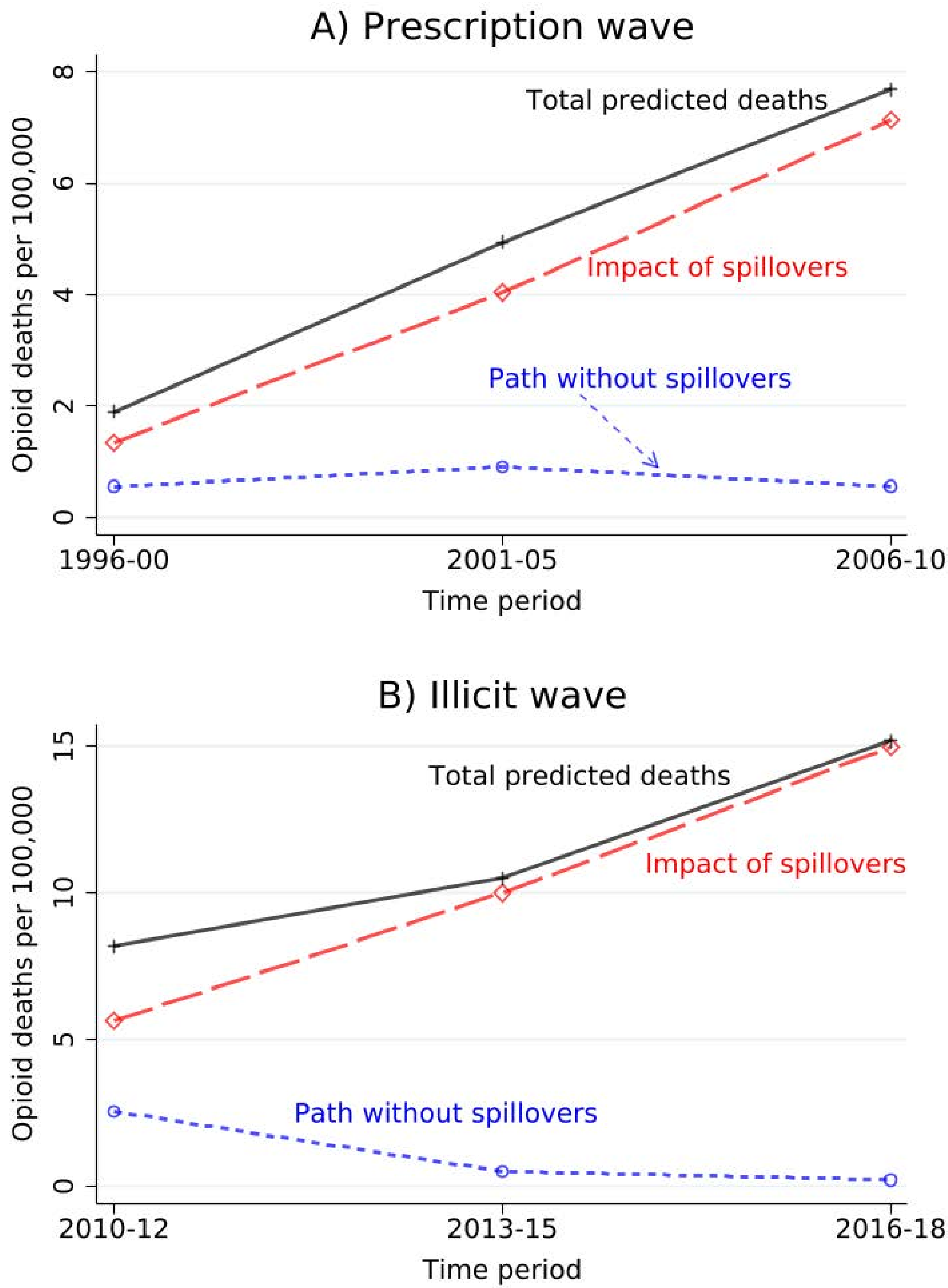
Notes. Simulates death rates based on estimates from **Table 5** model 1. We consider a hypothetical shock that increases deaths by 1 in the initial period. This has spillover effects of 0.38. Both have some persistence (at rate 0.89) and then further spillovers in the future. See Section VI for more details.

