Prescription for Disaster: Changing Physician Treatment Patterns and the Drug Crisis

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Abstract

We show that changes in prescribing practices in the mid-1990s played an important role in the seven-fold increase in drug death rates since 1995. Conditional on having an ailment that causes pain, the probability a patient received an opioid prescription was flat until 1995, but rose considerably after that. Using the 1990 Social Security Disability Insurance rate as a proxy for pain in a county, we show that counties with more pain in 1990 had higher fatal overdose rates after 1995, but not before. Without changes in prescribing practices, drug death rates in 2015 would have been 50 percent lower.
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I. Introduction

The US is now in its fourth decade of rising drug poisoning death rates and there is no end in sight. There have been more than 1 million drug poisoning deaths in the United States since 2000 with 111,005 deaths from this cause in 2021 alone. The corresponding death rate, 33.3 per 100,000 people, is 13 times the rate in 1980. Previous work has demonstrated that the run-up in the drug poisoning death in the pre-2010 period was due in large part to abuse from prescription opioids rather than illicit drugs (Cutler and Glaeser, 2021). Scholars have attempted to explain this period with both demand and supply-side hypotheses. The demand-side story suggests that a decline in institutions (Case and Deaton, 2015, 2017, and 2020; Giles et al., 2023) and changing economic fortunes (Pierce and Schott, 2020) may have led some people to take comfort in drug use. While this hypothesis has intuitive appeal given the high rates of drug deaths in low-educated adults and their concentration in places like West Virginia, Southern Ohio, and Western Pennsylvania, others point out that these demand-side stories have a difficult time explaining the magnitude of the change in drug deaths the 21st century (Hollingsworth et al., 2017; Ruhm, 2019; Currie and Schwandt, 2021). Supply side hypotheses focus on the introduction and early marketing of a particular prescription opioid, OxyContin (Alpert et al., 2022; Arteaga and Barone, 2023) as a major contributing factor. 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 explain the growth in deaths that is common in both high and low advertising of areas.

Prescription drugs are not a typical market in that there is an intermediary, a health care provider with prescribing authority, that must authorize a transaction before one can occur. Because of this gatekeeping role, one leading hypothesis for why drug death rates rose is that social norms and regulatory standards in the medical community changed in the 1990s. Past studies have shown that static differences across physicians are related to patients' opioid abuse and related outcomes (Barnett et al., 2017, 2019; Eichmeyer and Zhang, 2022, 2023; Finkelstein et al., 2022; Schnell 2017), but these papers do not explore the role of changes in prescribers' behaviors. In this paper, we estimate and

quantify the role of changes in prescribing practices in the rise of drug death rates from 1980 to the recent past.

We first provide a timeline 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 document opioid prescribing patterns for 18-64 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.

We document how these changes in prescribing practices impacted the current drug crisis as measured by the drug poisoning death rate. We do so by noting that as doctors became more aggressive at treating pain with opioids, the changes should have been more dramatic in local geographic areas that had higher concentrations of individuals with pain. Our hypothesis is outlined with the aid of a simple graph. In Figure 1, we plot the distribution of people with chronic pain levels on the x axis. The dashed (solid) line represents a county with lower (higher) pain levels. In the pre-1996 era, physician practice patterns were such that only people with pain above a "strict" standard would receive an opioid prescription. As social norms about prescribing practices changed, the pain level to receive a prescription declined to a more "lax" standard. This change caused a greater increase in use in the higher pain counties (areas A and B) versus the change in the lower pain counties (area B).

Large-scale health surveys at low levels of geography are limited so, following Cutler and Glaesar (2021), we proxy for a county's stock of people with chronic conditions with the fraction of working-aged people (18-64) that were on Social Security Disability Insurance (SSDI) at the start of 1990. SSDI recipients are an archetypical patient at the center of the drug crisis. As we document

below, 44 percent of them received an opioid prescription in 2016 alone and although this group only represents one in 37 people in the US population, they account for one in six opioid deaths. While those on SSDI have high rates of opioid problems themselves, we provide support to the hypothesis that the SSDI rate could measure pain more broadly by showing a strong positive correlation between pain among non-SSDI recipients and the SSDI rate in a local geographic area.

