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PRESCRIPTION FOR DISASTER: THE SSDI RATE, PAIN, AND PRESCRIBING PRACTICES

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ABSTRACT

A county's fraction of adults in 1990 on Social Security Disability Insurance (SSDI) is a strong predictor of growth in local drug death rates after 2000. The part of the SSDI rate related to drug deaths is not proxying for well-known contributors to the drug crisis, e.g. OxyContin. Instead, it appears to capture the fraction of people in chronic pain. We show that in the late 1990s, physicians began prescribing opioids more aggressively to treat pain. Taken together, our estimates suggest that drug deaths rates would be 43% lower in 2015 had prescribing practices stayed at 1995 levels.

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An online appendix is available at http://www.nber.org/data-appendix/w34265

I. Introduction

The US is now in its fourth decade of rising drug poisoning death rates and there is no end in sight with drug deaths exceeding 100,000 per year since 2021. The drug death rate in 2022 is 10 times the rate in 1979 and the annual rate of change has accelerated considerably in the 21st century. The solid blue line in Figure 1 is the drug poisoning death rate at the national level from 1979 to 2022.[1] The solid gold line reports predicted values from a regression during the 1979-1999 period that has as controls a quadratic time trend. The pre-2000 quadratic fits the first 20 years very well as the R^2 is 0.96. Predicting this model out until 2022 (the dashed yellow line), death rates would have increased, but they would have been half the value eventually obtained. This suggests something dramatically changed in the mid-to-late 1990s that accelerated the growth in drug death rates. In this paper, we examine a potential reason for this acceleration in deaths that has been suggested in the literature but the contribution to the crisis has not been documented.

The bulk of the academic research on the drug crisis has focused on the trajectory in the 21st century. Scholars have attempted to explain this period with both demand and supply-side hypotheses. The leading demand-side story suggests that a decline in institutions such as unions, manufacturing jobs, marriage, and religion, especially for lower-skilled workers, may have led some people to take comfort in drug use (Case and Deaton, 2015, 2017, and 2020). This hypothesis has intuitive appeal given the high rates of drug deaths in low-educated adults (Case and Deaton, 2022; Powell, 2023) and the concentration of deaths in places like West Virginia, Southern Ohio, and Western Pennsylvania. The hypothesis also has some empirical support with research showing that drug deaths are higher in areas impacted most by trade (Dean and Kimmel, 2019; Pierce and Schott, 2020), with the largest declines in manufacturing jobs (Charles, Hurst, and Schwartz, 2022; Venkataramani et al., 2020), with the greatest growth in the unemployment rate (Carpenter, McClennan, and Rees, 2017), and in areas

[1] The starting year in the series is the first year the ICD-9 classification system is in use and the final year in the series is the last year final data is available at the time we are working on this paper.

with the greatest declines in church participation (Giles, Hungerman, and Oostrom, 2023). Despite this evidence, others point out that these demand-side variables such as changing employment opportunities have a difficult time explaining the magnitude of the change in drug deaths in the 21st century (Hollingsworth, Ruhm, and Simon, 2017; Ruhm, 2019; Currie and Schwandt, 2021). Supply-side hypotheses focus on the introduction and early marketing of a particular prescription opioid, OxyContin, as a major contributing factor (Alpert et al., 2022; Arteaga and Barone, 2023). These efforts can explain the relative growth in drug death rates between states or commuting zones with high and low OxyContin advertising, but they do not directly explain the growth in deaths that is common in both high and low advertising areas.

In this paper, we identify and study a factor common to most geographic areas that helps explain the rapid rise in drug deaths in the 21st century. It is often argued in both the popular press (Quinones, 2015; Keefe, 2021) and academic literature (Jones et al., 2018; Bernard et al., 2018; Cutler and Glaeser, 2021; National Academy of Sciences, 2021; Vadivelu et al., 2018) that the antecedents for the current drug crisis were increased abuse of prescription opioids. This factor should then most likely be related to the use of prescription drugs. To begin to isolate this characteristic, we first conduct a simple empirical exercise where we use detailed data at the county level in 1990 and correlate the major drivers of the drug crisis identified by prior research with the changes in drug death rates between 1995 and 2015. The exercise indicates that areas with many low-educated workers, more veterans, and higher male unemployment rates experience greater changes in drug death rates over time. Interestingly, the county characteristic with the most predictive power for where the crisis will be the worst is the fraction of working age adults on Social Security Disability Insurance (SSDI). SSDI recipients are those adults that have worked for a minimum amount of time in jobs covered by Social Security and have a medical condition that limits their ability to engage in substantial gainful employment.

Cutler and Glaesar (2021) were the first to show that the SSDI rate prior to the rise of drug deaths is an important predictor of which geographic areas will have larger opioid shipments and opioid death rates. In a county-level panel data set from 1997 through 2010, Cutler and Glaeser (2021) use opioid shipments and drug deaths from prescription opioids as their key outcomes. In models with county and year fixed effects, they interact a measure of pain at the county level in the pre-1997 period with a measure of national opioid shipments. They use two measures of pain: the share of the labor force on disability insurance and self-reported joint-pain prevalence as it comes from the Behavioral Risk Factor Surveillance Survey. Their work concludes that "pain is a potent predictor of opioid shipments (p. 190)" and drug poisoning death rates.

We expand on this work along a number of dimensions. We initially analyze the simple correlation described above and confirm that the county-level SSDI rate is a strong predictor of the impending crisis. We document year-by-year, cross-sectional correlations between a county's 1990 SSDI rate and its drug death rate in subsequent years. We find that the fraction of a county's working age adults on SSDI in 1990 was not always related to drug deaths: In 1990, it was actually negative. Over time, the correlation increases modestly until around 1999 when it increases dramatically and by 2010, the raw correlation had become 0.35.

We then estimate event-study models where the outcome is drug death rates and the 1990 SSDI rate has been interacted with year indicators. We set the reference period to be 1995 to facilitate comparisons with the literature that has emphasized the importance of OxyContin's introduction in 1996 to the growth in drug death rates (Alpert et al., 2022; Arteaga and Barone, 2023). There is no clear differential pre-1995 trend while the post-1996 variables become positive, large in value, and statistically significant.

We then explore why the 1990 SSDI rate is so strongly associated with subsequent drug deaths. There are three general categories of interpretations we assess: 1) those related to opioid marketing and regulations that have been shown to be important by previous work, 2) local economic conditions that

in part determine the SSDI rate, and 3) changes in prescribing practices related to pain management. We first show that the 1990 SSDI rate is not simply picking up the impacts of supply-side factors that have been shown to affect opioid prescribing and death rates. Using the variation in triplicate laws leveraged by Alpert et al. (2022), we show that the 1990 SSDI rate has an independent association with drug death rates over and above the impacts of the introduction and marketing of OxyContin. Moreover, we show that counties with higher 1990 SSDI rates saw larger increases in opioids containing oxycodone in subsequent years, but those same counties also saw larger increases in other types of opioids as well. This is in contrast to the literature studying the effects of OxyContin's introduction and marketing which has found that of all the different types of opioids, only those containing OxyContin's active ingredient, oxycodone, increased in places where OxyContin was marketed.

Identifying how the SSDI rate in 1990 impacts the crisis is difficult because it is correlated with so many economic variables. Drug use potentially impacts employment prospects (Harris et al., 2020; Beheshti, 2023; Mukherjee, Sacks, and Yoo, 2023), SSDI benefits alter the incentives for work (Autor and Duggan, 2003; Moore, 2015), economic conditions alter the decision to enroll in SSDI (Coe et al., 2011; Gettens, Lei, and Henry 2023; Michaud, Moore, and Wiczer, 2024), and better employment prospects are associated with lower drug use (Carpenter, McClennan, and Rees, 2017). Given all of these relationships, it could be the case that the association we observe is driven by underlying economic forces. Using multiple measures for these factors individually and together (male unemployment rate, female unemployment rate, blue collar job rate, and the poverty rate), we rerun our analyses and do not find evidence that the relationship between the 1990 SSDI rate and post-1995 drug overdose death rates is driven by local economic or labor market conditions.

Because the 1990 SSDI rate is a measure of disability in an area, it likely is related to the geographic area's underlying health. We draw a distinction between health directly related to pain and health that is not directly related to pain because opioids are meant to treat pain conditions. To measure

non-pain health, we use the infant mortality rate, the number of inpatient (hospital) days per capita, and the outpatient visit rate. Again, when these variables are included in our analyses, the estimated association between the 1990 SSDI rate and drug death rates is essentially unchanged.

Given the lack of evidence that the association between 1990 SSDI rates and subsequent drug death rates is driven by the introduction and marketing of OxyContin, local economic conditions, or local health unrelated to pain, we turn to evaluating the hypothesis that the 1990 SSDI rate is a proxy for pain and that physicians changed their opioid prescribing behaviors in the mid-to-late 1990s. The underlying logic is that as the standards for writing an opioid prescription fall, geographic areas with a higher rate of pain are more at risk of receiving more opioid prescriptions. Because the current drug epidemic started with medicines prescribed by medical providers, these additional opioid prescriptions would eventually lead to more abuse and more drug overdoses.

We find evidence consistent with this hypothesis. In particular, we find that the 1990 SSDI rate is predictive of pain in the local geographic area, not just for those on SSDI, but for those not on SSDI as well. We document a series of events that indicate that social norms and regulatory policies in the medical community related to opioid prescribing were changing in the second half of the 1990s, especially for chronic pain. Using data from the National Ambulatory Medical Care Surveys from 1980 through 2015, we present opioid prescribing patterns for 18 to 64-year-old individuals with medical conditions that are likely to cause either acute or chronic pain. For both types of pain, the chance of receiving an opioid prescription was flat between 1980 and 1995. From 1995 to 2015, it increased by almost 70 percent for acute pain but by roughly 300 percent for chronic pain. Data on whether patients sought out a prescription suggest that these changes were not likely to have been driven by changes in patient demand rather than changes in prescribers' behaviors.

Why is this important? Previous work has established that the prescribing practices of physicians were a key contributor to the crisis prior to the movement to illicit synthetic opioids. Barnett, Olenski, and Jena (2017), Barnett et al. (2019) and Eichmeyer and Zhang (2022, 2023) show

that Medicare patients and veterans being treated by physicians with a higher propensity to prescribe opioids are more likely to suffer a drug poisoning than patients of other physicians. Finkelstein, Gentzkow, and Li (2024) show that moving from a low to high prescribing area greatly increases the chance of opioid abuse for those on federal disability roles. Schnell (2022) shows that the drug poisoning death rate is higher in areas with more opioid prescriptions per capita and grew more in areas with a greater growth in prescriptions. Schnell and Currie (2018) examine how fixed differences in a physician's education relate to their opioid prescribing. While much of this work shows the critical role that static differences in prescribing practices across physicians play, many do not provide evidence that prescribers became more likely to write an opioid prescription holding patients' behaviors and characteristics constant or on the role that changing prescribing practices had in shaping the epidemic, which is what this paper adds to the discussion. In our work, if we interpret the effect captured by the 1990 SSDI rate as the impact due to a change in prescribing practices, a simple back of the envelope calculation suggests that if prescribing practices were held at pre-1996 levels, there would have been approximately 43 percent fewer drug deaths since the late 1990s.

Our paper also contributes to the broader literature on the causes and consequences of the drug crisis. Case and Deaton (2015, 2017, and 2020) have argued that deaths of despair—deaths from drugs, alcohol, and suicides—have increased, especially among those with lower education, because basic institutions such as unions, the manufacturing sector, marriage, and religion have all decayed slowly over the last 40 or so years. Although there is some quasi-experimental evidence consistent with this hypothesis, a number of authors including Hollingsworth, Ruhm, and Simon (2017), Ruhm (2019), and Currie and Schwandt (2021) argue against economic factors as being the major drivers of the drug crisis after 1999. We too find little role for the decline in institutions as an explanation for the surge in drug death rates that began in the mid-to-late 1990s.

II. Contributors to the Drug Crisis

We begin our analysis with a simple exercise and ask whether there are some characteristics of counties that are predictive of a larger increase in drug death rates in the future. We generate three sets of variables that past research suggests played important roles. Our first set of variables are based on Case and Deaton's (2015, 2017, and 2020) hypothesis that declines in institutions such as the unionized sector, mining and manufacturing jobs, the institution of marriage, and the general decline in formal religion may have encouraged drug use. We use the percent of adults aged 20-64 that are married, the percent of workers that are in the manufacturing, mining, or agricultural industries, and the percent of adults that are adherents to a formal religion to represent Case and Deaton's hypothesis.[2] We use the data from the 1990 Census STF files, curated by IPUMS NHGIS (Morden et al, 2024), for the first two variables, while the third variable is obtained from the Association of Statisticians of American Religious Bodies (1992).

In the next block, we define a number of characteristics that can be considered to be demand-based measures for drugs. These include the poverty rate, median family income, the percent of males aged 25-54 not in the labor force, the percent of adults ages 18 and up with a high school degree or less, the male and female unemployment rates, the fraction of adults aged 18-64 that are on Social Security Disability Insurance in 1990 (a variable we call the 1990 SSDI Rate),[3] and finally, the non-drug deaths of despair death rate.[4] The first six are from the IPUMS coding of the Census STF files used above, the SSDI Rate is from Social Security Administration (1990) and Moore (2020), while the final one us is from the restricted-use version of the Multiple Cause of Death files for 1990.

[2] Some variables we present in our rough set of categories could be put into multiple groups. For example, the employment rate has been used to assess the Deaths of Despair hypothesis in the past, but we include it in a different group in our categorization. The groupings themselves are purely for exposition and do not affect our analysis.
[3] The numerator is the number of people on SSDI as of December 1989. For the denominator, we use the 18-64 year old population from the National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) program for 1990.
[4] These include alcohol deaths plus non-drug suicides.

In the third block, we include some basic measures of the size of the medical establishment at the county level and include physicians and hospital beds per 100,00 people, plus inpatient and outpatient visits per 1,000. These are all collected from the Area Resources File for 1990.

These measures will be correlated with county-level drug death rates that are constructed from the restricted-use Multiple Cause of Death mortality files and population data from the National Cancer Institute's Surveillance, Epidemiology, and End Results program. The mortality rates are drug deaths for 18-64 year olds per 100,000 population of that same age group.[5] In Appendix B, we outline how we define drug deaths in these data. We also clarify how we define two other death rates: deaths from alcohol and non-drug suicides. These two death categories are the non-drug components of Case and Deaton's deaths of despair. As the data spans many years, we include counties that can be consistently defined over the entire time period. Appendix C outlines how we aggregate counties across years to generate a balanced panel of counties.

Figure 1 suggests that drug death rates began to grow more quickly in the late 1990s or early 2000s. As such, we construct the change in the county-level drug poisoning death rate at the county level between 1995 and 2015. We choose the former date because it is before the trend break and the existing research suggests much of this increase in the 2000s was due to a series of supply shocks: first, the rise of Oxycontin, then a switch to heroin, and finally, the emergence of fentanyl. As OxyContin was released in 1996, 1995 is a natural starting date. We selected the latter date as this is the early stages before synthetic opioids like fentanyl drastically reshaped the landscape.

In column 1 of Table 1, we report correlation coefficients between descriptive variables measured at the county level in 1990 and the change in the drug poisoning death rate between 1995 and 2015. We calculate the correlation coefficients weighting observations by the average of the county's

[5] The mortality data is a census of deaths in the US and contains an underlying cause of death and any multiple causes of death. Causes of death are defined by the ICD-10 classification system starting in 1999 and the ICD-9 system beforehand. The restricted-use versions provide the county of residence for the deceased.

adult population for 1995 and 2015. Given the variety of data sets and vagaries in how the county FIPS codes are used across sources, there are 3,014 counties in this sample. This is not meant to be causal, but rather descriptive of where someone might look for explanations for the rapid increase in drug deaths in the post-2000 period.

Some of the correlations are expected. The increase in deaths was higher in areas with more veterans and in areas with less education (more adults with a high school degree or less). Surprisingly, the variable with the largest correlation coefficient is the SSDI rate, a coefficient that is 56 percent larger than the next largest correlation, the fraction of adults with a high school degree or less.

Next, we standardize all the 1990 county characteristics to have a mean of 0 and a standard deviation of 1 and regress the change in drug death rates from 1995 to 2015 on the standardized values one by one. These regressions also weight observations by the county average adult population in 1995 and 2015. The coefficients and standard errors are reported in column (2). As each variable is standardized, the coefficients represent the difference in the growth of drug death rates associated with a one standard deviation increase in the county characteristic. The two largest marginal effects are on the 1990 SSDI and the fraction of adults with less than a high school education.

In the final column of the table, we present results from running a single regression that includes all of the characteristics at once (rather than one-by-one as in column 2). Variables such as percent adults married, percent workers in mining and manufacturing, and the median family income, are statistically significant. However, the SSDI rate once again has one of the largest marginal effects. Because it has the largest correlation, is consistently, strongly related to changes in drug death rates, and is less well known than education to be related to drug death rates, we further explore the relationship between the SSDI rate and drug death rates.

As a first step, we study how well the 1990 SSDI rate correlates with drug death rates in different time periods. In Figure 2, we provide a bubble plot of the SSDI rate in 1990 and the drug poisoning death rate in the same year at the county level with the size of the bubbles being proportional

to the population aged 18 and over; the figure also shows the regression line. Interestingly, the correlation is small and negative which suggests that prior to the run-up up in drug deaths, SSDI rates and drug poisoning death rates were not closely related.

Figure 2 stands in stark contrast to the correlation coefficient we report between the SSDI rate in 1990 and the growth in the drug poisoning death rate between 1995 and 2015. To see how this relationship changed over time, in Figure 3, we plot the correlations between 1990 SSDI rates over time with a more general category, the death rate for all non-drug deaths. The correlation coefficients between this more general measure and the 1990 SSDI rate increases slightly from 1990 through 2015. On the other hand, there is a dramatic increase in the correlation between the 1990 SSDI rate and drug poisoning death rates beginning in the late 1990s; the correlation coefficient increases steadily to a value of 0.40 in 2010 before declining somewhat to 0.33 in 2015.