Figure 9: Predicted paths of epidemics following an initial shock to opioid death rates



Notes. Figure plots the effects of an exogenous shock to opioid use (which leads to 1 death) in the first period. The blue line shows how death rates persist at higher rates in initially affected counties, due to opioid use being addictive and persistent. Each successive dashed line with markers shows a wave of spillovers (from higher use persisting in other areas), and how each of these persist (but fade) over time. The two simulations use models 1 (A) and 3 (B) of **Table 5**. See also **Figure 8** for numerical estimates for the first three waves of panel A.

Figure 10: Contribution of spillovers to overall opioid death rates, 1996–2018



Notes. Figure plots how much total opioid death rates (the solid black line) are explained by the direct effects of exogenous variables, lagged opioid death rates, and year fixed effects (the dashed blue line) and spillovers (the dashed red line) over time, separately for the phases of the epidemic that involved primarily prescription opioids (1996–2010) and illicit opioids (2010–2018). The simulations use models 3 and 6 from **Table 5**. See section VI.D for more details.

TABLES

Table 1: Spatial correlation of counties' average annual opioid deaths and shipments

	Weighting Based On:				
	Distance	Friendships	Distance + friendships		
	(1)	(2)	(3)	(4)	(5)
Moran I statistic	33,602.7	7,010.5	33,748.0	23,858.6	2,925.6
(p-value)	<0.001	<0.001	<0.001	<0.001	<0.001
Controls:					
State triplicate prescription program				X	X
1994–96 cancer mortality rate				X	X
Share with a college degree (2005–09)				X	X
Share males age 25–64 not working (05–09)				X	X
Share not married (05–09)				X	X
Share employed in mining (05–09)				X	X
Poverty rate (05–09)				X	X
State fixed effects					X
Number counties	3,117	3,117	3,117	3,117	3,117

Notes. The sample is all U.S. counties. The table presents Moran I statistics for spatial correlation of opioid death rates and shipments in terms of geographic proximity (model 1), social networks (model 2), and both geographic proximity and social networks jointly (models 3–5). P-values test the null hypothesis of no spatial correlation in opioid death rates. See section VI.a.

Table 2: The impact of opioid death rates in peer and neighboring counties on own opioid death rates, 1996 to 2010

	Ordinary least squares			*Spatial two-stage least squares*		
	(1)	(2)	(3)	(4)	(5)	(6)
Opioid deaths$_{t-1}$ (ρ)	0.90***	0.54***	0.52***	0.89***	0.59***	0.54***
	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Death spillovers (distance) (λ_d)	0.64***		-0.13***	0.42***		-0.18***
	(0.04)		(0.04)	(0.04)		(0.04)
Death spillovers (friends) (λ_f)		0.92***	0.97***		0.76***	0.90***
		(0.03)	(0.04)		(0.04)	(0.05)
<u>Instruments</u>						
Non-triplicate state				0.27**	0.30***	0.37***
				(0.11)	(0.09)	(0.10)
1994–96 cancer mortality rate				-0.09**	0.05	0.05
				(0.04)	(0.04)	(0.03)
1990 percent disabled				0.26***	0.14***	0.14***
				(0.03)	(0.03)	(0.02)
<u>Year (rel. 1996–00)</u>						
Year = 2001–05	0.38***	-0.07	0.15	0.96***	0.30**	0.44***
	(0.14)	(0.11)	(0.13)	(0.15)	(0.13)	(0.14)
Year = 2006–10	-0.91***	-1.21***	-0.69***	0.35	-0.51**	-0.11
	(0.28)	(0.18)	(0.24)	(0.27)	(0.22)	(0.25)
N (counties x intervals)	9,351	9,351	9,351	9,351	9,351	9,351
R^2 (1–3)/Pseudo R^2 (4–6)	0.50	0.61	0.61	0.49	0.53	0.52

Notes. Estimates of equation 7. The sample is all U.S. counties, and the dependent variable is the county's average annual opioid death rate per 100,000 people. Data are pooled from 1996 to 2010 in 5-year intervals (1996–2000; 2001–2005; and 2006–2010). Data from 1991–1995 also serve as a lag period for the 1996–2000 period. Models 1 to 3 use OLS. Models 4 to 6 use GS2SLS. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denotes p<0.01(0.05)0.1. See section V and Appendix A for more details.