Using data on the universe of legal prescription opioids from 1997 through 2015, we show that opioids increase more in counties with higher SSDI rates. These changes are not due just to the introduction of new opioid alternatives. In contrast to the literature on OxyContin's introduction which showed no change in shipments of non-oxycodone opioids in non-triplicate states, we observe a large increase in shipments of both non-oxycodone opioids and oxycodone in high SSDI counties compared to low exposure areas. Prior to the changes in prescribing practices that we document, the fraction of working age adults on SSDI is not an indication of drug use: In 1990, it is negatively correlated with the drug poisoning death rate. This correlation increases modestly until around 1999 when it increases dramatically and by 2010, the raw correlation is 0.35.

In event-study models using data from the multiple cause of death data from 1990 through 2015 at the county level, we interact year dummies with the county SSDI rate in 1990, leaving 1996 as the reference. When the drug death rate is the outcome of interest, there is no clear differential pre-1995 trend while the post-1996 dummies become positive, large in value, and statistically significant. To help summarize the estimates and facilitate robustness analyses, we implement difference-in-difference models that allow the 1990 county SSDI rate to impact drug poisoning death rates in five-year intervals (e.g., 1996-2000, 2001-2005, etc.). These models also show a pronounced increase in drug death rates over time in areas with higher 1990 SSDI rates. A simple back of the envelope calculation suggests that if there had been no changes in opioid prescribing practices, there would have been approximately 50 percent fewer drug deaths since the late 1990s. Of course, that calculation should be interpreted

cautiously, but it does suggest that changes in prescribing practices played an important role in the growth of the drug death rate since the mid-1990s.

Given the important role that the introduction of OxyContin played, we show that our results are not simply picking up impacts of pharmaceutical marketing. Using the variation in triplicate laws leveraged by Alpert et al. (2022), we show that both the 1990 SSDI rate and triplicate status in 1995 have independent effects on drug death rates that are quite comparable in magnitude. We then show that the results are not driven by prescription drug monitoring programs, the most common policy that states have implemented to reduce questionable opioid prescribing. Because the SSDI rate can depend on local economic conditions and labor markets, we explore whether those factors are driving our results and do not find any evidence that this is the case.

Our results do not appear to be driven by the type of demand side changes emphasized by Case and Deaton (2015, 2017, 2020). They argue 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. Our event study results show no post-1995 trends for the two major non-drug components of the deaths of despair: alcohol-related diseases and non-drug suicides. In the difference-in-difference models, there is no statistically significant post-1995 effect for alcohol death rates. There is a small but statistically significant effect in the 2011-2015 period in the models with non-drug suicide rates as the outcome, but this result turns statistically insignificant when we include state by year effects and is small in magnitude when compared to our main results on drug overdose death rates.

Our paper contributes to the broader literature on the causes and consequences of the drug crisis. Case and Deaton (2015, 2017, and 2020) have argued that a decline in institutions have encouraged deaths of despair, especially among lower-educated groups and there is some quasi-experimental evidence consistent with this hypothesis (Pierce and Schott, 2020; Giles et al., 2023). Hollingsworth et al. (2017), Ruhm (2019), and Currie and Schwandt (2021) argue against economic

factors as being the major drivers of the drug crisis. We 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 as two other components of deaths of despair, alcohol and non-drug suicides, do not seem to be impacted to nearly the same extent as drug deaths. The burgeoning literature that is beginning to study the impacts of the opioid epidemic on labor markets, families (Buckles et al., 2023), crime (Meinhofer, 2016; Wen et al., 2017; Bondurant et al., 2018; Dave et al., 2021; Smart and Reuter, 2022), and numerous other downstream outcomes has often relied on state-level sources of variation such as triplicate laws, must-access prescription drug monitoring programs, Medicaid law changes, or the changing supply of substance abuse treatment. While these sources of variation can be useful, our source of variation in susceptibility to the severity of the opioid problem, 1990 SSDI rates, varies at the county level and could allow researchers to the impacts of opioids within a given state. Of course, the validity of any non-experimental source of variation must be evaluated for the particular question being answered, but the 1990 SSDI rate is potentially valuable as a source of variation for many analyses.

Our work also contributes to the growing literature on the role that prescribers have played in drug overdose death rates. Barnett et al. (2017, 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 et al. (2022) show that moving from a low to high prescribing area greatly increases the chance of opioid abuse for those on federal disability roles. Schnell (2017) shows that the drug poisoning death rate is higher in areas with more opioids prescriptions per capita. Currie and Schnell (2018) examine how fixed differences in a physician's education relate to their opioid prescribing. While these papers show the critical role that static differences in prescribing practices across physicians play, they do not provide evidence on the role that changing prescribing practices had in shaping the epidemic, which is what this paper adds to the discussion.