The ability of the SSDI rate to predict the time series of the drug crisis can be seen in Figure 4A where we display the adult drug poisoning death rate from 1983 to 2015 for two groups: counties in the top and bottom quartiles of the 1990 SSDI rate. In the pre-1996 period, we see that drug poisoning death rates are actually higher in the lowest quartile SSDI rate counties and this difference between counties does not widen in the pre-1996 period. However, the numbers change dramatically in the 1996 period and after. Deaths in the lowest quartile of SSDI rates increase smoothly over time, by 140 percent from 1996 to 2015. In the top quartile, the drug death rate increases by 392 percent over the same period.

To more formally assess these correlations over time, we estimate an event-study specification for county c in year t between 1983 and 2015. More specifically, we estimate

$$(1) \qquad y_{ct} = x_{ct}\beta + \sum_{\substack{i=1983 \\ i\neq 1995}}^{2015} \theta_i (SSDI\ Rate_{c1990}) 1(year = i) + \mu_c + \lambda_t + \varepsilon_{ct}$$

where our dependent variable is the drug death rate, $\underline{x_{it}}$ is a basic set of demographics, [6] μ_c and λ_t are county and year fixed effects respectively, ε_{it} is a random error, and we omit 1995 as a reference year. The key covariates are a series of interactions between the SSDI rate and the year effects. The SSDI rate is per 100 adults. The interquartile range of this value across counties goes from 1.6 to 2.8 with a median of 2.1 and a simple mean across counties of 2.3. We cluster the standard errors by state and weight observations by the adult population in the county and year. Our sample includes a balanced panel of counties. There is data for 3,106 counties for 33 years for a total of 102,498 observations.

Figure 4B plots the estimates for the θ parameters in model (1) and the 95 percent confidence intervals. The figure shows that the pre-1995 coefficients are small and not statistically significant—there is very little to suggest that there were differential pre-trends based on the 1990 county SSDI rate. After 1995, there is a stark increase in the coefficient through 2010 when the coefficient declines slightly and then begins to increase again. The decline in 2010 aligns with the reformulation of OxyContin (Alpert, Powell, and Pacula, 2018; Evans, Lieber, and Powers, 2019) and the rise aligns well with the increase in fentanyl deaths that start to spike up around 2014 (Rudd et al., 2016; Cutler and Glaeser, 2021). The interaction coefficients are all individually statistically significant beginning in 1999 at the ten percent level and at the five percent level thereafter.

In contrast, Appendix Figures A2 and A3 show the same set of results using deaths associated with alcohol and non-drug suicides, respectively, the other components of Case and Deaton's deaths of despair.[7] The figures show that there is no time series pattern in the post 1995 period associated with 1990 county-level SSDI rates. Note also that although the death rates for these two categories are on average larger than the drug poisoning death rate in the pre-1996 period, the coefficients are

[6] The demographic controls in the model are the share of the population ages 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 88+ (with ages 18-24 being the reference group), the fraction black, the fraction other Race (with the fraction white being the reference group), the fraction female, and the fraction Hispanic.

[7] We report the same results shown in Figures 4A and 4B as Appendix Figures A1A and A1B to facilitate comparison.

substantially smaller than what appears in Figure 4A. The results in Figures A2 and A2 strongly suggest that the 1990 SSDI rate in not just capturing something common to deaths of despair.

To provide some perspective on the magnitude of these results in Figure 4A, between 1996 and 2015, the drug death rate increased by 9.7 in the lowest quartile SSDI rate counties and 20.0 in the highest quartile SSDI rate counties, for a raw difference-in-difference of 10.3. The coefficient on the interaction of the 2015 dummy and the 1990 county SSDI rate is 5.5. The population weighted average of the SSDI rates in the top and bottom quartiles are 3.5 and 1.2, respectively. Moving from the bottom to the top quartile changes the SSDI rate by 2.3 points. Multiplying this by 5.5 is 12.7, which is 123% of the total increase of 10.3. It is not surprising that this number is well above 100%. Looking at the time series plots in Figure 4A, if the highest quartile counties had stayed on their pre-1996 trajectory until 2015, they would have had substantially lower drug death rates than the lowest quartile counties.

Another way to gauge the magnitudes of the estimates is to ask how many fewer drug poisoning deaths would have occurred if we removed the post-1995 impacts of the 1990 SSDI rate on drug death rates. In that case, the estimates suggest that there would have been approximately 43 percent fewer drug poisoning deaths between 1996 and 2015.[8] Taken together, the analyses suggest that the 1990 SSDI rate is very closely related to a substantial portion of the subsequent drug death rates. We now turn to exploring why the relationship is so strong.

III. Why is the 1990 SSDI Rate Related to Subsequent Drug Death Rates?

In this section, we consider a number of factors that could be driving the observed relationship between 1990 SSDI rates and drug death rates. We begin with factors from the literature on the opioid crisis and then provide evidence on factors that are known to affect SSDI rates.

[8] To create this estimate, we use the model to predict the number of drug overdose deaths in each county after we have set the post-1995 event-study coefficients to zero. We then report one minus the ratio of those predicted deaths to the actual deaths that occurred.

A. Factors Related to Drug Death Rates

It could be the case that the rise in the drug death rates across counties is due to the spectacular success of OxyContin, an extended-release form of oxycodone manufactured by Purdue Pharma and released in 1996. OxyContin was heavily advertised, and sales increased quickly. It soon became the drug of choice for many individuals that were using pain medicine for recreational purposes. The drug could be crushed and snorted or injected and individuals could access the entire milligram content at once. Alpert et al. (2022) and Arteaga and Barone (2023) provide evidence that Purdue Pharma's advertising strategies lead to geographic disparities in the much of the early growth in the opioid epidemic.

Alpert et al. (2022) argues that Purdue Pharma advertised more in states without triplicate prescription pads (all states except California, Idaho, Illinois, New York, and Texas) and that this made the drug epidemic was much worse in non-triplicate states than it was in the five triplicate states. Purdue's internal documents indicate that in pre-release focus groups, physicians in triplicate prescription states felt that they would use OxyContin sparingly.[9] The focus groups suggested that physicians did not like to be monitored and would avoid the hassle of the triplicate pads by using pain killers that were not subject to the regulation. The authors provide empirical evidence that the release of OxyContin in 1996 and its aggressive advertising produced substantially more opioid use—oxycodone specifically—and mortality in non-triplicate states than in triplicate states.

One element that made Alpert et al. (2022) and Arteaga and Barone (2023) persuasive was that there were stark differences in shipments for oxycodone based on triplicate status and pre-1996 cancer death rates, but there were almost no differences across these same dimensions for other opioids.

[9] Triplicate pads are an early form of prescription drug monitoring where physicians writing certain schedule II drugs used pressure-sensitive forms that produced three copies. The physician kept one copy, gave two to the patient who took them to the pharmacy. The pharmacy kept one for its records and sent the final copy to the state.

These findings were used to argue that the differences reflected the impacts of OxyContin's advertising rather than some other factor that affected all opioids such as increases in demand for painkillers or broader changes in prescribing practices. If the additional growth we are seeing in opioids in top quartile 1990 SSDI rate counties were being driven by opioid marketing, we would expect to see this growth exclusively in oxycodone.

To examine this in more detail, we use data from the Drug Enforcement Administration's (DEA) Automation of Reports and Consolidated Orders System (ARCOS). Within this system, drug manufacturers and distributors must report to the Drug Enforcement Administration all controlled substance transactions from manufacturers to points of sale or distribution.[10] Many of the drugs tracked in the ARCOS system are opioids and we use data for nine opioids.[11] The data measures grams of shipments by quarter to a three-digit zip code. Because different opioids have different strengths, we convert these data to morphine equivalent grams (MEG) at the county level and scale per 100,000 people.[12]

In Figure 5, we show that the 1990 county SSDI rate predicts the growth in the shipments of opioids at the county level. In this figure, we graph the shipments in MEG/100,000 for the counties in the top and bottom quartile of the 1990 SSDI rate. In the first quarter of 1997, the top quartile counties had 19 percent higher shipments per capita, but by the end of the series, this number jumps to a 74 percent difference.

In contrast to results in Alpert et al. (2022), the 1990 SSDI rate predicts an increase in all opioids, not just oxycodone. In Figure 6, we report the difference in the MEGs/100,000 shipped between the top and bottom quartiles for oxycodone (the grey line) and all other opioids (the black

[10] More information about the ARCOS data can be found at https://www.deadiversion.usdoj.gov/arcos/retail_drug_summary/2015/index.html

[11] These are codeine, fentanyl, hydrocodone, hydromorphone, methadone, meperidine, morphine, oxycodone, and oxymorphone.

[12] Details of this process, as well as additional information about the ARCOS data, can be found in Appendix D.

line). The difference for oxycodone rises over time, but so does the difference for non-oxycodone opioids. This is initial evidence that the 1990 SSDI rate is capturing something different from the introduction and marketing of OxyContin since OxyContin only appeared to affect opioids that contained oxycodone.

Alpert et al. (2022) trace out the time series of drug poisoning death rates in triplicate and non-triplicate states and demonstrate that death rates in the two sets of states were trending similarly pre-1996, but deaths in non-triplicate states increased much more dramatically in non-triplicate states after 1996. To confirm the results from Alpert et al. (2022) in our sample, we estimate our baseline event-study regression with three different specifications: the original model as reported in Figure 4B, a model with non-triplicate state dummy variables interacted with year dummies but with no SSDI/year interactions, then a model with both sets of variables. In Figure 7, we report the analogous event-study figures. In Figure 7A, the black lines are the baseline coefficients for the SSDI rate x year dummies alone while the grey lines are the results when non-triplicate x year dummies are added to the model. Controlling for non-triplicate effects does little to the series of estimates. On average, the coefficients on the SSDI rate interactions in the models that have non-triplicate effects are only 8.7 percent smaller in the 2000-2015 period. In Figure 7B, we repeat the exercise for the non-triplicate state x year interactions by themselves (black lines) and after controlling for the SSDI rate x year effects (grey lines). The results in Figure 7B are comparable to the results in Alpert et al. (2022) but the coefficients on the interactions between non-triplicate and time are about three-quarters of their original size after controlling for the SSDI rate times year interactions. These results suggest that both marketing and the variation captured by the 1990 SSDI rate played major roles in the rise of drug poisoning death rates.[13]

[13] Arteaga and Barone (2025) argue that opioid marketing was also tied to a local area's cancer death rate in the mid-1990s. To further test whether our results are driven by opioid marketing, we interact each county's 1995 cancer death rate with year indicators and include the resulting variables into our event-study specification. As seen in Appendix Figure A4, the estimated effects of the 1990 SSDI rate are virtually unchanged by including this alternative measure of opioid marketing.

Another argument could be that opioid manufacturers are concentrating their efforts in counties with high rates of non-malignant pain and hence, the increase in prescribing is a response to the advertising. Although there are only some limited, aggregate data on opioid advertising in the 1990s and 2000s, there are detailed microdata on opioid advertising in recent years. The Open Payments data set[14] includes all payments or transfers of value made by drug and medical device companies to certain healthcare providers since August, 2013. There are reasons to believe that current opioid advertising is positively correlated with past opioid advertising and so indicative of which counties were more likely to have received more advertising early on in the epidemic. First, Alpert et al. (2022) found that non-triplicate states had higher advertising activity than triplicate states in the Open Payments data, approximately two decades after OxyContin's maker indicated that it would advertise more heavily in non-triplicate states. Second, it is common practice in pharmaceutical marketing to send pharmaceutical sales representatives to prescribers that are writing many prescriptions for the product. This tends to reinforce existing prescribing patterns which in turn leads to further visits from the sales representatives.

Given this evidence of persistence, we identify counties with high and low advertising in the Open Payments data. Specifically, we keep any marketing effort ("payment" hereafter) in Open Payments from 2013-2016 for a prescription opioid and calculate the payments per 1,000 people in 2015, as well as the dollar value of payments per 1,000 people, both at the county level. Interestingly, there are 664 counties that had no payments to providers for opioids from 2013-2016, which is roughly 20 percent of counties. In Appendix Figure A5A, we graph the drug death rate for three sets of counties – those in the bottom decile of payments per 1,000 (which are counties with no advertising visits), those in the middle 60%, and those in the top 20 percent. In all three county groups, death rates follow a similar pattern where rates are increasing slowly between 1983 and the later 1990s then

[14] https://openpaymentsdata.cms.gov/

increase dramatically afterwards. In Appendix Figure A5B we report event-study results where we allow the coefficients on the year times 1990 SSDI rate coefficients to vary based on what advertising group the county is in. Although we do not observe opioid advertising directly in the 1990s and 2000s, the patterns across counties that received such different advertising in recent years, suggests that SSDI rates in 1990 are not simply a proxy for how much advertising a county would receive in the 1990s and 2000s. Another way to interpret this is that there is something happening in these low-advertising counties other than marketing to drive drug death rates.

As seen in Table 1, educational attainment is highly predictive of growth in drug death rates. To assess whether the relationship between the SSDI rate and drug deaths is being driven by education, we interact the fraction of the adult population in the county with a high school degree or less in 1990 with year indicators and include them in the event-study regression. Figure 8 displays the coefficients on the SSDI rate interactions both with and without the education controls included in the specification. There is very little difference between the two sets of results. This suggests that while education is highly related to the rise in drug mortality, it is not responsible for the relationship between the 1990 SSDI rate and drug mortality. In Appendix E, we examine the sensitivity of the result to a number of different specifications. We drop the event-study specification in lieu of a difference-in-difference model where the key covariates are interactions with the 1990 SSDI rate and indicator variables for groups of years: one for 1996-2000, 2001-2005, 2006-2010, 2011-2015. The basic results in this specification tell a similar story to the event-study model but allow us to more succinctly summarize results as we alter specifications. In Appendix Table E1, we use the difference-in-differences specification to compare what happens when we add the non-triplicate times year group interactions to the model. Given the results in Figures 7A and 7B, it is no surprise that these estimates show the basic SSDI rate results are not sensitive to this change. In Appendix Table E2, we report results from a series of alterations to the model. The results are not sensitive to whether we include demographic

controls, controls for PDMP laws, linear county time trends, state x year effects, or basic economic variables such as the employment rate or the Pierce and Schott (2020) China trade shock controls.

B. Factors Related to SSDI Rates

Previous research has shown that the SSDI rate at the local level is driven in part by economic factors such as employment opportunities (Coe et al., 2011; Gettens, Lei, and Henry 2023; Michaud, Moore, and Wiczer, 2024). Likewise, authors have linked higher drug use to higher unemployment rates (Carpenter, McClennan, and Rees, 2017), exposure to trade (Dean and Kimmel, 2019; Pierce and Schott, 2020), and the declining manufacturing sector (Charles, Hurst, and Schwartz, 2022; Venkataramani et al., 2020).

To examine whether the SSDI rate is capturing these other factors, we augment our event-study models with a series of county-level measures that capture these other characteristics. We interact these measures with year effects just like we did for the SSDI rate. By doing so, we allow these other factors to enter the model on an equal footing with the SSDI rate.

Using the same county-level data from the 1990 census described previously, we have five measures of economic activity: the poverty rate, the unemployment rate for males, the unemployment rate for females, and the percentage of workers that are in "blue collar" jobs which are mining, manufacturing, and agriculture, plus the percentage of males aged 24-54 that are not in the labor force. We also construct three health-related measures: the infant mortality rate, the number of short-term general hospital inpatient stays in 1990, and outpatient visits in 1990. The first group are all taken from the IPUMS data while the health measures are taken from the Area Resources File. The poverty rate, hospital use, and outpatient variables are calculated to be per 100 population, the employment rates are per 100 people in the labor force, and the infant mortality rate is infant deaths per 100 live births.

The results from including all of the measures at once are graphed in Figure 9. In panel 9A, the estimates in black are the coefficients for the SSDI rate interactions from the baseline model in Figure

4B. The numbers in grey are from the model where all seven other 1990 county measures are entered into the event study specification. There is very little difference in the point estimates between these two models. This suggests that these other 1990 characteristics are not driving our results. In panels 9B and 9C, we report the event study results for the economic variables and in 9D, we report the results from the three health variables. The last three panels do not indicate that the other measures have large impacts on the drug overdose death rate. This suggests that any aspects of the local labor markets, economic conditions, and health related to these measures are not differentially affecting drug overdose death rates. It also suggests that these other factors that are important to determining the SSDI rate itself are not driving the association between the SSDI rate and drug overdose death rates in the post 1995 period.

There are two potential concerns with these results. First, we might worry that the various measures of the local labor market are highly correlated and as a consequence, each individual measure appears to be uncorrelated with drug death rates even though the labor market itself is an important driver. To address this possibility, we ran event study models in which only one of the additional measures is included at a time. The results from those regressions are shown in Appendix Figure A6. In each graph, we report the event study coefficients for our main variable of interest, the year interactions with the 1990 SSDI rate, and the same interactions for the other 1990 county characteristic we included in the model.[15] We find very similar results to the picture painted in Figure 9, suggesting that this potential collinearity issue is not a serious problem in our case. Second, the units of these other variables might not be immediately comparable. While all of the variables have been converted to comparable rates, the variance across variables is different and so a one unit increase in each measure might not be the best way to compare each variables' importance to the outcome. To address this

[15] Because many people on SSDI are not 25-54 year old males, we have checked whether an alternative measure of labor force participation—the overall labor force participation rate—alter our results. As with the measure presented in the appendix, this alternative measure has very little impact on our main results.

issue, we used our back of the envelope calculation to estimate how many fewer drug overdose deaths there would have been had there been no post-1995 impacts of the economic, employment, and health variables. Our estimates suggest that there would have been approximately 48 percent fewer drug poisoning deaths since 1995 if all 1990 SSDI rates were zero (similar to the 43 percent we estimated previously). The results for other variables are much smaller: male unemployment (six percent fewer deaths); female unemployment (one percent fewer); blue collar jobs (five percent fewer); poverty rate (21 percent *more* deaths); infant mortality rate (six percent more deaths); inpatient days (one percent fewer deaths); and outpatient visits (less than one percent fewer).