Table 3: The impact of opioid shipments in peer counties on own opioid shipments, 2007 to 2009

	Ordinary least squares			*Spatial two-stage least squares*		
	OxyContin	*Other opioids*	*All opioids*	*OxyContin*	*Other opioids*	*All opioids*
MME per capita$_{t-1}$ (ρ)	0.96***	1.07***	1.06***	0.97***	1.05***	1.06***
	(0.01)	(0.02)	(0.02)	(0.01)	(0.02)	(0.02)
MME spillovers (friends) (λ_f)	0.21***	0.05***	0.07***	0.14***	0.06***	0.06***
	(0.02)	(0.01)	(0.02)	(0.02)	(0.02)	(0.02)
Instruments						
Non-triplicate state				1.34**	3.73***	4.53***
				(0.63)	(1.32)	(1.38)
1994–96 cancer mortality rate				0.10	1.79***	1.90***
				(0.22)	(0.58)	(0.61)
1990 percent disabled				-0.09	1.06	0.51
				(0.14)	(0.64)	(0.59)
Year (rel. 2007)						
Year = 2008	16.94***	-31.3***	-11.63***	18.09***	-30.6***	-11.35***
	(0.69)	(1.61)	(1.58)	(0.69)	(1.67)	(1.60)
Year = 2009	-5.32***	-6.83***	-12.78***	-4.75***	-6.32***	-12.18***
	(0.63)	(1.65)	(1.77)	(0.63)	(1.65)	(1.73)
N (counties x years)	9,351	9,351	9,351	9,351	9,351	9,351
R^2 (1–3)/Pseudo R^2 (4–6)	0.87	0.95	0.96	0.87	0.95	0.96

Notes. Estimates of equation 7. The sample is all U.S. counties, and the dependent variable is the county's average annual rate of morphine milligram equivalent shipments per capita: for *OxyContin*, other prescription opioids (non-*OxyContin* oxycodone, hydrocodone, codeine, fentanyl base, hydrocodone, hydromorphone, and morphine), and all opioids. Data are from 2007 to 2009 and in 1-year intervals. Data from 2006 serve as the lag period for 2007. Models 1 to 3 use OLS. Models 4 to 6 use GS2SLS. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denotes p<0.01(0.05)0.1. See section V and Appendix A for more details.

Table 4: The impact of opioid death rates in peer and neighboring counties on own opioid death rates, 2010 to 2018

	Ordinary least squares			*Spatial two-stage least squares*		
	(1)	(2)	(3)	(4)	(5)	(6)
Opioid deaths$_{t-1}$ (ρ)	0.71***	0.44***	0.44***	0.63***	0.46***	0.44***
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)
Death spillovers (distance) (λ_d)	0.63***		-0.05*	0.44***		-0.04
	(0.03)		(0.03)	(0.03)		(0.03)
Death spillovers (friends) (λ_f)		0.86***	0.89***		0.68***	0.78***
		(0.03)	(0.03)		(0.03)	(0.03)
Instruments						
Prescription opioid doses per capita (1997–2010)				0.48***	0.19***	0.14***
				(0.04)	(0.04)	(0.04)
Heroin death rate (2005–09)				0.61***	0.48**	0.47**
				(0.22)	(0.22)	(0.22)
Share white powder heroin (2004–2010)				0.76***	0.36	0.04
				(0.39)	(0.32)	(0.35)
Heroin death rate × share white powder heroin				1.93***	1.58***	1.51***
				(0.42)	(0.41)	(0.41)
Year (rel. 2010–12)						
Year = 2013–15	-0.15	-0.10	-0.05	0.23	0.14	0.09
	(0.20)	(0.18)	(0.18)	(0.20)	(0.18)	(0.18)
Year = 2016–18	-0.001	0.13	0.29	1.16***	0.88***	0.69***
	(0.25)	(0.22)	(0.22)	(0.26)	(0.23)	(0.24)
N (counties x intervals)	9,351	9,351	9,351	9,351	9,351	9,351
R^2 (1–3)/Pseudo R^2 (4–6)	0.50	0.59	0.59	0.51	0.54	0.53

Notes. Estimates of equation 7. The sample is all U.S. counties, and the dependent variable is the county's average annual opioid death rate per 100,000 people. Prescription opioid doses are defined as 50 morphine milligram equivalents. Data are pooled from 2010 to 2018 in 3-year intervals (2010–2012; 2013–2015; and 2016–2018). Data from 2007–2009 serve as the lag period for 2010–2012. Models 1 to 3 use OLS. Models 4 to 6 use GS2SLS. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denotes p<0.01(0.05)0.1. See section V and Appendix A for more details.