II. Reevaluating the Role of Opioids in Treating Chronic Pain

A. Timeline of Events

Historically, opioids were reserved for those with acute pain such as post-surgical and cancer patients. Given this more limited use of opioids, 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.[1] Despite this, many were either not treated with prescription pain relievers or are 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 Marks and Sachar's 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 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, Porteney

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

(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% would discourage the practice of prescribing opioids for non-cancer pain or investigate it as a violation of law (Joranson et al., 1992).

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 American Pain Society (APS) and the American Academy of Pain Medicine (AAPM) (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 began to get some 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."[2]

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 (HCAHPS) among Medicare patients since 2006. Three questions on the survey ask if the patient's pain was adequately controlled during their hospital stay.

B. 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

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

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 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 computer code from Sherry et al. (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.[3,4]

In Figure 2, 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.[5] We exclude anyone with cancer

[3] 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.
[4] The number of prescriptions reported on NAMCS was either 5 (1985-1994), 6 (1985-2002), or 8 (1980-81, 2003-2015), depending on the year. To match data over time, we use the first 5 or 6 prescriptions reported in years. The results from NAMCS look very similar if we restrict the sample to the first 5 prescriptions in all years or take any prescriptions in all years. This is reported in Appendix Figures A1A and A1B.
[5] 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.

and anyone with co-occurring chronic and acute pain. The latter restriction only applies to 2.6 percent of the data.

In Figure 2A, 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).[6] 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 percent in 2015, an 82 percent increase. The results for acute pain (Figure 2B) 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 which 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 A2 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

[6] A regression of the rate on a time trend produces a coefficient (standard error) of -0.00048 (0.00051).

inclusion of the age dummies. This suggests that the trends we are seeing are not simply capturing the aging of the population.

While the 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. One important category of changes by patients are those that are a reaction to changes by prescribers. This type of patient change could be considered to be part of the equilibrium impacts of changing prescribing practices and so consistent with the general hypothesis in this paper.

An alternative that does pose a potential problem is if physicians did not change their behavior at all, but patients began asking for opioid medications. 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 would have expected to see similar increases in the use of opioids for both acute and chronic 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 3 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.[7] 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.

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

[7] 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.

The results in the previous section indicate that the prescribing practices of physicians changed dramatically after 1995 with providers much more likely to prescribe an opioid for individuals with any type of pain, but especially for those with chronic pain. Because a patient that exogenously receives an opioid prescription has an increased probability of long-term use and abuse (Barnett et al., 2017, 2019; Eichmeyer and Zhang, 2022, 2023), the change in prescribing likely led to increased drug-related mortality and could be responsible for an important portion of the increase in drug deaths that has occurred over the past 30 years.

To study the likely consequences of physicians' changing behavior on mortality rates, the ideal data would be a representative panel of individuals with data that includes pain conditions, prescription drug use, and mortality, all over a long time period. This ideal is not available. Instead, we note that the change in physician prescribing behavior will likely have had larger per-capita effects in areas where there is a higher rate of pain than in areas with a lower rate of pain. As a consequence, we need to identify geographic areas that are likely to have more or less pain.

We approximate rates of pain in a geographic area by using the fraction of working-age adults (18-64) who are on Social Security Disability Insurance (SSDI), as first suggested by Cutler and Glaeser (2021). SSDI recipients are those adults that have worked for a qualifying period in jobs covered by Social Security and have a severe medical condition that limits their ability to engage in substantial gainful employment.

SSDI recipients have a great deal of chronic and acute pain. In the 2011-2015 National Health Interview Survey (NHIS), some sampled adults answered a disability supplement and one question asked whether over the past three months, respondents experienced pain on no, some, most or all days. Using the ipums.org versions of the NHIS (Blewett, et al., 2022), among adults aged 18-64, the fraction with pain on most or all days among SSDI recipients[8] was 56.3 percent but only 15.3 percent among

[8] The NHIS does not ask whether a person is on SSDI but we can approximate this by whether a person 18-64 is on Medicare.

non-SSDI recipients for a difference (standard error) of 40 (0.8) percentage points. This difference (standard error) only falls to 36.1 (0.8) when we control for a full set of age effects.