IV. The Changing Nature of Pain Management and the 1990 SSDI Rate as Proxy for Pain

At this point, our results suggest that the relationship between the 1990 SSDI rate and drug death rates is not primarily driven by local economic conditions, general access to health services, the decline of institutions, or the rise and marketing of OxyContin. The question is then, what is it capturing? We know that the rise in drug death rates is driven by the rise in opioid death rates and that opioids are used to treat chronic pain. In addition, as seen in Figure 5, higher 1990 SSDI rate counties saw much larger increases in prescription opioid use than lower 1990 SSDI rate counties. Perhaps then, the part of the 1990 SSDI rate related to drug death rates is capturing something about the amount of pain in the county. When coupled with the change in opioid prescribing practices that occurred in the 1990s and beyond, this could explain why the SSDI rate in 1990 is so closely related to the dramatic rise in drug death rates from the 1990s to the 2010s. In the rest of this section, we present evidence for this explanation.

In section A below, we provide evidence that the SSDI rate is a proxy for pain not only for those on SSDI itself, but for others in the same geographic area as well. In section B, we note what other authors have pointed out, that starting in the mid-1990s, there was a re-evaluation of the use of opioids to treat chronic pain and as a result, physicians became more aggressive in treating chronic pain

with opioids. In section C we document this rise using data from the National Ambulatory Medical Care Survey from the late 1970s through 2015.

A. The Social Security Disability Rate as a Proxy for Aggregate Pain

Previous work has noted the large geographic variation in the SSDI rate across states (Coe et al., 2011), counties (Michaud et al., 2024), and at the subcounty level such as public use microdata areas (Gettens, Lee, and Henry, 2023). This research has attempted to explain the factors driving this dispersion. Although some of this heterogeneity is driven by variation in program administration, research suggests that another important determinant is the underlying health of the community. Michaud et al. (2024) found that 45 percent of the cross-county difference in the SSDI rate is explained by local health characteristics while Gettens, Lee, and Henry et al. (2023) conclude that two-thirds of the variation in the SSDI rate can be explained by differences in disability prevalence.

Given this existing literature, it is no surprise that the SSDI rate is correlated with contemporaneous measures of health. For example, the correlation coefficient between the all-cause mortality rate in 1990 and the SSDI rate in that same year is 0.72. In fact, of the county characteristics listed in Table 1, the SSDI rate has the largest (absolute value) correlation coefficient with all-cause mortality with the second largest coefficient being the fraction of adults with education less than or equal to a high school degree at 0.66.

The SSDI rate is closely related to the social determinants of health. The correlation of the SSDI rate with the poverty rate is 0.48, with median family income is 0.65, and with the fraction with a high school degree or lower is 0.68. The Behavioral Risk Factor Surveillance Survey (BRFSS) is an annual survey of the adult population and it identifies the county of residence for more populated counties. We pooled the 1990 through 1993 BRFSS surveys, calculated the fraction that smoked, fraction obese, and the fraction that had ever been told they had high blood pressure. Merging this with our data, we have aggregate measures of these risk factors for 531 counties representing about 65

percent of the adult population in the US. The correlation coefficient between the SSDI rate and these risk factors are 0.38, 0.37, and 0.38, respectively. Previous work has demonstrated a pronounced gradient between smoking, obesity, and high blood pressure rates with education. As a comparison, the correlation coefficient between the fraction with a high school degree or less and these same three outcomes and 0.27, 0.44, and 0.31. The SSDI rate is likely describing something much more general about the county than just the small fraction of people on this program.

While the SSDI rate is a general measure of a place's health, our previous analyses suggest that the part of the 1990 SSDI rate that is related to drug death rates is not necessarily about health in general (see Figure 9C). However, it could be capturing something about pain.

To document this possibility, we use a series of questions about pain from the National Health Interview Survey (NHIS). The NHIS collects a short list of variables for everyone in the household but asks a more detailed set of questions for a "sampled adult" in certain households. In the 1997-2001 period, the sampled adults were first asked whether they had pain, aching, stiffness, or swelling in and around a joint in the past 12 months. For those that answered yes, respondents were then asked where these symptoms present on most days for at least one month. In these surveys, we define someone being in chronic pain that answered yes to this second question. In the 2011-2015 NHIS, some sampled adults answered the Adult Functioning and Disability Supplement and one question on that survey asked whether over the past three months, respondents experienced pain on no days, some days, most days, or all days. We define someone as being in chronic pain if they responded they were in pain most or all days.

We separately pool the 1997-2001 and the 2011-2015 NHIS surveys into two analysis files.[16] In Table 2, we provide some descriptive information about these pooled surveys. Among adults 18-64,

[16] In the analysis that follows, we use the ipums.org versions of the NHIS (Blewett, et al., 2022).

the SSDI rate increased by 65 percent between the two periods (row 2) and fraction of adults aged 18 and up in chronic pain in these two surveys was roughly 19-20 percent (row 3).

It is no surprise that SSDI receipt is an excellent predictor of pain at the individual level. Looking only at people 18-64 years old, in row (4) we report the fraction in chronic pain among SSDI recipients and those not on SSDI in row (5). The difference in these means increases from 33.9 percentage point in the earlier sample to 41.7 percentage points in the later period. If we regression-adjust these differences to control for a cubic in age, sex, and race/ethnicity, the numbers fall slightly to 27.2 and 37.0 percentage points, still very large differences.

Interestingly, the SSDI rate predicts pain at the local level not just for those on SSDI, but for those not on SSDI as well. The earlier period sampled individuals in 678 primary sampling units (PSUs) while that number falls to 600 in the later period (row (8)). Aggregating the individual-level data to the PSU level, the correlation coefficient between the SSDI rate and the fraction of people in chronic pain increases from 0.37 in the early period to 0.53 in the later period. If we exclude people who are on SSDI and run the same analysis, we find that the corresponding correlation coefficients are 0.32 and 0.46. Although these correlations are slightly smaller when those on SSDI are excluded, they remain surprisingly large and positive. To give some perspective on how large these correlations are at the local level, the correlations coefficients between chronic pain and the percent with less than or equal to a high school degree, the poverty rate, and fraction of males in the labor force are 0.15, -0.03, and 0.14, respectively. Much like the literature above which showed that a large fraction of the between-county variation in the SSDI rate was due to health, these results suggest that the SSDI rate at the local level is an excellent predictor of pain, even for people not enrolled in SSDI.

B. The Changing Nature of Prescribing Practices and a Timeline of Events

Historically, opioids were reserved for those with acute pain such as post-surgical and cancer patients. Pain from chronic conditions often went untreated, which was viewed by many as a failure of

the medical profession. The number of people experiencing chronic pain is large. In the 2002 National Health Interview Survey, 18 percent of adults report having reoccurring pain sometime in the past year.[17] Despite this, many were either not treated with prescription pain relievers or were under-medicated. In a heavily cited paper, Marks and Sachar (1973) sounded an alarm that among inpatients in their small sample, 73 percent were in moderate or severe distress from pain at some point during their hospital stay. They concluded that patients were being systematically undermedicated with opioid analgesics with survey data suggesting a leading cause for undermedication being a physician's "excessive and unrealistic concern about the dangers of addiction (p.180)." Max (1990) revisited this issue and decried the lack of change in pain management in the 16 years after the Marks and Sachar study.

In addition to fearing that patients would become addicted to opioids, physicians feared potential legal liability for prescribing opioids to patients with chronic pain. A survey of Wisconsin doctors found that one half would routinely reduce dosage or prescribe a lower scheduled drug because of fear of regulatory scrutiny (Weissman et al., 1992). A survey of American Pain Society (APS) members found 40 percent reported that regulatory concerns led them to avoid prescribing opioids for non-cancer patients (Turk and Brody, 1992). Tucker (1998) reports the results of a survey of California physicians which found that 69 percent stated that the potential for disciplinary action made them more conservative in their use of opioids in pain management. Assessing the state of pain management in the mid-1990s, Portenoy (1996) notes that, "The available data suggest that medical decision-making regarding the use of opioids continues to be unduly influenced by regulatory policy" (p. 204). To some degree, the fears of physicians were justified. A survey of state medical board members found that 77 percent would discourage the practice of prescribing opioids for non-cancer pain or investigate it as a violation of law (Joranson et al., 1992).

[17] Authors' calculations using data from Blewett et al. (2022).

Things began to change in the mid-1990s. The Federation of State Medical Boards held a series of 11 workshops between 1994 and 1998 with the goal of educating medical boards about the proper use of opioid analgesics. In 1997, the Federation of State Medical Boards convened a task force of pain doctors, policy, and regulatory experts to produce model guidelines for the use of controlled substances to treat pain. The guidelines contain language that recognizes the need to use controlled substances for pain, encourages physicians to provide adequate pain management for all patients, but most importantly, recognizes and addresses fear of regulatory scrutiny. The guidelines were adopted by the Federation and endorsed by the APS and the American Academy of Pain Medicine (Gilson and Joranson, 2001).

In 1995, the APS issued quality improvement guidelines designed to encourage the effective treatment of pain (Max et al., 1995). Some recommendations include a more systematic measurement of patients' pain, better education about the role of analgesics in the treatment of pain, and collecting information from patients about the success of these efforts to control pain. Shortly after, in his 1995 presidential address to the APS, James Campbell introduced the notion that pain is the "5th vital sign." Campbell (1996) argued that "Quality care means pain is measured. Quality of care means pain is treated." In that same year, the APS and the American Academy of Pain (AAP) released a consensus statement outlining the need for greater opioid use, especially for chronic pain (Consensus Statement, 1997).

The efforts to be more aggressive in treating chronic pain gained traction in 1998 when the Veteran's Health Administration (VHA) announced plans for a National Pain Management Strategy (Kerns et al., 2011). The goal was to develop a comprehensive approach to pain management that reduced pain and suffering for both acute and chronic pain. It was estimated at the time that half of patients in the VHA were in pain (Kerns et al., 2003). The cornerstone of the plan was the introduction of pain as the fifth vital sign and in 2000, the VHA published a toolkit to promote the practice (Department of Veterans Affairs, 2000).

In October of 2000, Congress passed, and President Clinton signed into law, HR 3244, the "Victims of Trafficking and Violence Protection Act of 2000." Section 1603 of the bill provided for the establishment of the "Decade of pain control and research."[18]

In 2001, the Joint Commission on Accreditation of Healthcare Organization introduced standards for pain assessment and management in a variety of patient settings (Berry and Dahl, 2000). The standards focused on the patient's rights to appropriate pain care and the standards encouraged hospitals to make pain evaluation a priority and introduce pain scales. The Joint Committee statement also urged that patients should be taught that pain management is a part of treatment and that the quality of care should be measured in part by how well organizations treat pain. The Centers for Medicare and Medicaid have been fielding the 32-question Hospital Consumer Assessment of Healthcare Providers and Systems among Medicare patients since 2006. Three questions on the survey ask if the patient's pain was adequately controlled during their hospital stay.

C. Documenting the Change in Prescribing Practices of Doctors

The text in the previous section notes that the concerns about untreated pain were present for quite some time, but substantial changes in the medical profession did not start to occur until the mid-1990s. A series of events over the next six years greatly increased the ability for physicians to prescribe opioid analgesics for non-malignant pain. In this section, we use data from the National Ambulatory Medical Care Survey (NAMCS) to document how the prescribing practices of physicians have changed over time with an eye towards whether there were material changes in the post-1995 period. NAMCS is an annual survey of office visits to non-federally employed physicians. NAMCS surveys about 30 patients from a one-week period for each physician. The survey has been fielded in 1973, 1975-1981, 1985, then annually from 1989. We use data from 1980 through 2015, the years in which the survey identifies which prescriptions the patient received. The survey records basic demographics about the

[18] https://www.congress.gov/bill/106th-congress/house-bill/3244/text

patient, up to three ICD-9 diagnosis codes supplied by the physician describing the patient's conditions, payer, any procedures conducted during the visit, plus up to eight prescriptions the patient received during the visit. The files in our sample vary in size from 71,594 visits to 20,922.

Using the coding scheme from Sherry, Sabety, and Maestas (2018), we flag conditions that are likely to produce chronic pain (e.g., neck or back pain, joint pain, migraines) or acute pain (e.g., broken bones, contusions, cuts). This does not mean that the person is currently experiencing pain, just that they have a condition likely to generate pain. We then identify whether the person received a prescription for an opioid using the Multum Classification of Therapeutic Classes.[19,20]

In Figure 10, the grey line represents the fraction of visits that were likely to have chronic pain (panel A) or acute pain (panel B) for those who are between 18 and 64 years of age. We also plot in black, the fraction of visits that resulted in an opioid prescription, conditional on the visit including conditions that indicate the type of pain specified in the panel. We focus on ages 18-64 as the mortality data suggests these are the ages most likely to engage in opioid abuse.[21] We exclude anyone with cancer and anyone with co-occurring chronic and acute pain. The latter restriction only applies to 2.6 percent of the data.

As seen in Figure 10A, the fraction receiving an opioid prescription with a likely chronic pain condition was relatively flat from 1980 (7.0 percent) through 1995 (7.6 percent).[22] After that, the fraction increases dramatically, peaking at 25 percent in 2014. The fraction with a condition likely to produce chronic pain is also increasing over this period, rising from 13.1 percent in 1980 to 23.8

[19] The Multum code was not originally included in the surveys through 2005 but the producers of the original data have added a cross walk for these earlier years that matches the Multum code to prescriptions. We select as opioids narcotic analgesics and narcotic analgesic combinations.
[20] The number of prescriptions reported in the NAMCS was either five (1985-1994), 6 (1985-2002), or eight (1980-81, 2003-2015), depending on the year. To match data over time, we use the first five or six prescriptions reported. The results from NAMCS look very similar if we restrict the sample to the first five prescriptions in all years or take any prescriptions in all years. This is reported in Appendix Figures A7A and A7B.
[21] Over the 2000-2015 time period, the death rate from drug poisonings for people aged 18-64 was 17.2. For those aged 65 and older, this number was only 4.1.
[22] A regression of the rate on a time trend produces a coefficient (standard error) of -0.00048 (0.00051).

percent in 2015, an 82 percent increase. The results for acute pain (Figure 10B) show a similar trend in prescribing behavior in that the fraction of patients with an acute pain condition that receive an opioid prescription is flat between 1980 (9.5 percent) and 1995 (10 percent), but then increases by 66 percent by 2015. In contrast to the trends for chronic pain, acute pain condition rates bounce around from 14 to 18 percent but show no obvious trend.

Although the percentage of patients with acute pain conditions is stable throughout our observed time period, the rising percentage of patients with chronic pain could reflect compositional changes in the population such as aging rather than a change in the way providers are treating pain. To address this possibility, we estimate simple linear probability models of the outcomes on year effects and a complete set of age dummies to flexibly control for age. We also estimate the same models without the age dummies and compare the two. The year effects in the two models and the 95 percent confidence intervals are plotted in Appendix Figure A8 with 1980 serving as the omitted year. The black lines are the raw year differences and the grey lines are the age-adjusted differences. The results show that the aging of the population can account for about 20 percent of the increase in chronic pain rates (panel A). However, an opioid prescription conditional on chronic pain (panel B), acute pain (panel C), and an opioid prescription conditional on acute pain (panel D) are not affected by the inclusion of the age dummies. This suggests that the trends we are seeing are not simply capturing the aging of the population.

While the age composition of patients does not appear to be driving the observed changes in prescribing, changes in patients' opioid seeking behavior could have also played a role in the observed increases in pain diagnoses and subsequent opioid prescribing. There are two types of patient behavioral changes we consider: 1) those that are a reaction to prescribers' behaviors and 2) those that are not a reaction to changes in prescribers' behaviors. As an example of the first type of change, a patient with pain might go to the doctor and ask for an opioid prescription because the patient has heard that the doctor is prescribing a lot of opioids. This is distinct from the same patient going to the

doctor in the absence of having heard that doctors are prescribing more opioids. The former could be considered a part of the equilibrium impacts of changing prescribing practices and so consistent with the general hypothesis in this section of the paper.

The latter possibility, that patients changed their opioid seeking behavior in ways that were not reactions to changes in opioid prescribing practices, poses a potential problem for interpreting our results to mean that prescribers' behavior changed. While this is a possible interpretation of the results presented thus far, it is not particularly likely for a few reasons. First, if the increase in prescribing were exclusively patient driven, we might have expected to see similar increases in the use of opioids for both acute and chronic pain: in the absence of type-of-pain specific demand shocks, more demand for opioids by patients means more opioids for all types of pain. Between 1995 and 2005, prescribing conditional on the relevant pain conditions rose by 75 percent for chronic pain, but by only 8 percent for acute pain; between 1995 and the end of our sample in 2015, the corresponding increases are 206 percent for chronic pain and 68 percent for acute pain. Although prescribing has increased for both types of pain, it has done so at very different rates. Second, it is not clear why there would have been large changes in consumer attitudes towards pain specifically in the middle-to-late 1990s. In fact, at least one pharmaceutical firm that played a central role in the opioid epidemic, Purdue Pharma (Alpert et al., 2022), created promotional materials for doctors' offices to ease patients' concerns about the dangers of opioid addiction (Purdue Pharma, 1997). That suggests that at least some patients were likely resistant to using opioids to treat pain. Third, pharmaceutical firms' promotional materials were heavily weighted towards health care providers. Purdue Pharma spent huge sums of money promoting their drug OxyContin. In doing so, they targeted prescribers, hospitals, pharmacies, and other components of the opioid supply chain. When conducting their pre-launch market research, they held numerous focus groups with providers; we have not found any evidence that they held similar focus groups with patients. Between 1996 and 2002, Purdue Pharma's yearly marketing strategy explicitly laid out physicians, nurses, managed care organizations, and long-term care as the primary audiences. Patients

only show up as part of the heading "patients and caregivers" in the "secondary audiences" table along with residents/fellows at teaching hospitals, wholesalers, and pharmacies (Purdue Pharma, 1996-2001). Fourth, patients' stated reasons for visiting a physician do not suggest that they were actively seeking medications. Using the same NAMCS data from before, Figure 11 shows the percentage of patients with chronic pain conditions who received an opioid prescription as well as the percentage of patients who were seeking a prescription in their physician visit.[23] Although the percentage seeking a prescription conditional on having a chronic pain condition rises over time, it is on average less than 30 percent of the fraction receiving an opioid and not increasing nearly as quickly. There was a general rise in the use of prescription drugs during this time period and many are pharmaceuticals that people can potentially take for a lifetime (e.g. statins, anti-depressants, blood pressure medications, anticoagulants, etc.), but those with chronic pain do not appear to have been any more likely to seek medications than those who did not have chronic or acute pain. Taken together, the evidence does not support the notion that the changes in prescribing were being driven entirely by patients.