Table 5: Implied dynamics of opioid death rates, accounting for persistence and spillovers

	Prescription opioid era: 1990–2010			*Illicit opioid era: 2010–2018*		
	Distance	Friends	Both	Distance	Friends	Both
	(1)	(2)	(3)	(4)	(5)	(6)
Persistence ($\hat{\rho}$)	0.89 [0.83, 0.96]	0.59 [0.53, 0.65]	0.54 [0.48, 0.60]	0.63 [0.37, 0.51]	0.46 [0.42, 0.51]	0.44 [0.39, 0.48]
Average spillover $\left(\frac{\hat{\lambda}}{N}\sum_{i=1}^{N}\sum_{j\neq i}^{N} w_{i,j}\right)$	0.38 [0.31, 0.46]	0.76 [0.67, 0.84]	0.73 [0.65, 0.82]	0.40 [0.34, 0.46]	0.68 [0.61, 0.74]	0.75 [0.68, 0.81]
Next period multiplier $\hat{\rho}\left(1+\frac{\hat{\lambda}}{N}\sum_{i=1}^{N}\sum_{j\neq i}^{N} w_{i,j}\right)$	1.24 [1.12, 1.35]	1.04 [0.95, 1.14]	0.94 [0.84, 1.04]	0.89 [0.82, 0.95]	0.78 [0.71, 0.84]	0.76 [0.69, 0.83]
Unstable dynamics?	Yes	Yes	No	No	No	No
% of deaths due to spillovers	57%	92%	86%	62%	84%	90%

Notes. Reports the average persistence effect, spillover effect, and next period effect of a shock that increases opioid death rates by one initially. Results use the instrumental variables regressions (Tables 2 and 4, models 4 to 6). Robust 95% confidence intervals, which use the delta method, are shown in brackets. If the next period effect is greater than one, the epidemic has unstable dynamics, and deaths will increase in perpetuity. The bottom row re-simulates death rates without spillovers in any period and quantifies the percent of deaths due to spillovers and their dynamic effects.

Table 6: Spillovers in opioid death rates across areas are specific to the type of opioid used.

Index Deaths:	*Heroin Deaths*				*Fentanyl Deaths*			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Index deaths$_{t-1}$ (ρ)	0.55***	0.87***	0.72***	0.47***	0.61***	0.30***	0.70***	0.33***
	(0.04)	(0.04)	(0.04)	(0.04)	(0.06)	(0.03)	(0.06)	(0.04)
Spillovers (friends weighting)								
Heroin deaths other counties	0.91***			1.00***	1.64***			0.12**
	(0.04)			(0.05)	(0.06)			(0.05)
Fentanyl deaths other counties		0.27***		-0.09***		1.26***		1.25***
		(0.02)		(0.03)		(0.04)		(0.05)
Rx opioid deaths other counties			0.13***	0.02			0.41***	-0.10***
			(0.01)	(0.01)			(0.03)	(0.02)
Year (rel. 2010–12)								
Year = 2013–15	0.14***	1.21***	1.10***	0.06	-1.43***	-0.13*	0.65***	-0.25***
	(0.07)	(007)	(0.07)	(0.07)	(0.12)	(0.08)	(0.09)	(0.09)
Year = 2016–18	-0.50***	1.10***	0.10	-0.33	1.01***	-0.71***	4.89***	-0.98***
	(0.09)	(0.09)	(0.10)	(0.11)	(0.14)	(0.14)	(0.15)	(0.15)
N (counties x intervals)	9,351	9,351	9,351	9,351	9,351	9,351	9,351	9,351
R^2	0.52	0.41	0.44	0.53	0.47	0.62	0.31	0.62