Not surprisingly, SSDI recipients have been at the center of the opioid crisis as well. Morden et al. (2014) estimate that 44 percent of SSDI recipients received an opioid prescription in 2011 and 23 percent were chronic users of these prescriptions. Although SSDI recipients represented 2.7 percent of the total population in 2016, they accounted for 15.7 percent of opioid poisonings in that year.[9]

We calculate what we call the SSDI rate in 1990 and use that as a proxy for aggregate pain in the local population. We focus on 1990 because it pre-dates the push to liberalize opioid prescribing described in Section IIA and we calculate this at the county level as this is the lowest level of aggregation we have for mortality data. Specifically, by county, we take the ratio of SSDI recipients to hundreds of adults between the ages of 18 and 64. We obtain the numerator from the Social Security Administration (1990) and Moore (2020). This data reports SSDI recipients as of December 1989 so we use the 18-64 year old population from the National Cancer Institute Surveillance, Epidemiology, and End Results (SEER) program for 1990.[10] In December of 1989 there were 2.9 million adults on SSDI, or a rate of 1.8 per 100 adults aged 18-64. The interquartile range of this value across counties goes from 1.6 to 2.8 with a median of 2.1 and a mean of 2.3.

We now provide evidence that the SSDI rate proxies for pain in a geographic area even among those not on SSDI. First using the NHIS data introduced above, we show that geographic areas with higher rates of SSDI have higher incidence of pain. Between 1997 and 2003, the NHIS asked a set of four questions related to pain: did the respondent have pain in the neck, lower back, face, or in joints. We create an indicator for whether the individual responded yes to any of the four pain questions and

[9] Kou et al. (2019) estimate that the drug death rate among SSDI recipients was 77.6/100,000 by 2016. Applying this rate to the 8.809 million recipients in December of that year (https://www.ssa.gov/policy/docs/statcomps/oasdi_sc/2016/oasdi_sc16.pdf), this implies roughly 6,836 opioid-related deaths to the SSDI population, which is 15.7 percent of the opioid deaths that year.
[10] https://seer.cancer.gov/popdata/

aggregate across years for 18-64 year-olds who were not on SSDI. Over this time, the NHIS sampled data in 678 distinct geographic areas that are identified by a consistent PSU x strata variable that allows researchers to identify that respondents in different periods were from the same geographic area, but we do not know what that area is. In Figure 4A, we present a bubble plot which shows the relationship between each geographic area's average SSDI rate from 1997-2003 against the percent of individuals who have pain but who are not on SSDI. The size of the bubble is determined by the sum of the weights for individuals in the same PSU x strata over time. There is a positive association with a correlation coefficient of 0.261. Since we are measuring pain for individuals not on SSDI, the positive relationship suggests that geographic areas with higher SSDI rates also have higher rates of pain in the non-SSDI population. Ideally, we would be able to measure the correlation between SSDI rates and pain among the non-SSDI population in a time period prior to 1995. While we have not found data that allow us to make that comparison, we can restrict ourselves to the 1997 NHIS data to minimize any relationship between pain and SSDI rates that is due to the increase in opioid prescribing. When we restrict the data to 1997, the correlation between the SSDI rate and the percent with any of the four pain conditions among the non-SSDI population is still positive, 0.158. In Appendix B, we provide additional evidence on the relationship between SSDI rates and pain. In particular, we show that there is a positive correlation between the SSDI rate and each of the four pain variables as well as that eliminating some high SSDI rate counties (which could be viewed as outliers) does not alter the positive association between SSDI rates and pain among those not on SSDI.

In Figure 4B, we run a similar analysis using the 2011-2015 NHIS that asked about having pain on most/all days in the past three months. This correlation coefficient is 0.422 across the 600 geographic areas in the NHIS over this time period.