Overall, the NAMCS data suggest that there are large changes in the willingness of physicians to write opioid prescriptions and these changes appear to occur after 1995, which aligns with the timeline we established in the previous section.

VII. Conclusion

Between 1979 and 1999, drug death rates were increasing at a fairly steady pace at which point they began to balloon. In this paper, we show that the SSDI rate in 1990 is a strong predictor of subsequent drug death rates. We present evidence that this relationship is not due to local economic conditions, general access to health services, the decline of institutions, or the rise and marketing of

[23] In the NAMCS, patients can list up to three reasons for the physician visit. The NAMCS then codes those reasons into broad categories including, "Medication, other and unspecified kinds" which excludes allergy medications, birth control, and any injections. Note that these could be prescription renewals rather than new prescriptions for a medication that the patient is not currently taking. If any of the reasons for the visit fall into this category, we code the person as seeking a medication.

OxyContin. Instead, the evidence is consistent with the hypothesis that the part of the 1990 SSDI rate that is correlated with drug death rates is proxying for the amount of pain in the county. This, combined with the liberalization of opioid prescribing in the 1990s and 2000s that we document, could explain a significant portion of the rise in drug deaths between 1995 and 2015. A simple back of the envelope calculation suggests that if pre-1996 differences in drug poisoning mortality across 1990 SSDI rates had been maintained after 1995, drug mortality would have been 43 percent lower in 2015.

Just as doctors were at the heart of the epidemic, they can also be part of the solution. Dun et al. (2023) and Zhang (2023) study recent interventions that are meant to reduce opioid prescribing and find that prescribing does change in response to the stimulus. In contrast, a similar RCT by Sarcany et al. (2016) among physicians that treat Medicare patients had little impact.

Although anecdotal, some support for our results comes from medical providers themselves. Noting that "physicians played a key role in starting the so-called opioid epidemic by overprescribing pain medication, and now must do their part to end it," the American Medical Association in 2016 passed a resolution that pain be removed as the 5th vital sign.[24]

[24] https://www.painnewsnetwork.org/stories/2016/6/16/ama-drops-pain-as-vital-sign

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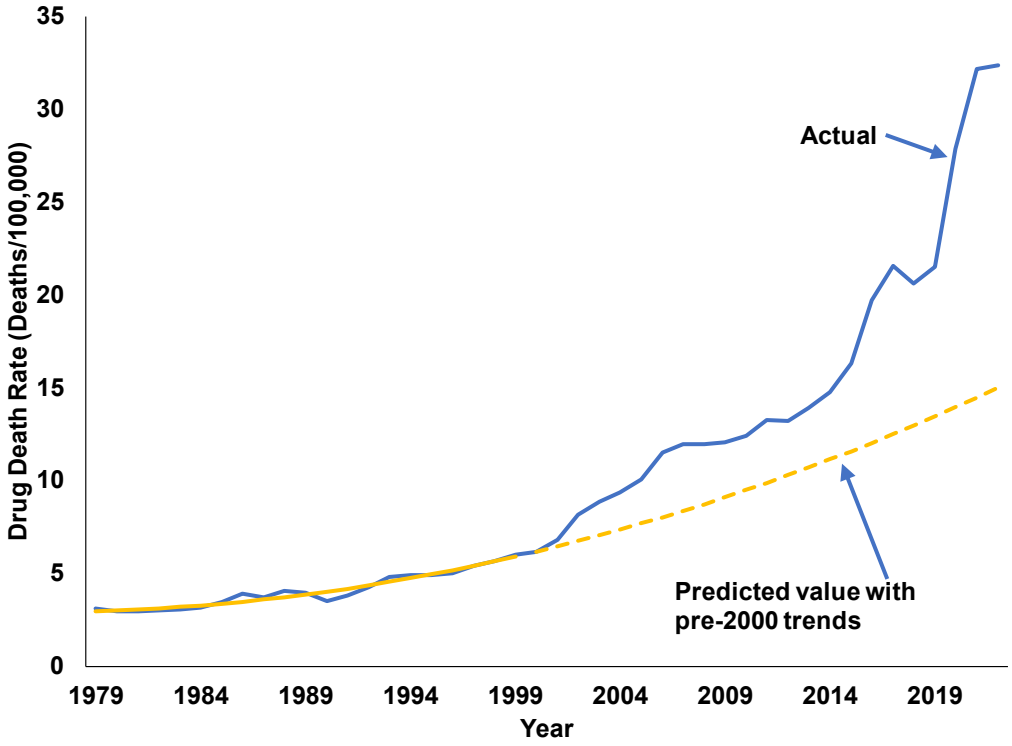
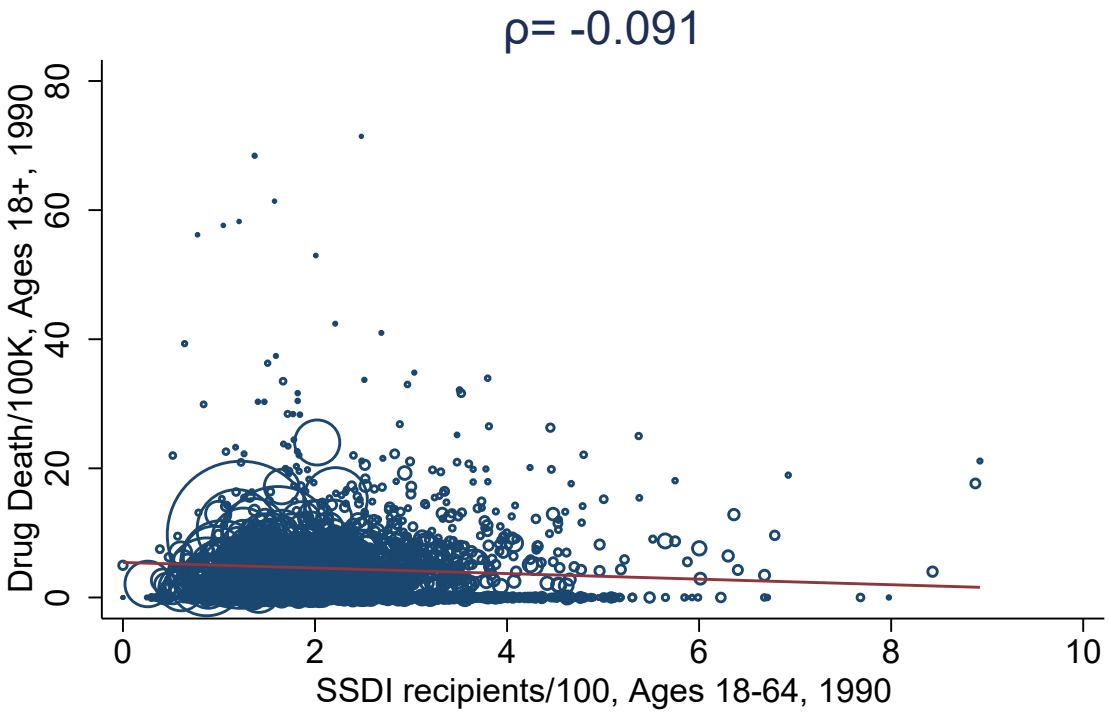
Figure 1
The Drug Poisoning Death Rate (Deaths/100,000) in the United States, 1979 to 2022



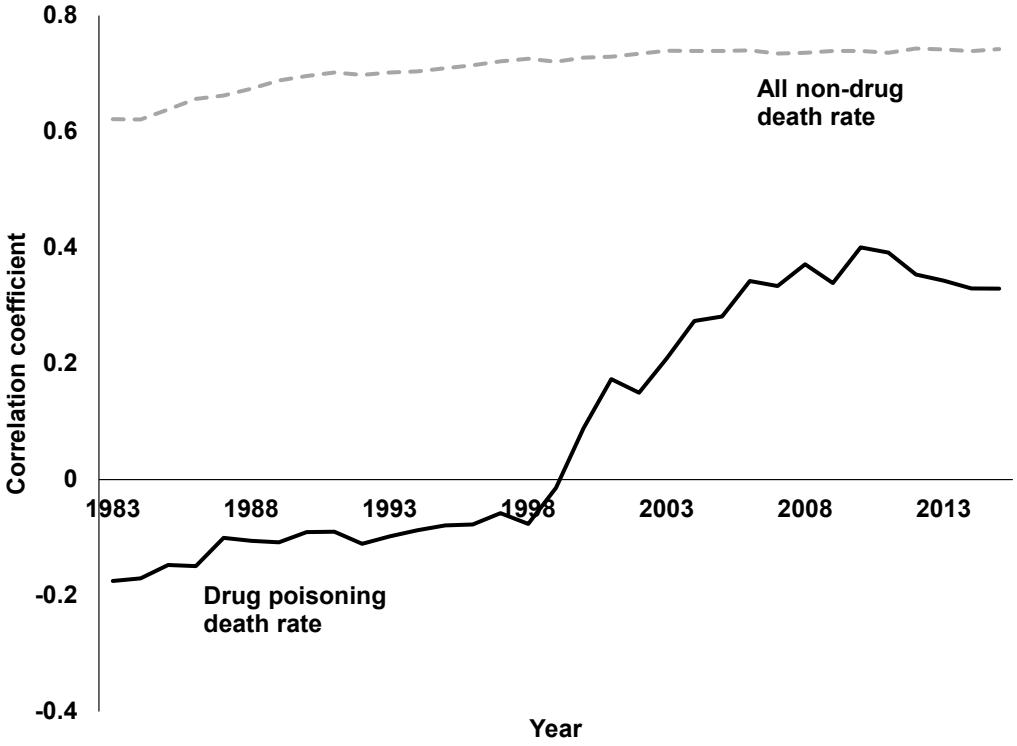
Notes: Data from CDC Wonder. The blue line is the actual drug poisoning death rate (deaths/100,000). The solid gold line is the predicted value using a quadratic trend for 1979-1999. The gold dotted line is predicted values using the pre-2000 regression.

Figure 2
Bubble Plot, the 1990 County SSDI Rate and the County Drug Death Rate in 1990



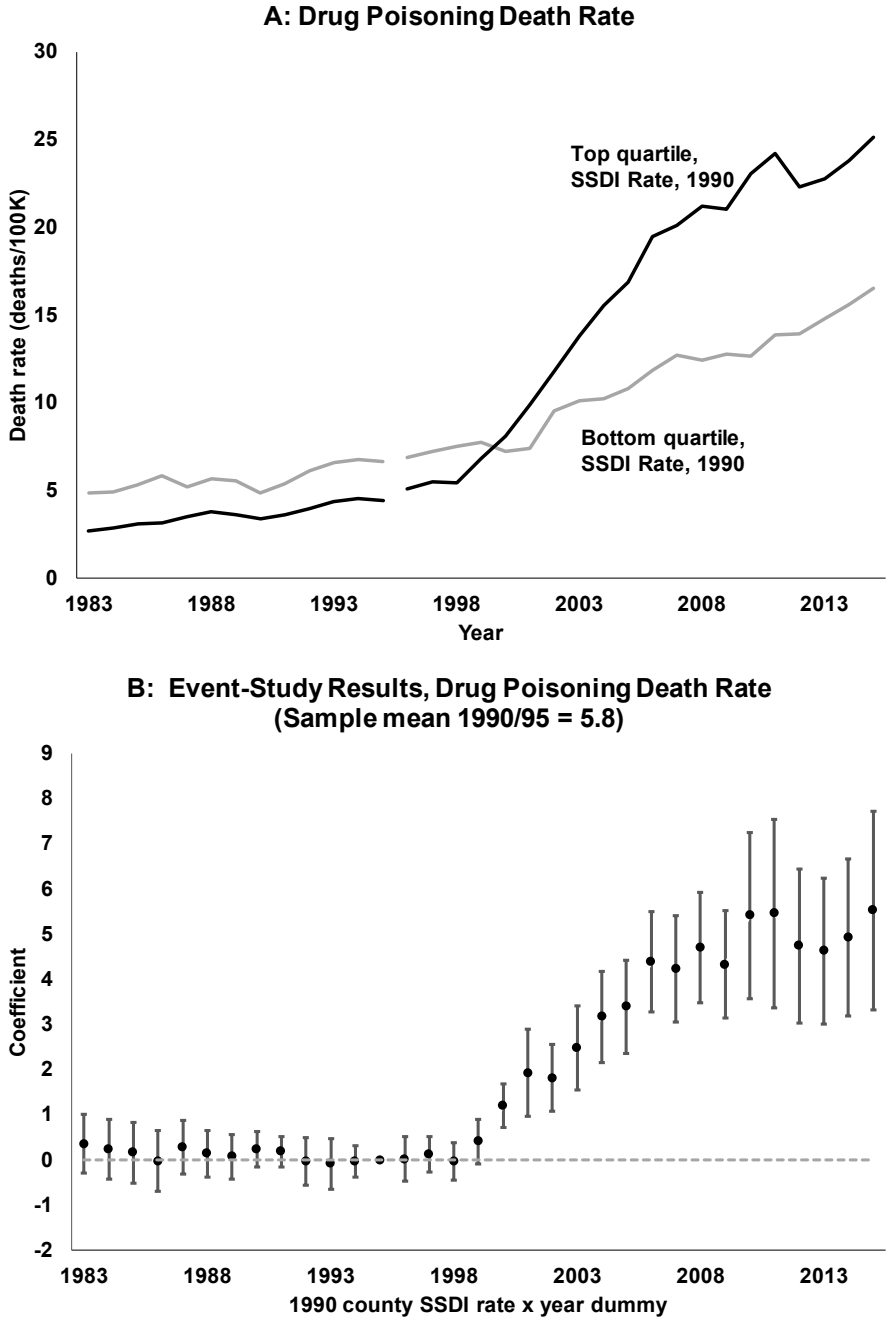
Notes: The size of the bubble is proportional to the size of the adult population, ages 18 and up.

Figure 3
Correlation Coefficient Between 1990 County SSDI Rate and
Adult County Death Rate for Various Years, 1990-2015



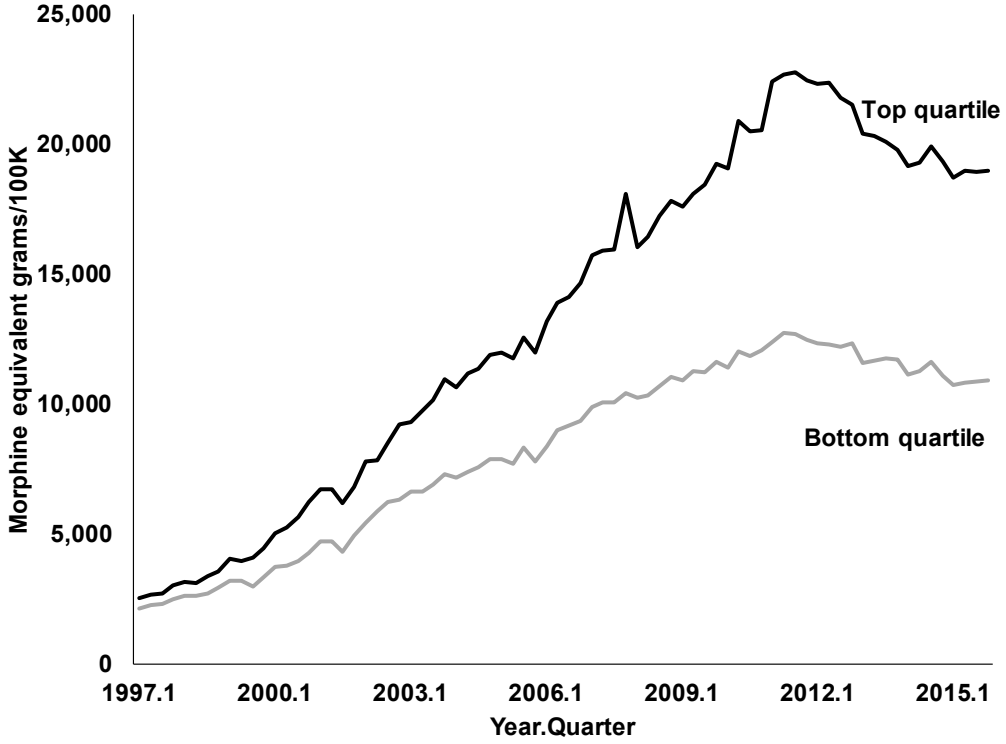
Notes: The graph represents the correlation coefficient at the county level between the cause of death rate in a particular year and the SSDI rate at the county level in 1990. The correlation coefficients are calculated by weighting by the adult population (ages 18 and over) in the county for that year.

Figure 4
Drug Death Rates for Adults Ages 18+ by Quartile of 1990 County SSDI Rate and Event Study Results, 1990-2015, Parameter Estimates and 95% Confidence Intervals



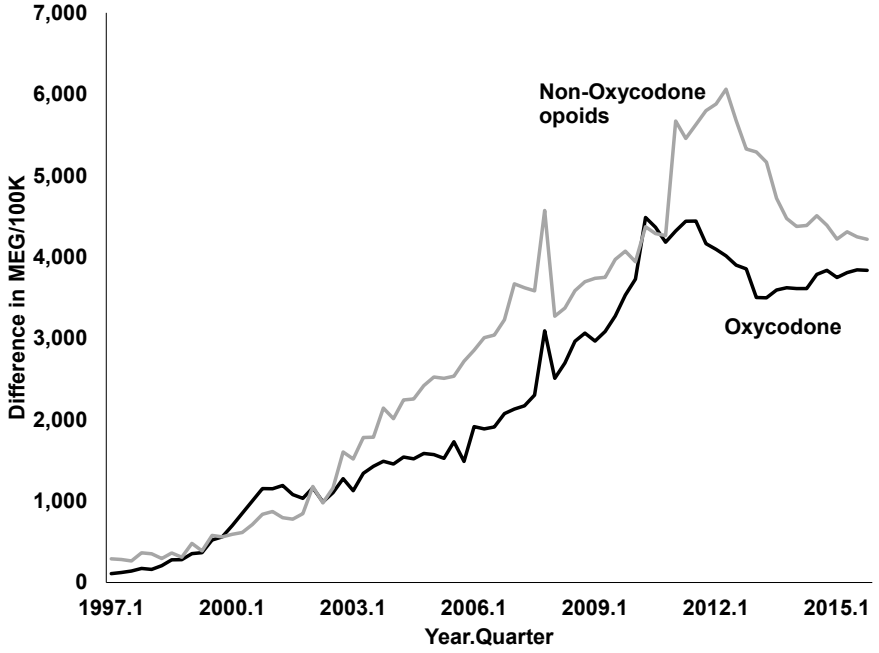
Notes: In graph A we aggregate deaths at the county level to counties that are in the top and bottom quartile in the 1990 SSDI rate at the county level, then divide by 100,000 in adult population for that county group. In Graph B, we report event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction between a year dummy variable and the SSDI rate in 1990. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level. The regressions have data for 3,106 counties over 33 years or 102,498 observations.