Notes. Table presents results from estimating equation 7 using OLS. The sample is all U.S. counties, and the dependent variable is the county's average annual heroin (models 1 to 4) and fentanyl (models 5 to 8) death rate per 100,000. The spillover variables are weighted averages of opioid overdose death rates in the previous time-period in other counties, for different types of opioid deaths. Weighting is based on the relative probability of a cross-county Facebook friendship. Dependent variable observations are pooled from 2010 to 2018 in 3-year intervals, with the three-year interval from 2007–2009 serving as a lagged period for 2010–2012. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denote statistical significance at level $p < 0.01(0.05)0.1$.

APPENDIX A: Additional Details on Theory and Estimation

a. Parameters of demand equation 4

The parameters for the demand equation for addictive substances when consumers are rational and have perfect foresight (equation 4 in the text) are derived in Reif (2019) and are as follows:

- $\alpha_1 = \frac{b_{as} - (1-d)^2 \beta (b_{aS} + b_{SS})}{\Delta}$;
- $\alpha_2 = \frac{(1-d)\beta(b_{aa} + b_{aS})}{\Delta} > 0$;
- $\gamma_1 = \frac{b_g}{\Delta} > 0$;
- $\gamma_2 = -\frac{(1-d)\beta b_g}{\Delta} < 0$;
- $\pi_1 = -\frac{\lambda}{\Delta} < 0$;
- $\pi_2 = \frac{(1-d)\beta\lambda}{\Delta} > 0$;
- $\delta_1 = \frac{b_{ax}}{\Delta}$;
- $\delta_2 = \frac{(1-d)\beta(b_{Sx} - b_{ax})}{\Delta}$;
- $k = \frac{b_a - (1-d)\beta(b_a - b_s)}{\Delta}$; and
- $\Delta = b_{aa} + (1-d)^2\beta(b_{aS} + b_{SS}) > 0.$

b. Simulation Details (Figure 2)

Our simulation uses a closed population of 1,000 individuals with heterogeneous tastes for opioids x_{it}, drawn from a normal distribution with mean -1.25 and standard deviation 1. Parameter values are as follows, with utility maximization parameters following the simulation of Reif (2019):

Parameter	**Meaning**	**Value**
Panel A. Utility Maximization		
β	Discount rate	0.75
d	Depreciation rate (consumption stock)	0.5
λ	Marginal utility (MU) of income	1.0
b_a	Marginal utility of drug use	15
b_S	Marginal utility of consumption stock	10
b_{aS}	Adjacent complementarity (addiction)	0.2
$b_{aa} = b_{SS} = b_{xx}$	Slope of MU of drug use	0.3
b_{ax}	Complementarity between drug and composite use	2.0
b_{Sx}	Complementarity between consumption stock and composite use	4
b_g	Spillover effect	0.079
Panel B. Price Shock		
p_t for $t \in [0,5)$	Initial price	12
p_t for $t \in [5,T]$	Reduction in price (80%) after period 5	9.6
Panel C. Hazard Rate		
ψ	Constant	-10
ψ_a	Increase in hazard with drug use	0.05
ψ_{aa}	Increase in slope of hazard with drug use	0.005
ψ_{aS}	Reduction in mortality of use with higher tolerance	-0.005

The simulation starts from the steady state with initial prices as defined in section II. It is then allowed to adjust to new prices, under a situation with and without spillovers. The simulation including social interactions requires a multistep procedure outlined in Reif (2019). Specifically, we start by generating consumption without social interactions and calculate mean consumption: denote a_0. One important difference from Reif is that we also account for mortality by calculating mean consumption among the living: i.e., averaging consumption times each person's predicted survival rate up to that point in time. We then calculate mean consumption again (incorporating the spillovers) and iterate each of these steps (denote each iteration attempt i) until $|\bar{a}_i - \bar{a}_{i-1}| < 0.01$. This algorithm is repeated in each period, until the conclusion of the simulation.