One concern with using pain as our cross-sectional measure of exposure to the changes in prescribing practices is that places with more pain may have already had other methods of coping with that pain, such as illegal drugs, alcohol abuse, or in the extreme, suicide. This type of concern is in the

spirit of the work of Case and Deaton (2015, 2017, 2020) who argue that a decline in institutions has led to deaths of despair. In this case, if demand for pain relief were rising over time (rather than changes in prescribing practices being the main driver), we might inadvertently interpret the effects of increases in demand for drugs as due to the changing behavior of prescribers. To explore this possibility, we use restricted-use versions of the National Vital Statistics System (NVSS) Multiple Cause of Death (MCOD) mortality files to calculate county-level mortality rates (deaths per 100,000) from various causes. The MCOD data is a census of deaths in the US and contains an underlying cause of death and any multiple causes of death. The restricted-use versions provide the county of residence for the deceased. In Appendices C and D, we provide more information about the mortality data and population data that we use to calculate death rates from various causes.

We begin with Figure 5A, the correlation between the SSDI rate in 1990 and the drug poisoning death rate in 1990. Interestingly, the correlation is small and negative which suggests that prior to the changes in prescribing behavior, SSDI rates and drug poisoning death rates were not closely related. Figures 5B and 5C show some positive correlation between SSDI rates and alcohol (0.13) and non-drug suicide rates (0.24). To take this a step further, Figure 6 plots the correlations between 1990 SSDI rates over time with the three categories of death rates from Figure 5. While the correlation of 1990 SSDI rates with non-drug suicide death rates and alcohol death rates vary slightly from 1990 through 2015, the correlations are relatively stable throughout. The same is true for a more general measure, all non-drug death rates. 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. These series suggest that a demand side story in isolation cannot be driving the increased drug death rates over time since the correlations between the SSDI rate and these other measures of deaths of despair are not also increasing dramatically.

As a check on our measure of aggregate pain, we explore whether counties with higher SSDI rates in 1990 had larger subsequent flows of opioids to the county. We use data from the Drug Enforcement Administration's (DEA) Automation of Reports and Consolidated Orders System (ARCOS). Within this system, drug manufacturers and distributers must report to the DEA all controlled substance transactions from manufacturers to points of sale or distribution.[11] Many of the drugs tracked in the ARCOS system are opioids and we use data for nine opioids.[12] The data measures grams of shipments by quarter to a three-digit zip code. We convert these data to morphine equivalent grams (MEG) at the county level and divide by population.[13] In Figure 7, we report the aggregate MEGs per 100,000 people by quarter for the top and bottom quartile of the SSDI rate from the first quarter of 1997 to the last quarter of 2015 for nine opioids. Unfortunately, ARCOS data prior to 1997 are not available. The figure illustrates that per capita shipments of opioids are very similar across counties at the start of the sample and that both county groups grow, but the growth is much more substantial in the top quartile.

IV. The Initial Stock of People in Pain and the Growth in Drug Poisoning Death Rate

A. Main Results

In this section, we provide evidence that the initial stock of people in pain, as proxied by the 1990 SSDI rate in the county, is predictive of the time path of the drug crisis. We use the mortality data from 1983 through 2015. We begin in 1983 because that is the first year that the data has both a census of deaths for all states and the file reports FIPS county codes available in our other datasets. We end the data in 2015 as during this period fentanyl becomes the leading cause of most drug poisonings. An

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

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

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

argument can be made that we should end the series in 2012 before fentanyl has much of a presence in drug markets. Causes of death are defined by the ICD-10 classification system starting in 1999 and the ICD-9 system beforehand. We use data for counties or county groups that we can define consistently over time and in Appendix D, we outline how we merge certain counties to make the SEER and MCOD data compatible over time. In our sample, there are 33 years of data for 3,106 counties leading to 102,498 observations.

We begin with plots of raw data as well as event-study regression specifications. For the latter, the dependent variables will be a death rate (y) that varies across counties (c) over a 26-year period (t). We will control for basic demographics ($\underline{x_{ct}}$) including the age distribution of the population, the fraction Black, the fraction Hispanic, fraction women, as well as county and year fixed effects (μ_c and λ_t, respectively). To capture the changing prescribing practices of physicians, we interact the 1990 SSDI rate at the county level with year dummies. The specification requires that one of the years be normalized to zero. As it is not clear when "treatment" begins in this context, we restrict the 1995 coefficient to be zero which is the year when Campbell proposes pain as the 5th vital sign and a year before the APS begins the formal program to encourage its adoption. More formally, our specification is

$$(1) \qquad y_{ct} = x_{ct}\beta + \sum_{\substack{i=1990 \\ i\neq 1995}}^{2015} \theta_i (SSDI\ Rate_{c1990}) 1(year = i) + \mu_c + \lambda_t + \varepsilon_{ct}$$

where ε_{ct} is a random error. We cluster the standard errors by state and weight observations by the adult population in the county and year.