Figure 5
Time Series of Quarterly Morphine Equivalent Grams (MEG) per 100,000 Population for all Opioids by Quartile of County SSDI Rate in 1990, ARCOS Data, 1997.1 – 2015.4



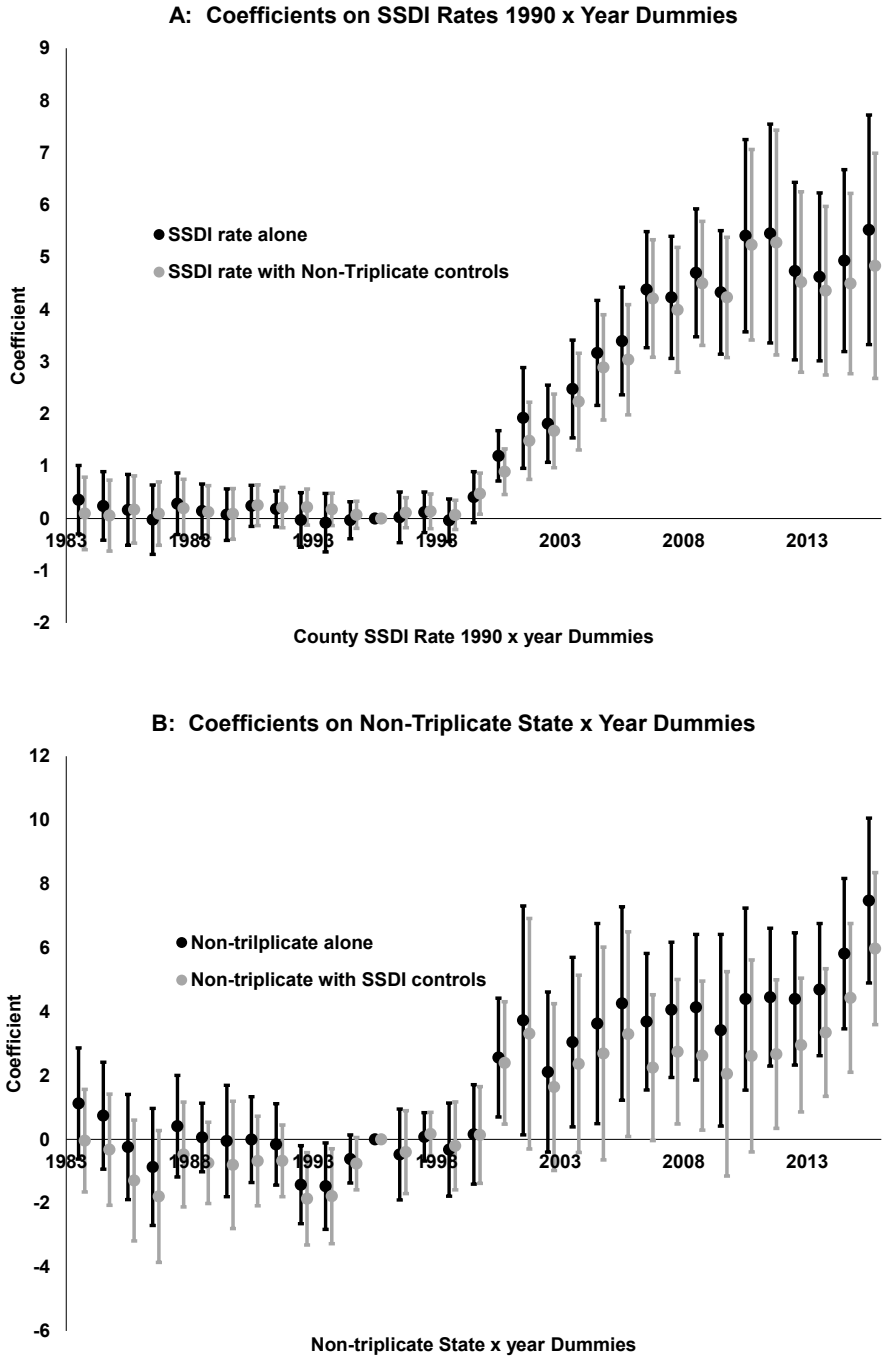
Notes: The graph shows the total morphine equivalent grams (MEG) per 100,000 people shipped to the counties in the top and bottom quartile of the 1990 county SSDI rate. The data is from ARCOS.

Figure 6
Time Series in the Difference in Quarterly Morphine Equivalent Grams (MEG) per 100,000 Population Between the Top and Bottom Quartile County in SSDI Rate in 1990, ARCOS Data, 1997.1 – 2015.4



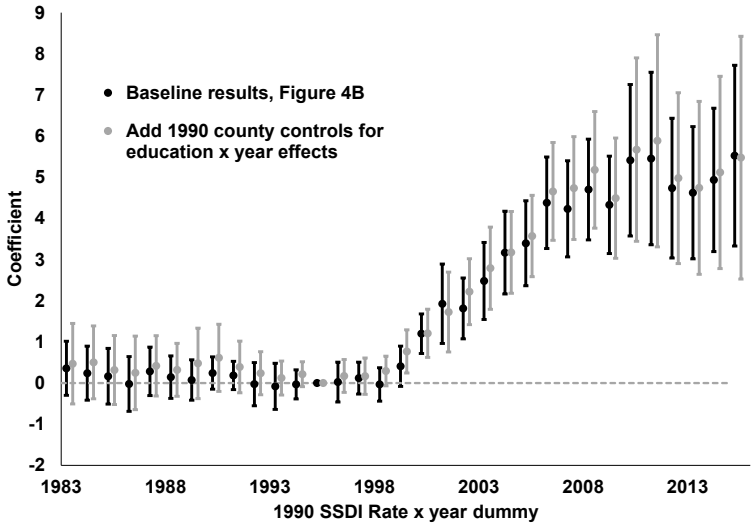
Notes: The graph shows the difference in total morphine equivalent grams (MEG) per 100,000 people shipped to the counties in the top and bottom quartile of the 1990 county SSDI rate. The data is from ARCOS.

Figure 7
Event Study Results, Drug Death Rate at the County Level, 1983-2015, SSDI and Non-Triplicate Treatments, Parameter Estimates and 95% Confidence Intervals



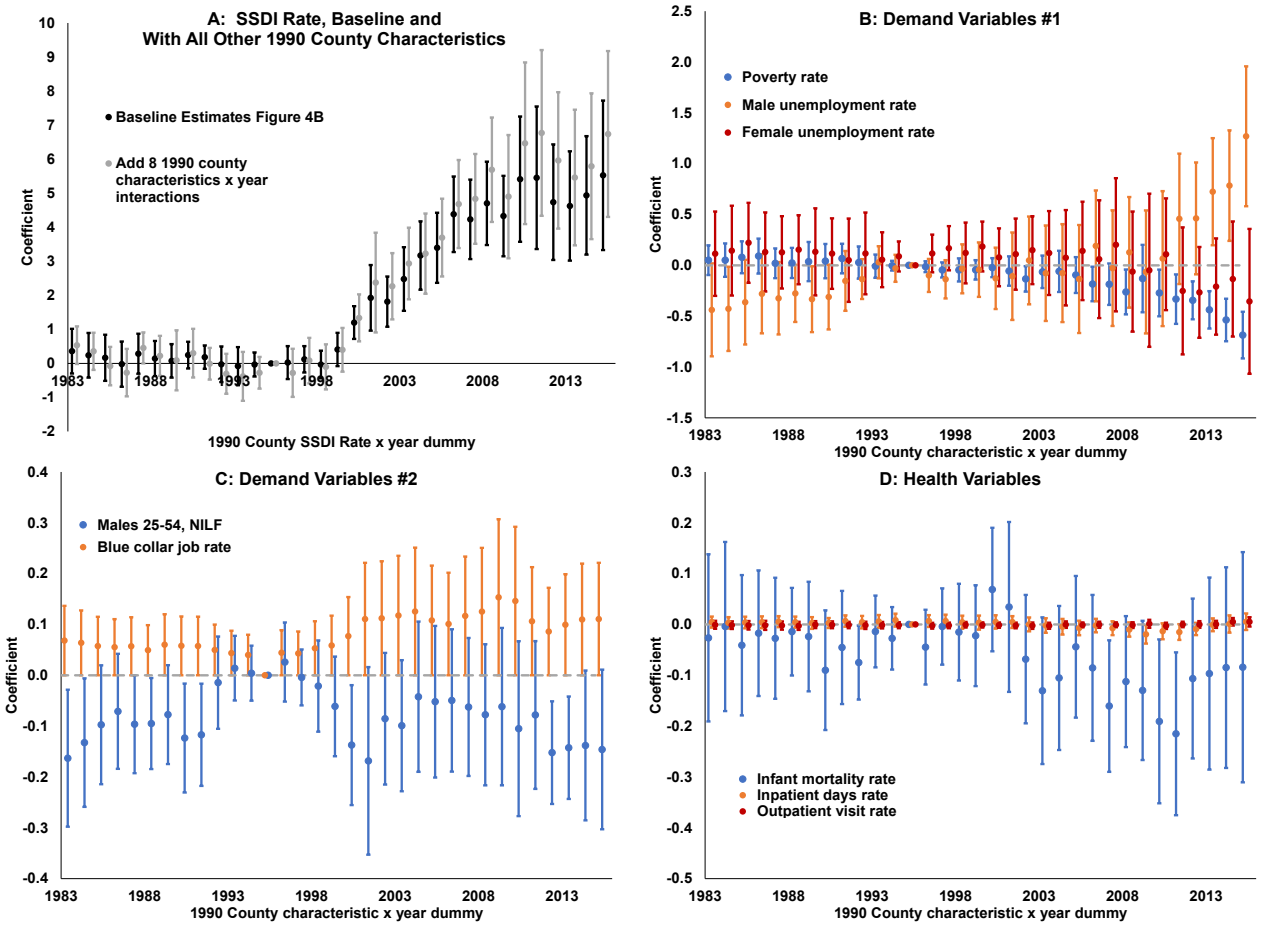
Notes: These are event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction between a year dummy variable and the SSDI rate in 1990 and interactions for non-triplicate states and year effects. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level.

Figure 8
Event Study Results, Drug Death Rate at the County Level, 1983-2015,
Adding County Level Education Variables in 1990 Interacted with Year Effects,
Parameter Estimates and 95% Confidence Intervals on the 1990 County SSDI Rate x Year Effects



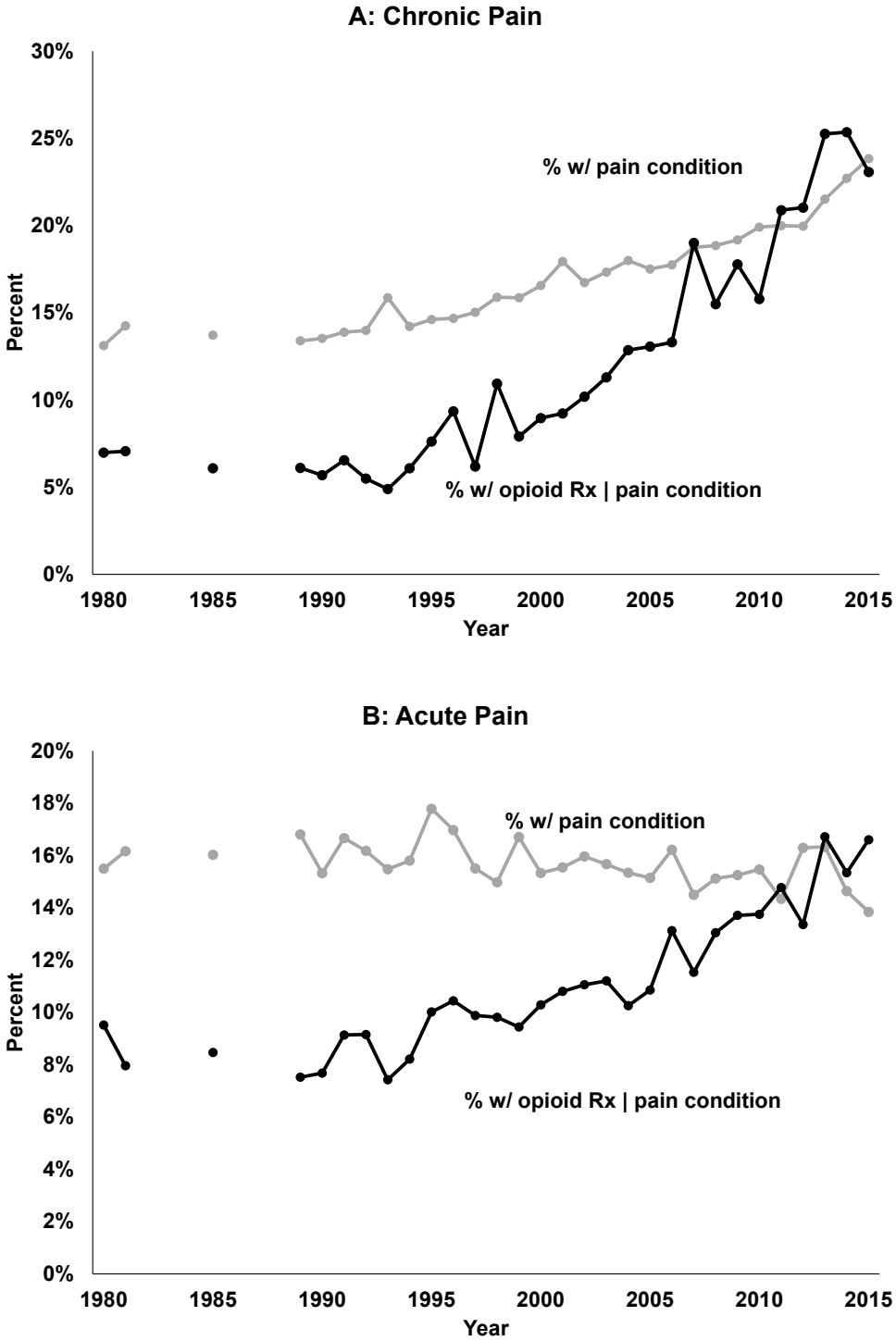
Notes: These are event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction between a year dummy variable and the SSDI rate in 1990 and interactions between year indicators and the fraction of the county's population in 1990 with a high school degree or less. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level.

Figure 9
Event Study Results, Drug Poisoning Death Rate at the County Level, 1983-2015,
Parameter Estimates and 95% Confidence Intervals



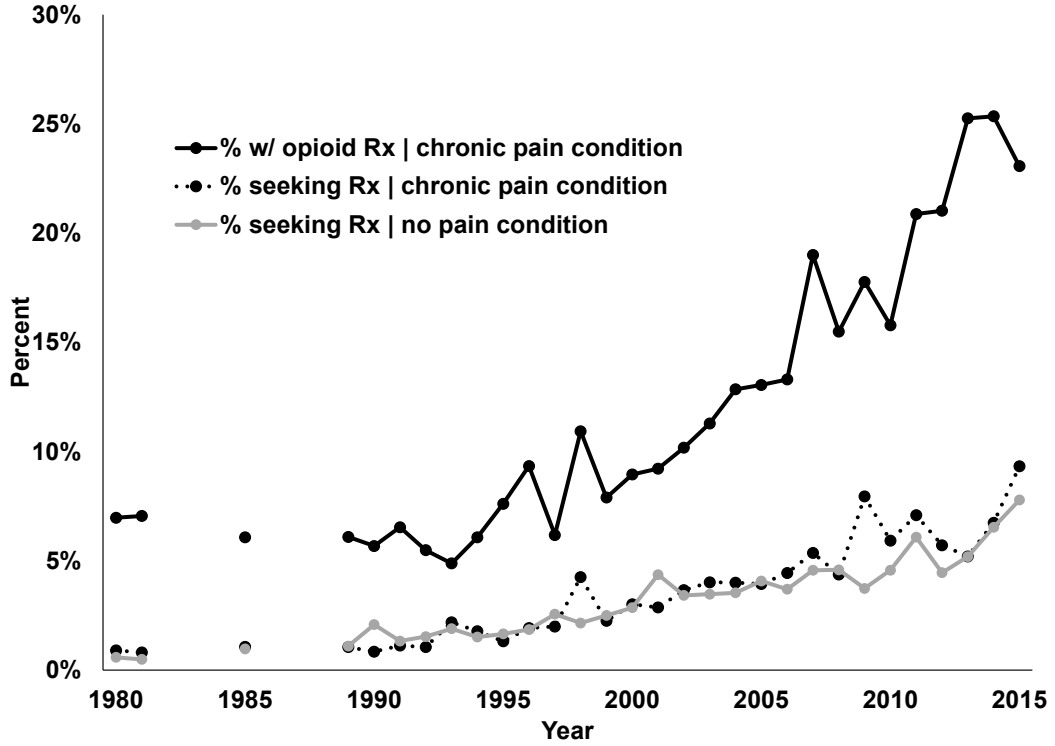
Notes: The estimates in Panel A are the same coefficient estimates and 95 percent confidence intervals for the event study model reported in Figure 4B. The figures show estimates of the 1990 SSDI rate times year interactions from equation (1). The estimates in grey are the same coefficients but with all eight county-level controls for 1990 (e.g., poverty rate, blue collar job rate, infant mortality rate, etc.) interacted with the year effects added to the model. In Panels B, C, and D, we report the parameter estimates and the 95 percent confidence intervals for these seven variables. Please see the notes to Figure 4 for more details about the estimation.