c. Generalized Spatial Two Stage Least Squares Estimation

Estimation of equation 4 with generalized spatial two stage least squares (GS2SLS) follows the approach developed in (Arraiz et al. 2010; Drukker, Prucha, and Raciborski 2013; Kelejian and Prucha 1998, 1999, 2004, 2010). To implement the estimator, we rewrite equation 7 in compact form:

$$\boldsymbol{Y_t} = \boldsymbol{Z_t}\delta + \boldsymbol{\epsilon_t},$$

With $\boldsymbol{Y_t} =$ an $NT \times 1$ vector of opioid death rates for counties and time periods. $\boldsymbol{Z_t} = [\boldsymbol{WY_t}, \boldsymbol{X}, \boldsymbol{Y_{t-1}}, \boldsymbol{T}]$ (an $NT \times k$ matrix of a weighted averages of deaths in other counties, determinants of use $\boldsymbol{X_t}$, lagged death rates $\boldsymbol{Y_{t-1}}$, and indicators for time $\boldsymbol{T}$); $\delta = [\lambda, \gamma, \rho]'$; and $\boldsymbol{\epsilon_t} =$ a vector of error terms. Define $\boldsymbol{X_t^e} = [\boldsymbol{X}, \boldsymbol{Y_{t-1}}, \boldsymbol{T}]$ as a matrix that excludes the endogenous variable $\boldsymbol{WY_t}$ (weighted averages of deaths in other areas). We then form an instruments matrix $\boldsymbol{H}$ from linearly independent columns of $[\boldsymbol{X_t^e}, \boldsymbol{WX_t^e}, \boldsymbol{W^2X_t^e}]$, where the weighting matrix is interacted with exogenous variables up to the second power. In theory, interacting lower- or higher-powers of the weighting matrix with the exogenous variables may be included as instruments. However, including up to the second order has been shown to perform best in Monte Carlo simulations (see references above). With projection matrix $\boldsymbol{P_H} = \boldsymbol{H}(\boldsymbol{H'H})^{-1}$ and $\widetilde{\boldsymbol{Z}} = \boldsymbol{P_H Z_t}$ and exogeneity of $\boldsymbol{X_t^e}$ (i.e., $E[\boldsymbol{\epsilon_t'}\phi\boldsymbol{\epsilon_t}] = 0$ for some weighting matrix ϕ that satisfies $tr(\phi) = 0$), the estimating equation is analogous to traditional two-stage least squares:

$$\hat{\delta} = \left(\widetilde{\boldsymbol{Z}}'\boldsymbol{Z_t}\right)^{-1}\widetilde{\boldsymbol{Z}}'\boldsymbol{Y_t}.$$

For implementation of the estimator, we use **spregress** in Stata. Readers should see Stata's reference manual for **spregress** for further details.[38]

[38] Available at: https://www.stata.com/manuals/spspregress.pdf.

APPENDIX B. Additional Tables

Table B1: The impact of opioid shipments in neighboring counties on own opioid shipments, 2007 to 2009

	Ordinary least squares			*Spatial two-stage least squares*		
	OxyContin	*Other opioids*	*All opioids*	*OxyContin*	*Other opioids*	*All opioids*
MME per capita$_{t-1}$ (ρ)	1.00***	1.07***	1.07***	1.01***	1.06***	1.07***
	(0.01)	(0.02)	(0.01)	(0.01)	(0.02)	(0.02)
MME spillovers (distance) (λ_d)	0.11***	0.05***	0.04***	0.08***	0.03***	0.04***
	(0.01)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)
<u>Instruments</u>						
Non-triplicate state				1.59**	3.12**	3.80***
				(0.65)	(1.28)	(1.34)
1994–96 cancer mortality rate				-0.05	1.12*	1.21*
				(0.22)	(0.66)	(0.67)
1990 percent disabled				-0.09	1.52	0.88
				(0.15)	(0.94)	(0.83)
<u>Year (rel. 2007)</u>						
Year = 2008	18.81***	-31.26***	-11.60***	19.21***	-30.78***	-11.21***
	(0.70)	(1.61)	(1.61)	(0.70)	(1.70)	(1.66)
Year = 2009	-4.88***	-6.83***	-12.53***	-4.58***	-5.67***	-11.71***
	(0.66)	(1.65)	(1.74)	(0.65)	(1.71)	(1.78)
N (counties x years)	9,351	9,351	9,351	9,351	9,351	9,351
R^2 (1–3)/Pseudo R^2 (4–6)	0.87	0.95	0.96	0.87	0.95	0.96