Beginning with the drug poisoning death rate, Figure 8A displays the raw data for adults 18 and above from 1983 through 2015 for two groups: counties in the top and bottom quartile 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 but the difference between counties does not widen in the pre-1996 period. This result is consistent with the negative scatter plot we saw in Figure 5A. However, the

numbers change dramatically in the 1996 period and after. Deaths in the lowest quartile of SSID 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.

Turning to the event study specification, Figure 8B 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 first declines and then begins to increase again. The decline in 2010 aligns with the reformulation of OxyContin (Alpert et al., 2018; Evans et al., 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 at the 95 percent level starting in 2000 with the 1999 coefficient just at the 90 percent level..

These are dramatic increases. To provide some perspective, 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 8A, 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 had there had been no behavioral changes by prescribers, as captured by our post-1996 interaction coefficients. In that case, the estimates suggest that there would have been roughly half the realized drug poisoning deaths had there been no liberalization of opioid prescribing.

Figures 8C and 8D show analogous results for opioid poisoning death rates (including heroin). The numbers for opioid deaths in Figure 8D show a similar pattern to those for all drugs just with smaller impacts. There is no large differential pre-1996 trend in coefficients and the rise in coefficients is particularly dramatic starting around 1999. Comparing the basic time series in Figure 8A and 8C, roughly 70 percent of the rise in drug deaths were due to opioids. It is then no surprise that the ratio of the interaction terms by year in Figures 8D and 8B from 2000 on is, on average, 0.67.

As noted above, the socioeconomic characteristics of SSDI recipients tend to be lower than non-recipients. This in turn could mean that the 1990 county SSDI rate indicates a high rate of despair and the growth of death rates in higher SSDI rate counties could be demand driven rather than supply driven. There is some evidence of this concern in Figures 8E and 8G which showed that areas with higher 1990 SSDI rates had higher rates of alcohol disease death rates and higher rates of non-drug suicides in the pre-1996 period. However, there appears to be little growth in these forms of deaths of despair after 1995. In Figures 9F and 9H, we report the event study results for alcohol disease and non-drug suicide death rates, respectively. The means are shown at the top of the figures and they indicate that these death categories had substantially larger mean values from 1990 to 1995 period than did drug deaths. We've kept the vertical axes on the same scale for all four event-studies in Figure 8 to make it easier to compare the event studies across outcomes. In the pre-treatment period there is one statistically significant coefficient for alcohol (1986), and only two statistically significant coefficients in the post-1995 period (1996 and 1998). For non-drug suicides, there is little trend in the pre-1996 coefficients, but in the post-1995 period, there are statistically significant at the 95 percent level in years 2000, 2001, and 2015. The coefficient in 2015 is however one seventh the size of the coefficient for the same year in the drug poisoning death rate equation.

B. Difference-in-Differences Specification and Robustness

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 1 presents results from estimating Equation (2) where our dependent variable is the drug poisoning death rate. The first column presents results from the baseline specification. 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; Buchmueller and Carey, 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 next column of Table 1, including these additional fixed effects does not change the message provided by our baseline estimates.

The SSDI Rate in 1990 at the county level is correlated with any number of socio-economic and health variables. One might be concerned that this metric is simply capturing some other underlying

economic condition and not fulfilling its intended purpose as a proxy for pain. In Appendix Figure A3, we re-estimate our basic event-study models and swap out the SSDI rate in 1990 for two omnibus measures of economic activity available at the county level from the 1990 Census. First, in panel A, we use the poverty counts per 100 people (sample mean of 13.0) in just this fashion. Although the correlation coefficient between poverty/100 and the SSDI rate per 100 across counties is 0.48, in that graph there are no statistically significant coefficients of the interactions between county poverty and the year effects for any year. In panel B, we use the number of adult males aged 16-64 that are not employed per 100 (mean=20.9). The correlation between this variable and the SSDI rate across counties is 0.56.