Figure 10
Time Series of the Percent Adults 18-64 Visiting a Physician in the NAMCS Data with Acute or Chronic Pain and Percent with Pain Receiving an Opioid Prescription, 1980-2015 NAMCS



Notes: These graphs use data from the 1979-2015 NAMCS. In each figure, the grey line is the fraction of the patients in the data with a chronic (panel A) or acute (panel B) condition. The black lines represent the fraction of the patients with that condition that received a prescription for an opioid at that visit.

Figure 11
Medication Seeking Behavior by Patients in the NAMCS, 1980 - 2015



Notes: This graph uses data from the 1979-2015 NAMCS. The solid black line is the fraction of the patients with an acute condition that received a prescription for an opioid at that visit. Patients in the data were asked whether they were seeing the physician because they were seeking a prescription. The solid line is the fraction of people with a chronic condition that reports that they are seeking a prescription while the doted black line represents people that do not have a chronic condition, but are seeking a prescription from the physician.

Table 1
Correlations Between 1990 County Characteristics and the Change in the Drug Poisoning Death Rate from 1995 to 2015

County characteristic in 1990	Correlation coef. between change in the county drug death rates, 2015-1995 (1)	OLS Estimates: Dep. var. is the change in the county drug death rates, 1995 to 2015	
		Variables one at a time (2)	All at once (3)
Measures of institutions			
% of adults 20-64 that are married[a]	0.2301	2.61 (0.20)	0.39 (0.31)
% adults that are adherents to formal religion[b]	0.0098	0.29 (0.28)	0.87 (0.30)
% workers in manufacturing/mining/agriculture[a]	0.1489	2.20 (0.27)	-0.15 (0.34)
Demand-based characteristics			
% population in poverty[a]	-0.0465	-0.74 (0.29)	-2.78 (0.69)
Median family income[a]	-0.1426	-1.42 (0.18)	0.50 (040)
% males aged 25-54 not in the labor force[a]	0.0218	0.41 (0.34)	-1.03 (0.43)
% adults 18+ with a high school degree or less[a]	0.2489	27.8 (1.96)	8.03 (3.67)
SSDI rate[c]	0.3880	5.90 (0.28)	7.31 (0.41)
Male unemployment rate[a]	0.0726	1.36 (0.32)	5.34 (0.54)
Female unemployment rate[a]	0.0082	0.13 (0.31)	-3.53 (0.60)
Share of people 16+ that are veterans[a]	0.2286	2.90 (0.21)	2.79 (0.31)
Non-drug deaths of despair death rate[a]	-0.0379	-0.91 (0.48)	-2.85 (0.47)
Supply-based characteristics			
Doctors/100K[d]	-0.1586	-1.59 (0.18)	-0.31 (0.24)
Hospital beds/100K[d]	0.0352	0.62 (0.36)	1.51 (1.57)
Inpatient days/1K[d]	0.0198	0.32 (0.35)	-1.61 (1.51)
Outpatient visits/1K[d]	0.0320	0.50 (0.30)	0.91 (0.38)

Notes: Results are generated by weighting observations by the average population at the county level for 1995 and 2015. There are 3,014 observations in these calculations

[a]Data is from 1990 Census Summary Tape Files, aggregated to the county level. Data is from IPUMS NHGIS (Manson et al., 2024).

[b]Data is from Association of Statisticians of American Religious Bodies (1990)

[c]Data is from Social Security Administration (1990) and Moore (2020) and represents the number of people enrolled in the Social Security Disability Insurance program as of December 1989. The denominator is the fraction of people aged 18-64 at the county level from the SEER data.

[d]Data is from the Area Resource File for the county level in 1990.

Table 2
The Link Between SSDI and Pain at the Individual and Local Area Level,
1997-2001 and 2011-2015 NHIS

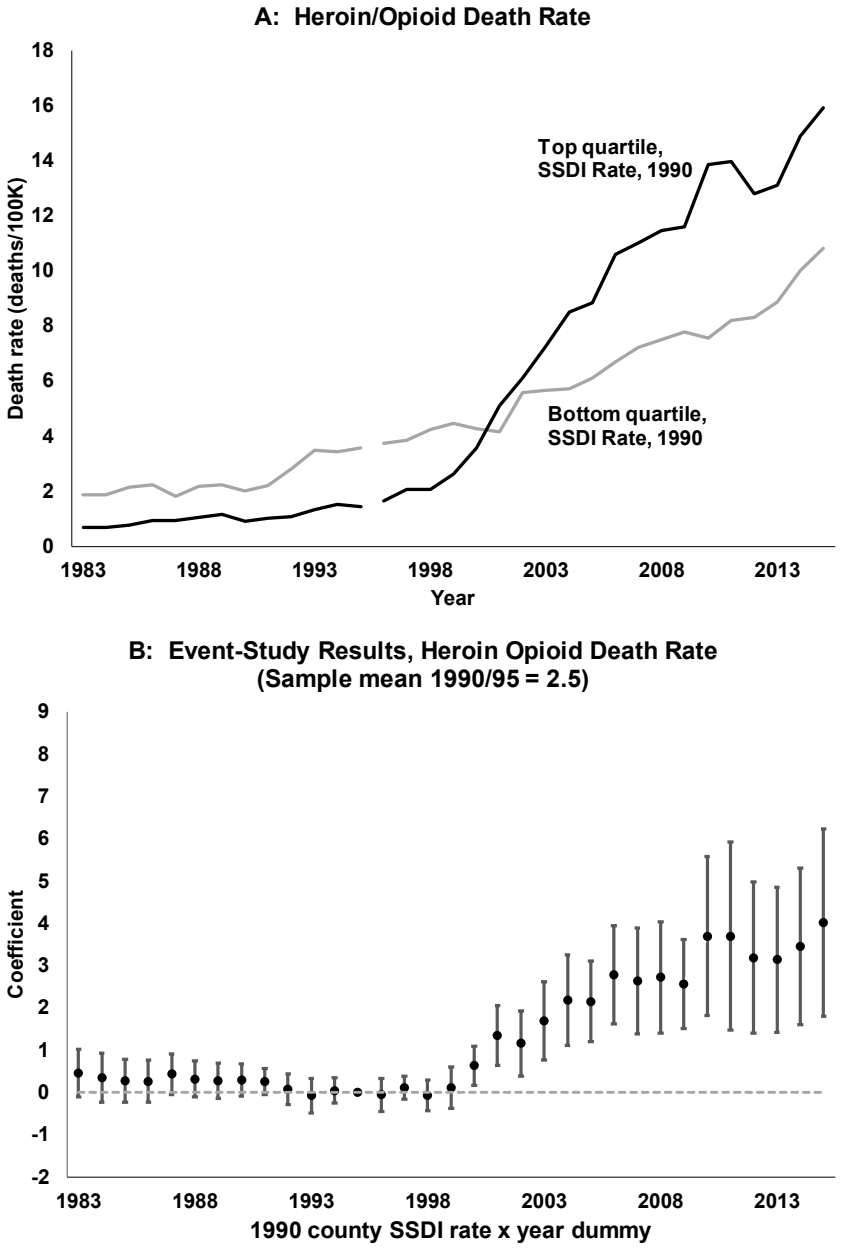
Row/Variable		Years of NHIS	
		1997-2001	2011-2015
(1)	Definition or chronic pain	Joint pain for more than a month in previous year	Pain on most/all days over the past 3 months
Sample means			
(2)	Adults 18-64 on SSDI [Obs.]	0.020 [301,913]	0.033 [318,617]
(3)	Adults 18+ with chronic pain [Obs.]	0.198 [165,057]	0.189 [74,517]
Chronic pain rates among people aged 18-64			
(4)	On SSDI	0.499	0.569
(5)	Not on SSDI (5) – (6)	0.161	0.152
(6)	Raw Difference (std. error)	0.339 (0.007)	0.417 (0.008)
(7)	Regression-adjusted difference (std. error)	0.272 (0.006)	0.370 (0.008)
(8)	Number of survey areas	678	600
Correlation coefficients: Between SSDI rate among people aged 18-64 and:			
(9)	Adults 18+	0.37	0.53
(10)	Adults aged 18+ NOT on SSDI	0.32	0.46

Notes: SSDI status is defined as being on Medicare aged 18-64. SSDI status can be obtained for all adults ages 18-64. The pain questions are asked of the sampled adult in a household for the 1997-2001 surveys, and those that were given the Adult Functioning and Disability Supplement. In row 7, we control for sex, a cubic in age, and three race/ethnicity groups (black non-Hispanic, other race non-Hispanic, and Hispanic). In row 9, we calculate a correlation coefficient between the SSDI rate for the 18-64 population and the chronic pain rates for adults ages 18 and up. In row 10, we calculate the same coefficient but drop those in the chronic pain mean aged 18-64 that are on SSDI.

Appendix A: Additional Tables and Figures

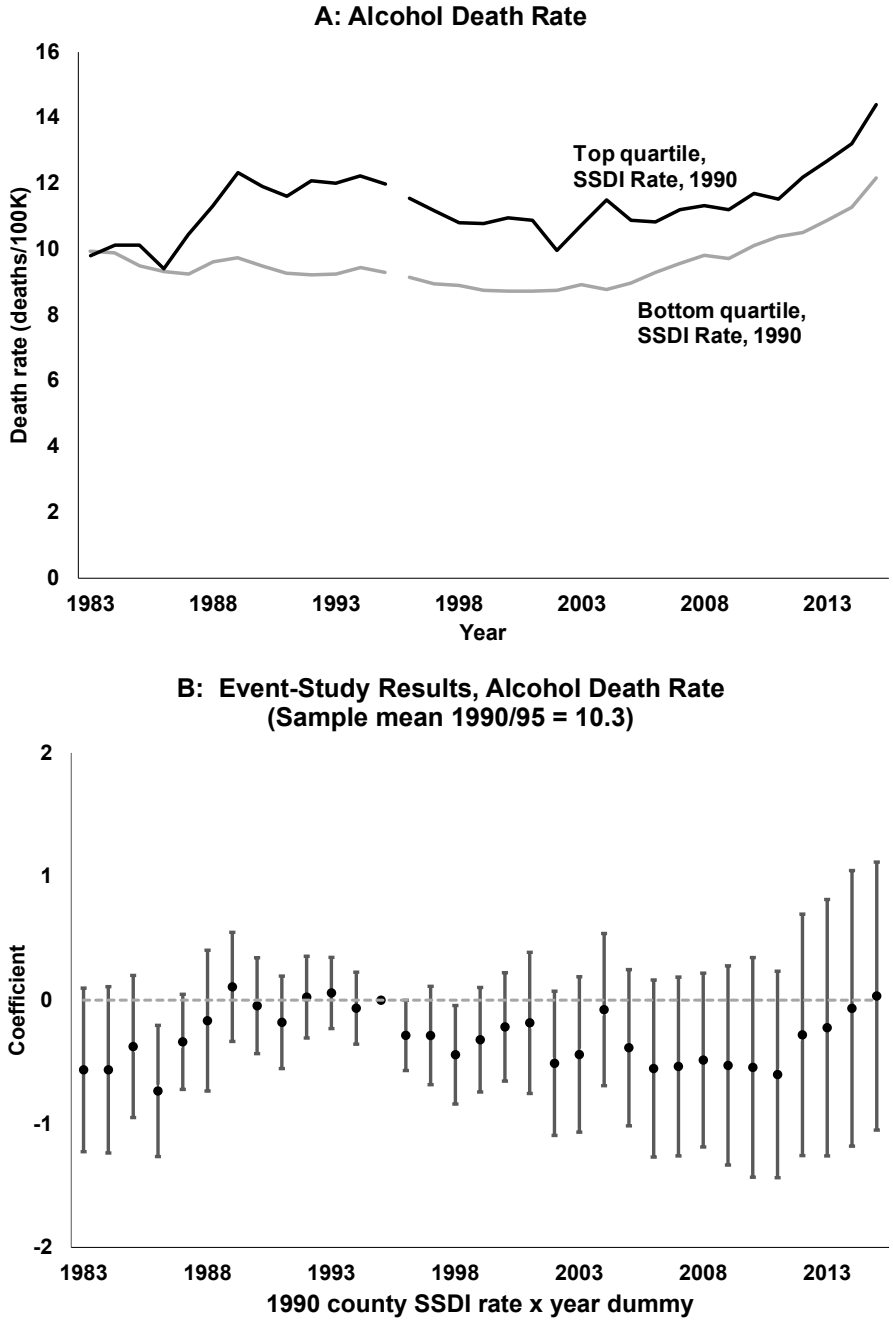
Appendix Figure A1

Drug Death Rates from Heroin/Opioids for Adults Ages 18+ by Quartile of 1990 County SSDI Rate and Event Study Results, 1983-2015, Parameter Estimates and 95% Confidence Intervals



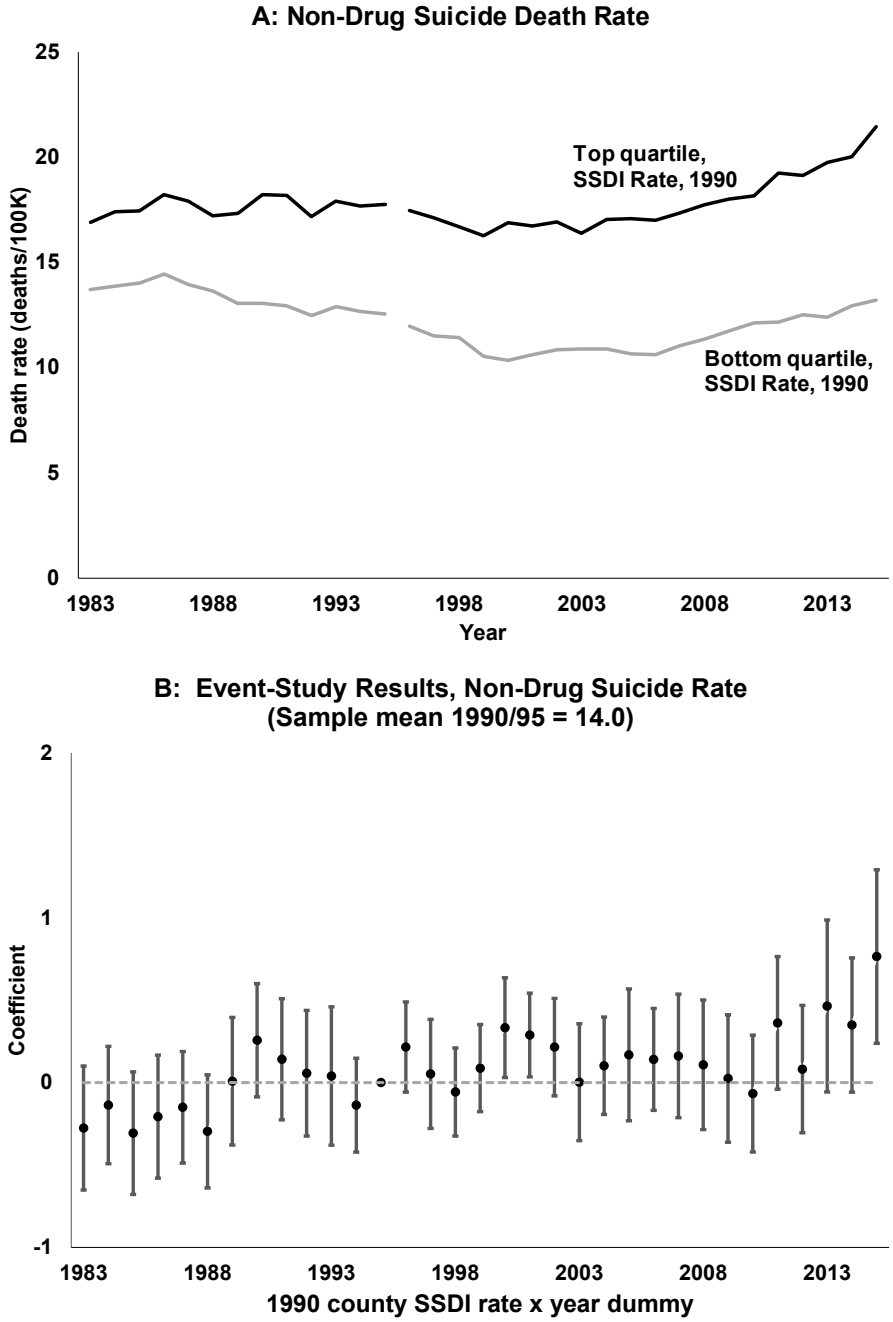
Notes: In graph A we aggregate deaths at the county level to counties that are in the top and bottom quartile in the 1990 SSDI rate at the county level, then divide by 100,000 in adult population for that county group. In Graph B, we report event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction between a year dummy variable and the SSDI rate in 1990. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level. The regressions have data for 3,106 counties over 33 years or 102,498 observations.