Notes. Estimates of equation 7. The sample is all U.S. counties, and the dependent variable is the county's average annual opioid death rate per 100,000 people. Data are pooled from 1996 to 2010 in 5-year intervals (1996–2000; 2001–2005; and 2006–2010). Data from 1991–1995 also serve as a lag period for the 1996–2000 period. Models 1 to 3 use OLS. Models 4 to 6 use GS2SLS. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denotes p<0.01(0.05)0.1. See section V and Appendix A for more details.

Table B2: The impact of opioid death rates in peer and neighboring counties on own opioid death rates, 2010 to 2018

	(1)	(2)	(3)
Opioid deaths$_{t-1}$ (ρ)	0.73***	0.49***	0.46***
	(0.02)	(0.02)	(0.02)
Death spillovers (distance) (λ_d)	0.59***		-0.03
	(0.03)		(0.03)
Death spillovers (friends) (λ_f)		0.76***	0.85***
		(0.03)	(0.03)
Instruments			
Non-triplicate state	0.28	0.64***	0.59***
	(0.20)	(0.18)	(0.18)
1994–96 cancer mortality rate	0.12	0.22***	0.25***
	(0.07)	(0.07)	(0.07)
1990 percent disabled	-0.24***	-0.12***	-0.14***
	(0.06)	(0.05)	(0.05)
Year (rel. 2010–12)			
Year = 2013–15	-0.12	-0.02	-0.06***
	(0.20)	(0.18)	(0.05)
Year = 2016–18	0.12	0.45**	0.27
	(0.24)	(0.22)	(0.22)
N (counties x intervals)	9,351	9,351	9,351
Pseudo R^2	0.47	0.51	0.50

Notes. Estimates of equation 7. The sample is all U.S. counties, and the dependent variable is the county's average annual opioid death rate per 100,000 people. Data are pooled from 2010 to 2018 in 3-year intervals (2010–2012; 2013–2015; and 2016–2018). Data from 2007–2009 also serve as a lag period for the 2010–2012 period. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denotes p<0.01(0.05)0.1. See section V and Appendix A for more details.

Table B3: Impact of lagged opioid death rates in neighboring/friend counties on own death rates

	1996 to 2010			*2010 to 2018*		
	(1)	(2)	(3)	(4)	(5)	(6)
Opioid deaths$_{t-1}$ (ρ)	0.89***	0.62***	0.62***	0.72***	0.52***	0.52***
	(0.03)	(0.03)	(0.03)	(0.02)	(0.03)	(0.03)
Death spillovers$_{t-1}$ (distance) (λ_d)	1.07***		0.34***	0.69***		0.23***
	(0.09)		(0.08)	(0.04)		(0.04)
Death spillovers$_{t-1}$ (friends) (λ_f)		1.15***	1.05***		0.80***	0.70***
		(0.07)	(0.07)		(0.04)	(0.04)
<u>Year (rel. 1996–00)</u>						
Year = 2001–05	1.38***	1.49***	1.33***			
	(0.11)	(0.10)	(0.10)			
Year = 2006–10	-0.78**	-0.10	-0.94***			
	(0.32)	(0.20)	(0.31)			
<u>Year (rel. 2010–12)</u>						
Year = 2013–15				-0.12	0.11	-0.09
				(0.21)	(0.19)	(0.20)
Year = 2016–18				1.11***	1.61***	1.17***
				(0.24)	(0.23)	(0.23)
N (counties x intervals)	9,351	9,351	9,351	9,351	9,351	9,351
R^2	0.49	0.53	0.53	0.48	0.51	0.51

Notes. Table presents results from estimating equation 7 using OLS. The sample is all U.S. counties, and the dependent variable is the county's average annual opioid death rate per 100,000. The spillover variables are weighted averages of opioid overdose death rates in the previous time-period in other counties. Weighting is done separately for inverse geographic distance and the relative probability of a cross-county Facebook friendship. Dependent variable observations are pooled from 1996 to 2010 in 5-year intervals (models 1 to 3) and 2010 to 2018 in 3-year intervals (models 4 to 6); each have a lag period of the same interval. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denote statistical significance at level $p < 0.01(0.05)0.1$.