As mentioned previously, past work (Hollingsworth et al., 2017; Ruhm, 2019) has found that economic conditions explain only a small part of the rise in drug deaths since the 1990s. In our context, local economic conditions could be very important because they might affect not only our outcome variables, but also the local SSDI rate. Although SSDI eligibility is set by federal rules, opportunities in local labor markets affect whether an individual is able to be substantially gainfully employed and thereby, SSDI rates. As a first attempt at addressing this possibility, we add controls for the county employment rate (constructed from the Bureau of Economic Analysis's employment data). The point estimates again are very similar to our baseline estimates, suggesting that the part of the SSDI rate in 1990 that varies with drug death rates is not especially correlated with local economic conditions. As another way to get at local economic conditions, we include controls for the trade shock exploited in Pierce and Schott (2020). In that paper, the authors found that counties that were more exposed to the normalization of trade relations with China saw increases in drug deaths. Because counties that were most exposed to the trade shock might also have higher SSDI rates, our results could be capturing the effect they estimate rather than changes in prescribing practices. However, as seen in Table 1, including the measure of exposure to the trade shock (interacted with year effects) has almost no impact on our point estimates, and if anything, increases them slightly.

C. Changes in Prescribing Practices or Pharmaceutical Marketing?

It could be the case that the rise in the disparity 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 pad states felt that they would use OxyContin sparingly.[14] 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 ARCOS 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. These findings were used to argue that the differences reflected the impacts of OxyContin's

[14] 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.

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. In Figure 9, we plot the differences and 95-percent confidence intervals between top and bottom quartile counties for oxycodone and for all opioids minus oxycodone. Not surprisingly, there is a large increase in oxycodone over time in high 1990 SSDI rate counties relative to low 1990 SSDI rate counties. However, in contrast to past work on the importance of opioid marketing, there is just as large of a differential increase in the other opioids in our sample. This likely means that the cross-sectional variation in 1990 SSDI rates we are using is not simply capturing something about the introduction and marketing of OxyContin.

To further explore this possibility, we estimate our models, but include controls for the variation in advertising used by Alpert et al. (2022). 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. The first column of Table 2 repeats our baseline difference-in-difference regression estimates from Table 1. To confirm the results from Alpert et al. (2022) in our sample, we estimate our baseline regression with two changes: 1) we omit the 1990 SSDI rate by year-group interactions and 2) we include interactions between an indicator for whether the state is a non-triplicate state (and so would have received more marketing) and the year group indicators. We report the results in the second column of Table 2 and see that, consistent with past work, non-triplicate states saw differential growth in drug poisoning death rates after 1995. 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 2, both sets of interactions continue to be important determinants of drug poisoning death rates. In Appendix Figure A4, we report the analogous event-

study figures. In Figure A4A, 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 A4B, we repeat the exercise for the Non-triplicate state x year interactions by year by themselves (black lines) and after controlling for the SSDI rate x year effects (grey lines). The results in both models tell the same story but coefficients after controlling for the SSDI rate are about a quarter smaller. These results strongly suggest that changes in prescribing practices and marketing both played major roles in the rise of drug poisoning death rates.

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[15] 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.

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

Given this evidence of persistence, we then identify counties with high and low advertising in the Open Payments data. Specifically, 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 and the dollar amount transferred to providers per 1,000, 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 A5, we graph the drug death rate for three sets of counties – those in the top decile of open payments per 1,000, those in the 5th decile, and the lowest 20 percent (which has no advertising). In all three county groups, between 1996 and 2015, drug poisoning death rates increased by almost 400 percent. 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. We believe that event is the changing prescribing practices of physicians.

D. An Alternative Proxy for Aggregate Pain: Work Limitations

As an alternative proxy for the amount of pain in a county, we consider the share of working-age adults with a condition that limits their ability to work. In the 1990 Census, long-form respondents aged 16 and above were asked whether a physical, mental, or other health condition limits the kind and amount of work they can do. Using data from the National Historical Geographic Information System (NHGIS) (Manson et al., 2022), we construct the share of individuals aged 16-64 that answered yes to this question at the county level and call this the share with a work limitation. The share with a work limitation is a much broader measure than the SSDI rate in that the population-weighted average across counties is more than than four times the SSDI rate. As such, it is not as extreme of a measure and may better capture pain in the local population.