Appendix Figure A2
Alcohol Death Rates for Adults Ages 18+ by Quartile of 1990 County SSDI Rate and Event Study Results, 1983-2015, Parameter Estimates and 95% Confidence Intervals



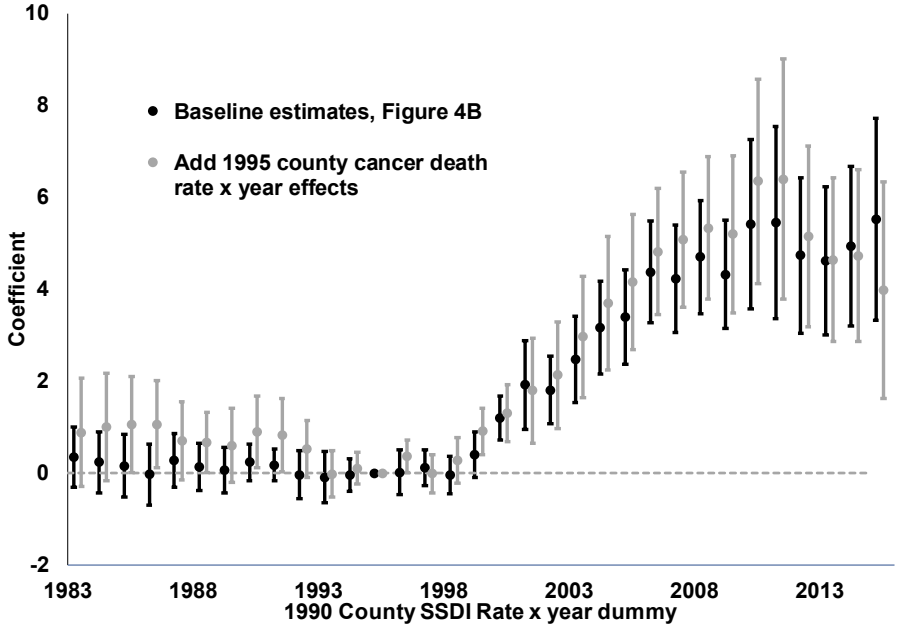
Notes: In graph A we aggregate deaths at the county level to counties that are in the top and bottom quartile in the 1990 SSDI rate at the county level, then divide by 100,000 in adult population for that county group. In Graph B, we report event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction between a year dummy variable and the SSDI rate in 1990. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level. The regressions have data for 3,106 counties over 33 years or 102,498 observations.

Appendix Figure A3
Non-Drug Suicide Death Rates for Adults Ages 18+ by Quartile of 1990 County SSDI Rate and Event Study Results, 1983-2015, Parameter Estimates and 95% Confidence Intervals



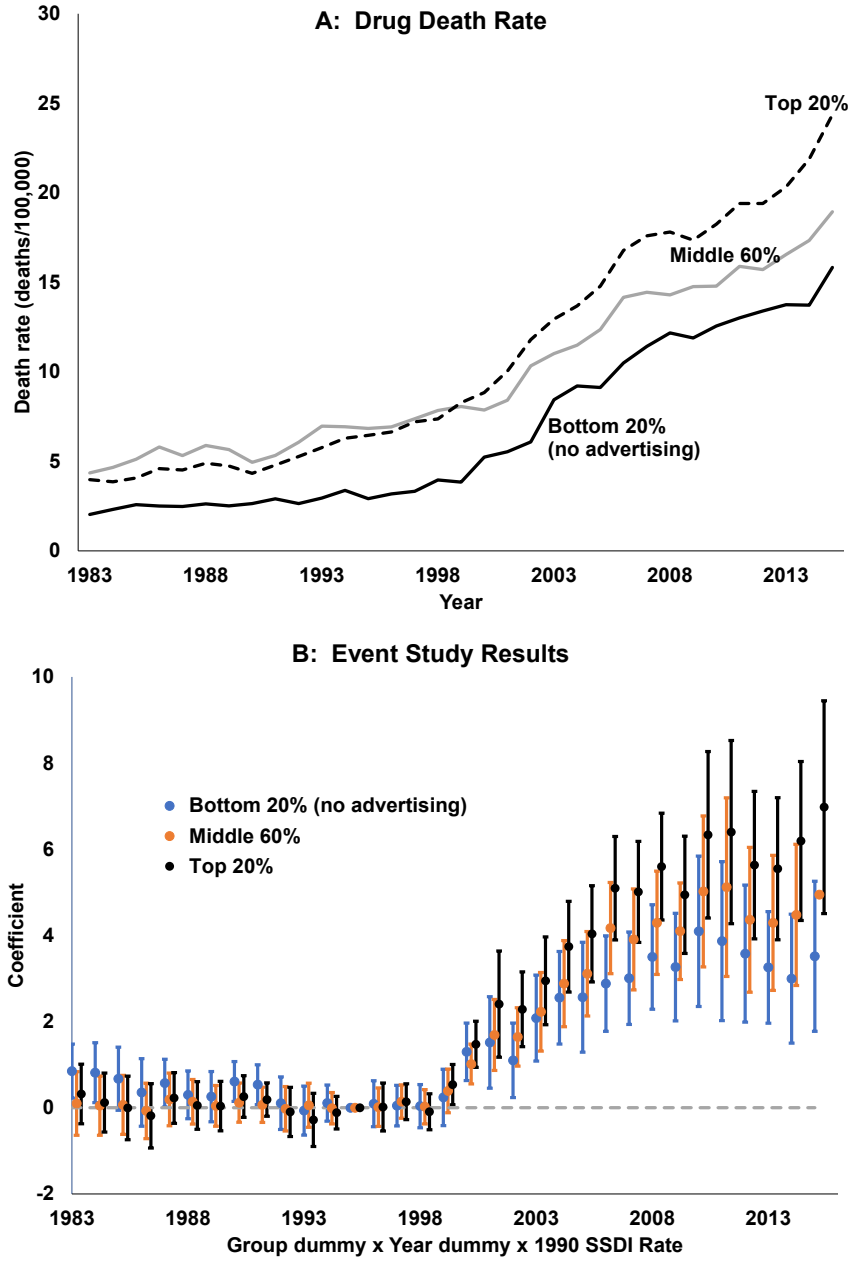
Notes: In graph A we aggregate deaths at the county level to counties that are in the top and bottom quartile in the 1990 SSDI rate at the county level, then divide by 100,000 in adult population for that county group. In Graph B, we report event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction between a year dummy variable and the SSDI rate in 1990. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level. The regressions have data for 3,106 counties over 33 years or 102,498 observations.

Appendix Figure A4
Drug Poisoning Deaths Rates, Event Study Estimates with and without the 1995 Cancer Death Rate Interacted with Year Effects



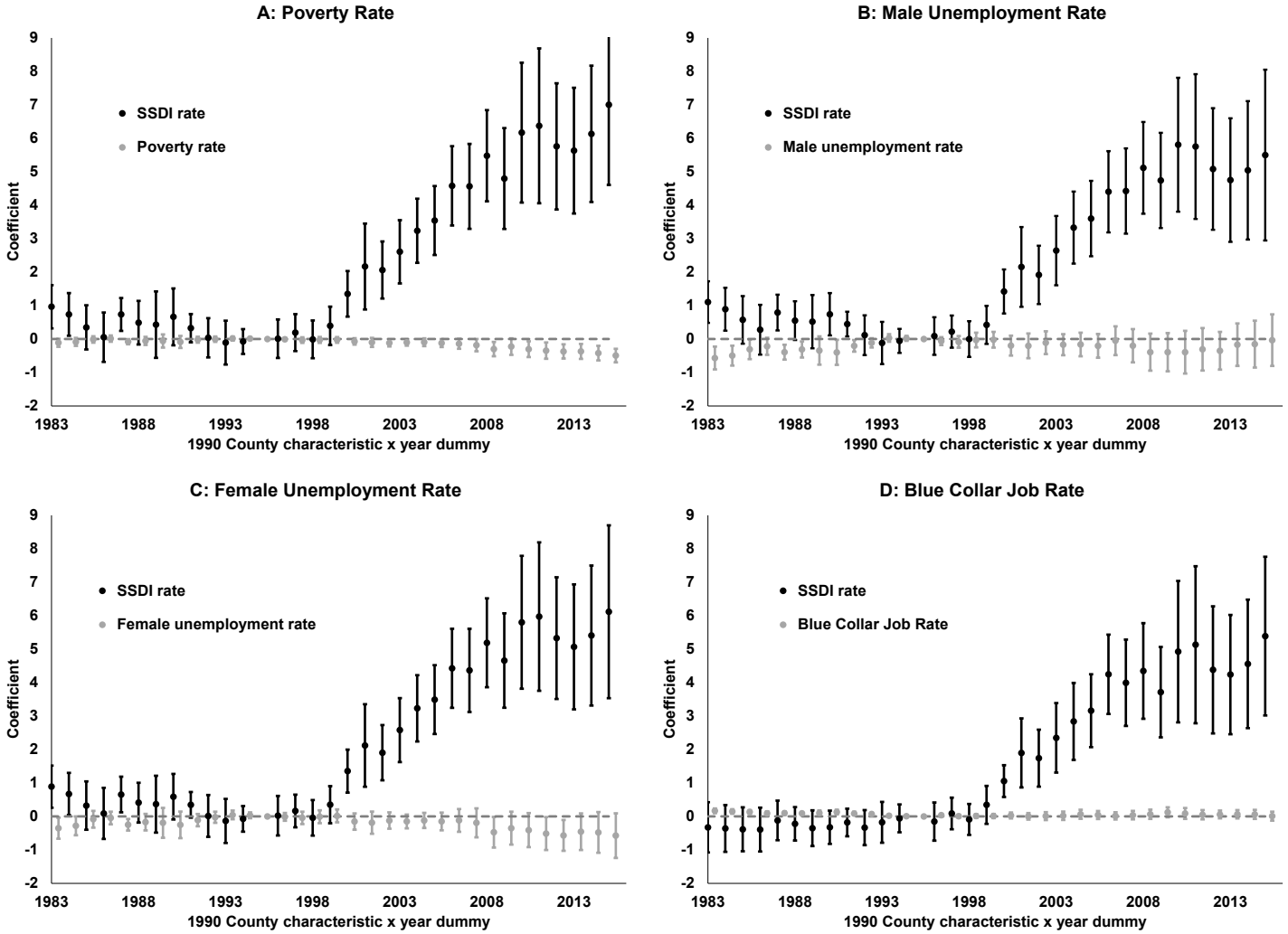
Notes: We report event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction between year dummy variables and the SSDI rate in 1990 with and without a set of variables (1995 cancer death rate interacted with year indicators) included in the regression. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level. The regressions have data for 3,106 counties over 33 years or 102,498 observations.

Appendix Figure A5
Drug Poisoning Deaths Rates Over Time and Event Study Estimates Based on Levels of Advertising Payments/1,000, in the Open Payments Data Set in 2016-2019

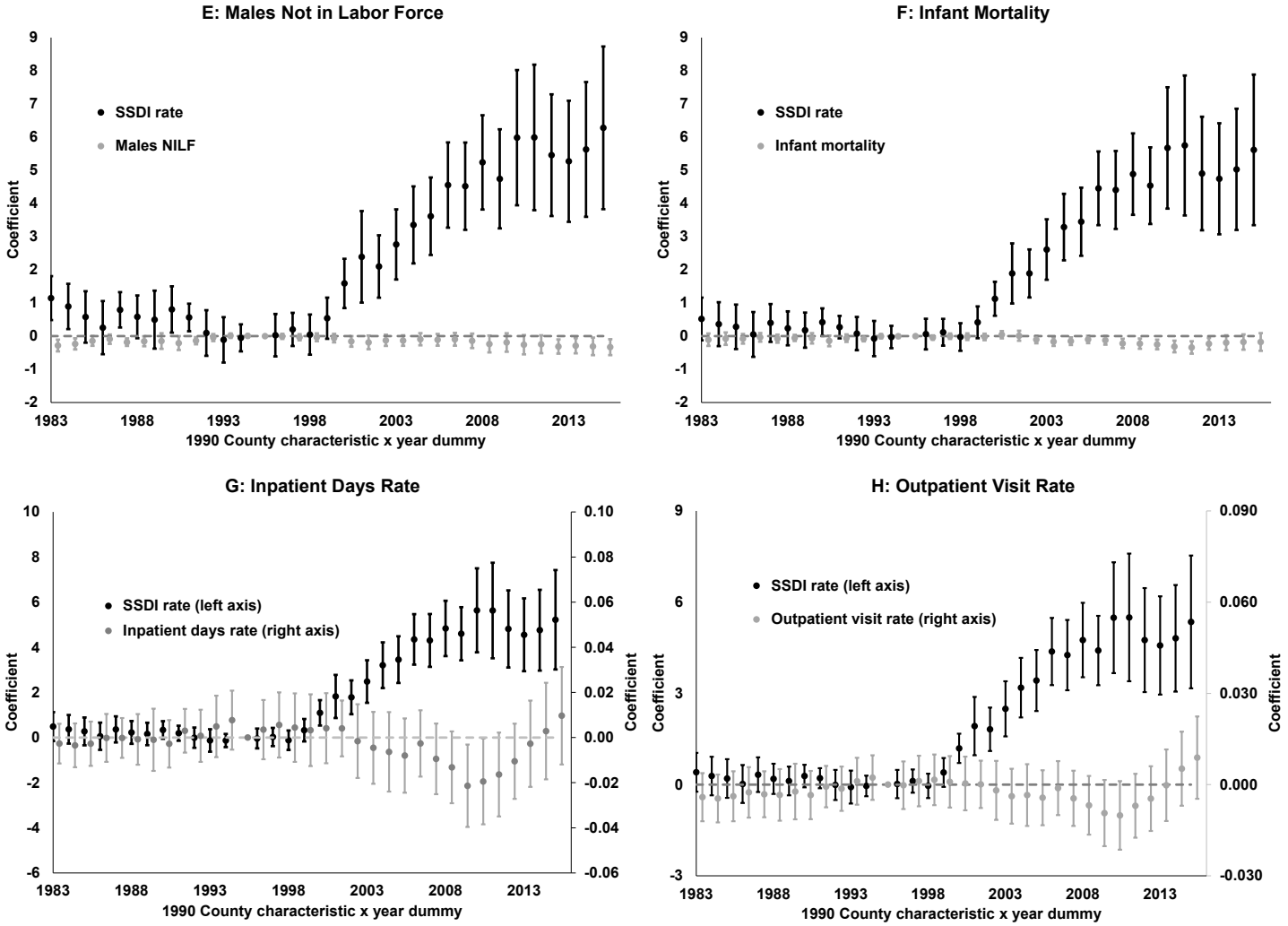


Notes: In graph A we aggregate deaths at the county level to counties based on the level of advertising in the Open Payments data set. We consider the lowest 20, middle 60%, and the top 20% of visits/1000 people in the county. We selected the bottom grouping as these are counties that have no advertising visits/1000. Graph B, we report event-study estimates from equation (1) and the 95 percent confidence intervals for the period 1983-2015 with the reference year being 1995. The coefficients are an interaction beta year dummy variable, the SSDI rate in 1990, and a dummy for what level of advertising there is in the Open Payments data set. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level. The regressions have data for 3,106 counties over 33 years or 102,498 observations.

Appendix Figure A6
Event Study Results, Drug Poisoning Death Rate at the County Level, 1983-2015,
Adding in One Additional 1990 County Characteristics Interacted with Year Effects at a Time
Parameter Estimates and 95% Confidence Intervals

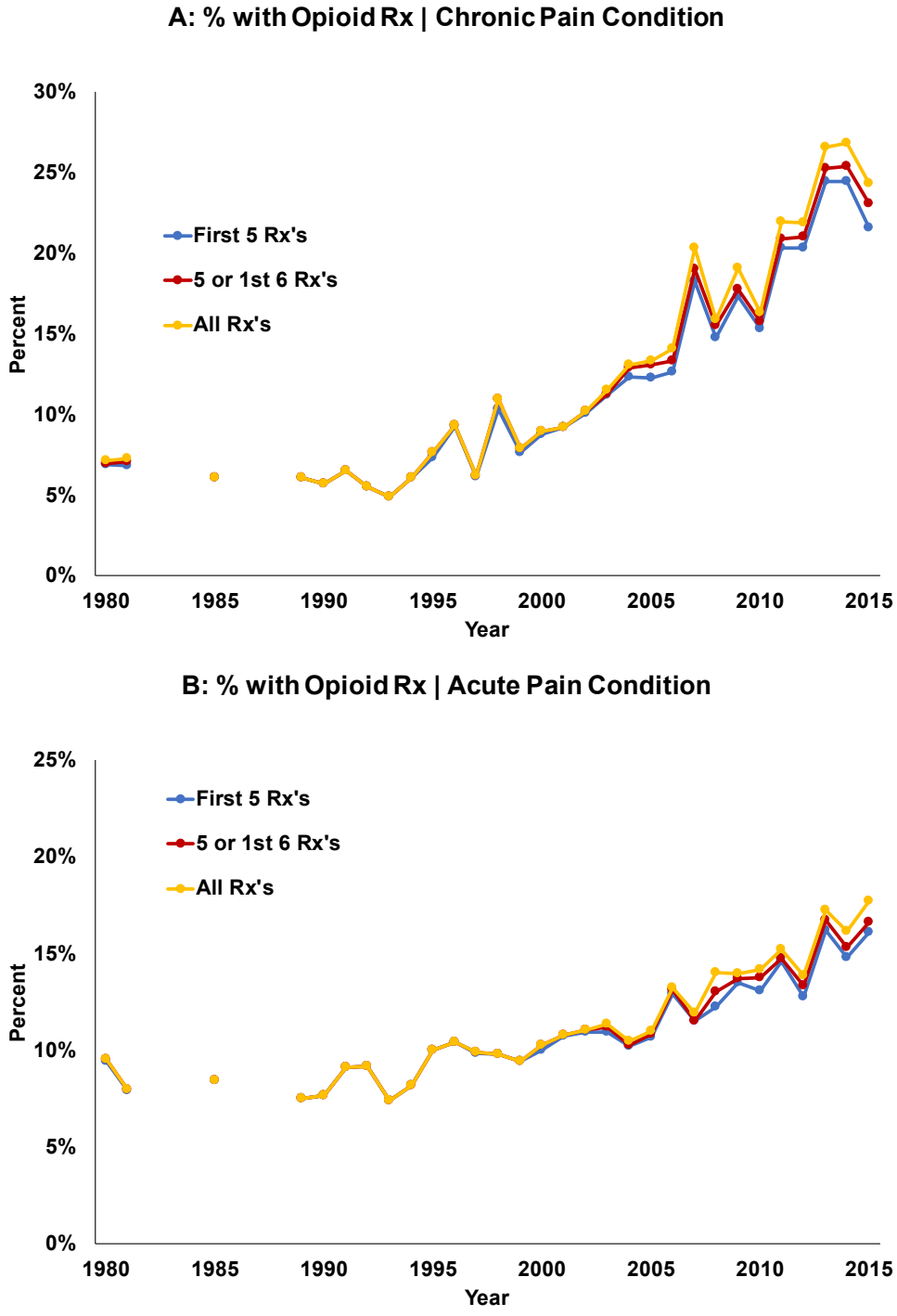


Appendix Figure A6 (continued)



In each of these graphs, we report event-study estimates from equation (1) for two sets of variables. The black dots and lines represent the interactions between a year dummy variable and the SSDI rate in 1990 while the results in grey are the year interactions with the other 1990 county characteristics (e.g, infant mortality in Panel A, etc.). In both sets of variables, 1995 is set as the reference year. Other controls include county and year effects, the fraction of people in the county that are 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, and 85 and up (with 18-24 being the reference group), the fraction black and other race (with white being the reference group), and the fraction Hispanic. The regressions are weighted by the adult population in the county/year and standard errors are clustered at the state level.