Table B4: The impact of opioid death rates in peer and neighboring counties on own opioid death rates, 1996 to 2010

	(1)	(2)	(3)	(4)	(5)	(6)
Opioid deaths$_{t-1}$ (ρ)	0.60***	0.59***	0.59***	0.60***	0.59***	0.58***
	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
Death spillovers (friends) (λ_f)	0.78***	0.80***	0.76***	0.79***	0.81***	0.78***
	(0.05)	(0.04)	(0.04)	(0.04)	(0.05)	(0.05)
<u>Instruments</u>						
Non-triplicate state	0.47***					0.29***
	(0.03)					(0.09)
1994–96 cancer mortality rate		0.13***				0.04
		(0.03)				(0.04)
1990 percent disabled adults			0.16***			0.18***
			(0.02)			(0.03)
2005–07 percent without a college degree				0.02***		-0.01
				(0.004)		(0.01)
Predicted increase in import competition from China (%)					-0.03**	-0.07***
					(0.02)	(0.02)
<u>Year (rel. 1996–00)</u>						
Year = 2001–05	0.24*	0.19	0.31**	0.21	0.19	0.28**
	(0.13)	(0.13)	(0.13)	(0.13)	(0.14)	(0.14)
Year = 2006–10	-0.65***	-0.72***	-0.49***	-0.69***	-0.77***	-0.67**
	(0.22)	(0.22)	(0.21)	(0.22)	(0.22)	(0.22)
N (counties x intervals)	9,351	9,351	9,351	9,351	9,294	9,294
Pseudo-R^2	0.52	0.52	0.53	0.52	0.52	0.53

Notes. Estimates of equation 7. The sample is all U.S. counties, and the dependent variable is the county's average annual opioid death rate per 100,000 people. Data are pooled from 1996 to 2010 in 5-year intervals (1996–2000; 2001–2005; and 2006–2010). Data from 1991–1995 also serve as a lag period for the 1996–2000 period. All models use GS2SLS, with different instruments (described above). Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denotes p<0.01(0.05)0.1. See section V and Appendix A for more details.

Table B5: The impact of opioid death rates in peer and neighboring counties on own opioid death rates, 2010 to 2018

	(1)	(2)	(3)	(4)
Opioid deaths$_{t-1}$ (ρ)	0.48***	0.47***	0.47***	0.44***
	(0.02)	(0.02)	(0.02)	(0.02)
Death spillovers (friends) (λ_f)	0.69***	0.64***	0.72***	0.67***
	(0.03)	(0.03)	(0.03)	(0.03)
Instruments				
OxyContin doses per capita (2008–09)	0.99***			0.35**
	(0.12)			(0.15)
Other prescription opioid doses per capita (2008–09)		0.44***		0.29***
		(0.05)		(0.06)
Heroin death rate (2005–09)			0.49**	0.48**
			(0.22)	(0.22)
Share white powder heroin (2004–2010)			0.46	0.19
			(0.32)	(0.33)
Heroin death rate × share white powder heroin			1.49***	1.46***
			(0.41)	(0.41)
Year (rel. 2010–12)				
Year = 2013–15	0.11	0.19	0.08	0.20
	(0.18)	(0.18)	(0.18)	(0.18)
Year = 2016–18	0.78***	1.01***	0.68***	1.02***
	(0.22)	(0.23)	(0.23)	(0.23)
N (counties x intervals)	9,351	9,351	9,351	9,351
Pseudo-R^2	0.52	0.52	0.53	0.54

Notes. Estimates of equation 7. The sample is all U.S. counties, and the dependent variable is the county's average annual opioid death rate per 100,000 people. *OxyContin* and other prescription opioid doses are defined as 50 morphine milligram equivalents. Data are pooled from 2010 to 2018 in 3-year intervals (2010–2012; 2013–2015; and 2016–2018). Data from 2007–2009 also serve as a lag period for the 2010–2012 period. Heteroskedasticity robust standard errors are reported in parentheses. ***(**)* denotes p<0.01(0.05)0.1. See section V and Appendix A for more details.