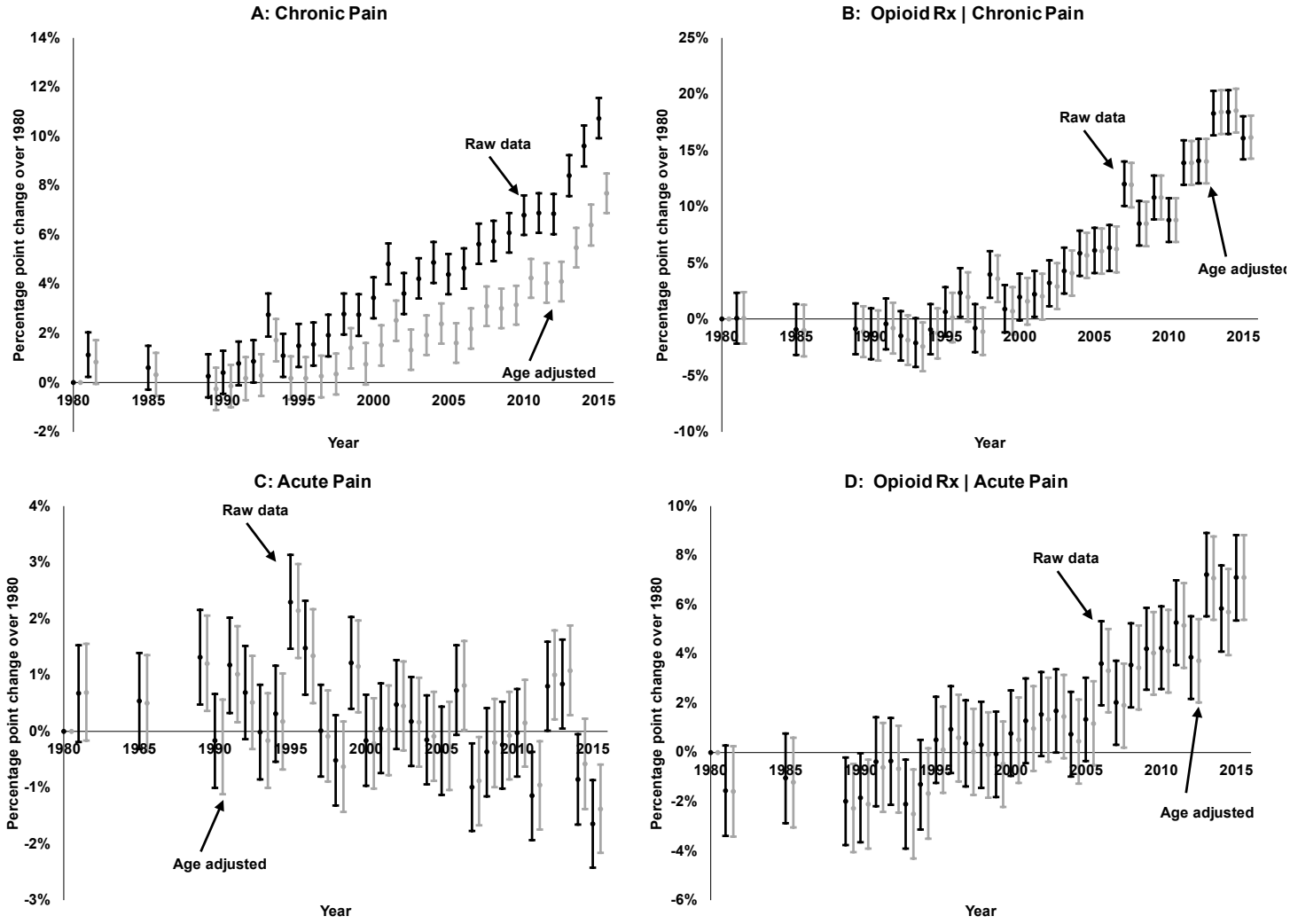
Appendix Figure A7
Fraction of Patients Presenting with a Chronic or Acute Pain Condition Receiving an Opioid Prescription Using Different Number of Prescriptions, Adults 18-64, 1980-2015 NAMCS Data



Notes: These graphs use data from the 1979-2015 NAMCS. The lines represent the fraction of the patients that received a prescription for an opioid at that visit where we use either the first five prescriptions listed, the first five prescriptions in years with only five available lines for prescriptions or the first six in years where there are six available lines, or using all listed prescriptions with no adjustments for different potential numbers of prescriptions in different years due to survey design.

Appendix Figure A8

Raw Difference in Outcomes from NAMCS Data using 1980 as The Base Year, and Regression-Adjusted Differences Controlling for Age



Notes: In each panel, we report two regressions. The black lines are the parameter estimates and the 95 percent confidence intervals of regression of the outcome (e.g., the respondent has a condition likely to produce chronic pain) on a set of year dummy variables with 1979 being the reference year. The grey lines are the same variables from a regression where we include a full set of age variables.

Appendix B: The Multiple Cause of Death Data

We obtained access to the restricted-use version of the MCOD files that identify the county of residence of the deceased. We start our analysis in 1983 for two reasons. Although publicly-available versions of the MCOD files identify counties in 1982 and before, county FIPS codes were not used until 1982. Prior to then, the data used an NCHS county code. One problem with the NCHS code is that they treat all five counties that make up New York City as one county (Bronx, Kind, Queen, Manhattan and Richmond). We do not use 1982 because in that year, there was a 50 percent sample for 19 states, instead of a full 100 percent sample for all states as in other years. We end our analysis in 2015 as fentanyl starts to dominate drug deaths around that time. Between 2010 and 2020, the fraction of drug deaths that were identified as caused by a synthetic opioid went from 8.2 to 62.5%. Between 2015 and 2016 alone, deaths involving a synthetic opioid death rose from 9,803 to 19,720.

The MCOD data uses cause of death codes from two different versions of the International Classifications of Diseases: ICD-9 (1978-1998) and ICD-10 (1999-2015). Identifying drug overdoses in both versions of the ICD system is relatively straightforward. In each year, there are three sets of codes that identify unintentional poisoning deaths, intentional poisonings (e.g., suicides), and drug poisoning of unknown intent. These codes vary by the class of drug. ICD 9 has some additional codes under mental health: drug psychoses (292) and drug dependence (304). These codes were dropped in subsequent versions. In the ICD 9 system, code E962.0 measures death from homicide due to drug poisonings. That code under the ICD 10 classification is X85. We list these codes in Table B1 below.

For alcohol deaths, we use a broader classification than just liver cirrhosis and mostly use the codes suggested by Unites States Congress (2019) with some exceptions.[25] We also construct a death rate for non-drug suicides. We include suicide by alcohol poisoning (X65) in both the alcohol and non-drug suicide groups. The codes for these categories are in the final two columns of Table B1.

Identifying opioid deaths is relatively easy in ICD 10 as there are codes that identify conditions present at death to indicate specific drugs. These include T40.1 (heroin), T40.2 (other opioids) T40.3 (methadone), and T40.4 (synthetic opioids). Like Alpert et al. (2022), we also include T40.6 (other and unspecified narcotics) as well. There are similar codes in the ICD 9 classifications: 965.0 (opiates and related narcotics), 965.1 (heroin), 965.2 (methadone), 965.9 (other opiates and related narcotics).

The problem we found is that in many cases during the ICD 9 era, the "965" condition codes are frequently not used when there was a drug death. In the ICD 9 era, we can identify opioids in some of the "E" codes – E850.0 (heroin), E850.1 (methadone), and E850.2 (opiates and related narcotics). Unfortunately, categories E950.0 and E980.0 (poisonings by analgesics, antipyretics, and antirheumatics for intentional and unknown intent, respectively) lump opiates in with other drugs (mostly non-opioid pain relievers).

In the ICD 10 era, the T39 condition code identifies non-opioid analgesics, antipyretics, and antirheumatics. In 1999 there were only 759 deaths from these drugs, but 8,645 of the T40.x opioid/heroin deaths. As a result, to make a more consistent series without a noticeable jump in opioid deaths as we move from the ICD 10 back to the ICD 9 era, we use a broader opioid death rate category that includes the T39 cases. In the ICD 9 era, we consider the "965" conditions listed above, those that include non-opioid analgesics, and any E850.x code which contains opiates and the non-opioid analgesics, plus deaths with E950.0 and E980.0 codes.

[25] The ones we excluded from this classification were associated with the ICD-10 system. We did not use P04.3 as that is a code for alcohol and newborns. We did not include codes G62.1 (alcohol polyneuropathy) and R78.0 (traces of alcohol in blood) as deaths from these were only recoded in a small number of years.

Table B1
ICD 9 and 10 Codes to Identify Causes of Death

Coding system	Drug deaths	Alcohol deaths	Non-drug Suicides
ICD-9 1978-1998	292, 304, E850-E858, E950.0 – E950.5 E980.0 - E980.5, E962.0	2971.3, 303, 350.0, 357.5, 425.5, 535.3, 571.0-571.3, 790.3, E860	E950.6-E959
ICD-10 1999-present	X40-44, X60-64, Y10-14, X85	E24.4, F10, G31.2, G72.1, I42.6, K29.2, K70, K85.2, K86.0, O35.4, Q86.0, X45, X65, Y15	X65-X84

In our analysis, we only consider deaths for adults ages 18 and above because the vast majority of drug poisoning deaths are for adults. Between 1999 and 2020 there were more than 932,000 drug poisoning deaths and less than 1 percent were among those under 18.

Appendix C: County Population Data

To obtain the denominator for death rates, we use county-level population estimates by age, race, ethnicity, and sex from the National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) program.[26] We use two of the SEER data sets. First, we use population counts for single ages broken down by race and sex. This gives us population by race for three groups: black, white, and other. Counts of Hispanics by age are only available back to 1990, so we use this sample for this variable. We then use data on county population counts for Hispanics by age from the NHGIS for 1980 and interpolate annual population values for the inter-census years assuming any change between 1980 and 1990 happens smoothly over the decade.

To match the SEER data, the aggregate Census data from 1980, and the MCOD data at the county level, we needed to process some counties to make definitions compatible over time. A catalog of these changes is below.

- Population data at the county level before 1990 in Alaska is limited. SEER does not have data by age prior to 1990 and population counts by age are not reported in Census tables for all counties. We were able to get complete series from 1982 forward for only 12 counties and the rest of the counties are aggregated into a "rest of state" county.
- Broomfield, CO was created out of four counties: Adams, Boulder, Jefferson, and Weld. For the analysis sample, we aggregate these into one county in all years.
- All data for Hawaii is aggregated to the state level.
- We merge South Boston, VA (independent city) into Halifax County in all years.
- We merge Bedford City, VA (independent city) and Bedford County into one county.
- We merge Clifton Forge, VA (independent city) into Alleghany County
- La Paz and Yuma counties in AZ are merged into one county.
- We delete all data for Yellowstone County, MT

[26] https://seer.cancer.gov/popdata/

Appendix D: Additional Information on the ARCOS Data

The most disaggregated ARCOS data publicly available come at the quarter-year by three-digit zip code unit of observation. We obtained these data for the years 1997 – 2015. Attempts to obtain data prior to 1997 via FOIA requests uncovered that the DEA no longer has pre-1997 data and attempts to find the pre-1997 data via the Wayback Machine were not successful.

To convert from three-digit zip codes to counties, we first used zip code level population data from the 2010 Census to convert from three digit to five-digit zip codes. From there, we used a crosswalk between five-digit zip codes and counties developed by the Department of Housing and Urban Development. The resulting crosswalk from three-digit zip codes to counties does not vary by year. It was then applied to the three-digit ARCOS data to recover grams of each drug in each county and quarter year.

Appendix E: Sensitivity Tests within a Difference-in-Difference Model

To help summarize the event study figures and to facilitate robustness checks, we also estimate the following difference-in-differences model.

$$(2) \quad y_{ct} = x_{ct}\beta + (SSDI\ Rate_{c1990})\left[\pi_{9600}1(1996 \le year \le 2000) + \pi_{0105}1(2001 \le year \le 2005)\right.$$
$$\left.\pi_{0610}1(2006 \le year \le 2010) + \pi_{1115}1(2011 \le year \le 2015)\right] + \mu_c + \lambda_t + \varepsilon_{ct}$$

Instead of interacting the 1990 SSDI rate with separate year dummies, we use dummies for five-year intervals starting with 1996 to 2000. The other covariates are the same as in equation (1). We again weight by adult population in the county and cluster standard errors at the state level.

Table E1 presents results from estimating Equation (2) where our dependent variable is the drug poisoning death rate. This table examine the sensitivity of the results to the inclusion of controls for non-triplicate states as in Alpert et al. (2022). The first column presents results from the baseline specification. By the 2011-2015 time period, the coefficient on the SSDI rate interacted with time is 4.92 with a t-statistic of almost 5. The mean drug death rate in the 1990/95 period is 5.8 and the mean SSDI rate across counties of 2.3. This means the county with average SSDI rates experienced an increase in drug deaths between 1995 and 2011/15 period by 11.3=4.92*2.3, nearly double the rate of the mean in the 1990/95 time period. In the second column, we only include dummies at the state level that indicate whether the state was a non-triplicate status interacted with year group dummies. The results in this column are very similar to the results in Alpert et al. (2022). To make sure our results are not being driven by this marketing effect, we re-estimate our main specification but include both our 1990 SSDI rate by year-group interactions as well as the non-triplicate by year-group interactions. As seen in the final column of Table E1, both sets of interactions continue to be important determinants of drug poisoning death rates. The SSDI interactions are reduced by 17 percent in the 2000-2004 period and by only 5 percent in the 2006-2010 period. In contrast, the results for the non-triplicate interactions are reduced by 21 percent in the 2006-2010 period and by 16 percent in the 2011-2015 period.

In Appendix Table E2, we present some other sensitivity results. The first column of the table replicates the results from the first column of table E1. Because the increased drug deaths were concentrated among a subset of demographic groups, we test the sensitivity of our results to omitting our demographic controls. The second column shows that this does not materially affect the results. In the third column, we include controls for prescription drug monitoring programs (Horwitz et al., 2018),

a common policy that states have implemented to combat the opioid epidemic, and again find that there is little impact on the point estimates.

Adding in linear, county-level time trends reduces our point estimates slightly, but their inclusion does not overturn the qualitative finding that higher 1990 SSDI rate counties saw considerably greater growth in drug poisoning death rates over time. As a final way to control for all policies that vary at the state level, we include state-by-year fixed effects. As seen in the fourth column, including these additional fixed effects does not change the message provided by our baseline estimates.

The final two columns show that when we control for the employment rate and the China trade shock, both factors that have been shown to be associated with drug overdose death rates in the past (Hollingsworth et al. 2017; Ruhm, 2019; Pierce and Schott 2020), our point estimates are very similar to our baseline specification that does not control for these factors. This suggests that our results are not being driven by local employment conditions.

Appendix Table E1
Difference-in-Difference Estimates for Drug Death Poisoning Rates
and the Role of Marketing, 1983-2015

Covariates	SSDI only	Non-triplicate only	Both
1990 SSDI rate x			
1996 - 2000	0.23		0.19
	(0.28)		(0.26)
2001 - 2005	2.44		2.12
	(0.53)		(0.50)
2006 - 2010	4.48		4.27
	(0.69)		(0.68)
2011 - 2015	4.92		4.53
	(0.99)		(0.99)
Non-triplicate x			
1996 - 2000		0.66	1.29
		(0.66)	(0.66)
2001 - 2005		3.60	3.50
		(1.31)	(1.46)
2006 - 2010		4.17	3.28
		(1.10)	(1.28)
2011 - 2015		5.61	4.70
		(0.98)	(1.10)
F-test, all SSDI rate interaction coefficients zero	<0.001		<0.001
F-test, all non-triplicate Coefficient interactions zero		<0.001	<0.001

Notes: Standard errors in parentheses are clustered at the state level. All models include year and county fixed effects plus the fraction female, fraction black, fraction other race, fraction Hispanic, and the fraction in age groups 24-34, 35-44, 45-54, 55-64, 65-74, 85-84, and 85 and above. All models weighted by the adult population in the county. There are data for 33 years from 3,106 counties and 102,498 observations.

Appendix Table E2
Difference-in-Difference Estimates for Drug Poisoning Death Rates, 1983-2015

			Add to the model				
	Baseline	No demographic controls	Controls for PDMP Laws	Linear county trends	State x year fixed-effects	Employment Rate	China trade shock
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
1990 SSDI rate x							
1996 - 2000	0.23	0.50	0.22	-0.06	1.03	0.19	0.43
	(0.28)	(0.26)	(0.27)	(0.13)	(0.40)	(0.29)	(0.28)
2001 - 2005	2.44	2.78	2.42	1.92	2.57	2.43	2.67
	(0.53)	(0.58)	(0.53)	(0.36)	(0.42)	(0.58)	(0.53)
2006 - 2010	4.48	4.87	4.43	3.68	4.11	4.51	4.63
	(0.69)	(0.74)	(0.69)	(0.57)	(0.39)	(0.72)	(0.73)
2011 - 2015	4.92	5.40	4.86	3.76	4.58	4.98	5.08
	(0.99)	(1.06)	(0.97)	(0.87)	(0.55)	(1.03)	(1.04)
F-test, all zero	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
Mean, 1990-1994	5.8	5.8	5.8	5.8	5.8	5.8	5.8

Notes: Standard errors in parentheses are clustered at the state level. All models include year and county fixed effects plus the fraction female, fraction black, fraction other race, fraction Hispanic, and the fraction in age groups 24-34, 35-44, 45-54, 55-64, 65-74, 85-84, and 85 and above. All models weighted by the adult population in the county. There are data for 33 years from 3,106 counties and 102,498 observations in total.