In Appendix F, we show that individuals with a work limitation have substantially higher rates of persistent pain than those without limitations. The share of adults with a work limitation in 1990 and the SSDI rate in the same year are highly correlated (Appendix Figure F1). Next, we reproduce much of the same analysis from above replacing the SSDI rate in 1990 with the share with a work limitation. In the 2011-2015 NHIS, the share with a work limitation is predictive of pain at the local level even for those without a work limitation (Appendix Figure F2). The annual correlation between this measure of aggregate pain with drug death rates at the county level shows the same pattern in that during the pre-1996 period these variables are weakly correlated, but the correlation grows substantially in the post-1995 period (Appendix Figure F3). The times series of drug death rates in the top and bottom quartile of the share work limitation are similar in the pre-1996 period but the death rates balloon in the top quartile counties after 1995 (Appendix Figure F4A). In event study graphs (Appendix Figure F4B) where we replace the year dummy x SSDI rate interactions with year x share work limitation, we get the exact same time patterns as in the graph above with no pre-1996 trends but pronounced effects in the post-1995 period. Replicating the basic difference-in-difference effects with the share work limitation (Appendix Table F1) produces very similar results to those we obtained using the SSDI rate. Similarly, our analysis of the separate role of marketing and work limitations produces results very similar to those we found when using the SSDI rate to proxy for pain (Appendix Table F2).

E. The Contract with America and the Termination of SSDI Benefits for Drug Addiction and Alcoholism

In 1996, Congress passed the "Contract with America" (PL 104-121) which contained a provision that terminated disability benefits in both the SSDI and Supplemental Security Income (SSI) programs for beneficiaries whose primary impairment was drug addiction or alcoholism (DA&A). The restriction went into effect on January 1, 1997. Moore (2015) estimates that the law change initially moved 100,000 SSDI recipients off the rolls, but about 90 percent applied for SSDI under a different

classification and about half returned to the DI rolls. Waid and Barber (2001) note that the law initially impacted 120,000 SSI recipients of which 86,000 eventually lost eligibility.

It could be that the law change affects our estimated relationship between the 1990 county SSDI rate and the drug poisoning death rate, though there are a few reasons why the law change should have a minor, if any, impact on our estimates. First, the number of impacted people is relatively small, amounting to about 130,000 people in both the SSI and SSDI programs. In 1995, the SSDI program alone provided benefits to more than 4 million individuals. Second, those suffering from alcoholism were the largest group impacted by the law change. Moore (2015) notes that 58 percent of those impacted were qualified because of alcoholism, 27 percent for both drug abuse and alcoholism, and only 15 percent were suffering from drug addiction alone. If the law change was impacting substance abuse, one would expect the alcohol death rate to be impacted more than the drug poisoning death rate. The raw correlations in Figure 6 and the event study results in Figure 8F suggest this is not occurring. Third, existing research demonstrates that these law changes had little impact on drug-related outcomes such as arrests (Orwin et al., 2004; Chatterji and Meara, 2010) or health care use such as emergency department visits and hospitalizations (Chatterji and Meara, 2010). In contrast, Orwin et al. (2004), Chatterji and Meara (2010), and Moore (2015) all show that impacted recipients had substantial increases in employment.

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've added some empirical content to an oft-suggested explanation for these trends – the changing prescribing practices of physicians. Concern about an epidemic of untreated pain encouraged some providers to argue for more aggressive use of opioids, especially for individuals with chronic pain. These calls for actions started in earnest in the late 1990s

and our analysis suggests they had measurable effects. Conditional on a diagnosis that a person has a condition likely to produce acute or chronic pain, the chance a patient received an opioid prescription increased dramatically starting after 1995, consistent with the timing of the current drug crisis. Using the 1990 SSDI rate as a proxy for pain in a geographic area, we find that counties with more pain experience considerably more growth in subsequent drug poisoning death rates. This analysis helps tie the changes in prescribing practices to drug death rates: opioid prescribing and drug deaths rose more in counties that likely had more pain in the early 1990s.

Our analysis helps quantify the role of prescribing changes in the rising drug deaths. 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 roughly half of what was observed. If we attribute these findings to changes in prescribing habits, that would suggest that liberalizing the use of opioids to treat pain can account for roughly 50 percent of the increase in drug mortality since 1995.

Just as doctors were at the heart of the epidemic, they can also be part of the solution. Dun et al. (2022) and Zhang (2023) study recent interventions that are meant to reduce opioid prescribing and find that prescribing does change in response to the stimulus.

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.[16]

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Figure 1
The Distribution of Pain in Two Hypothetical Counties

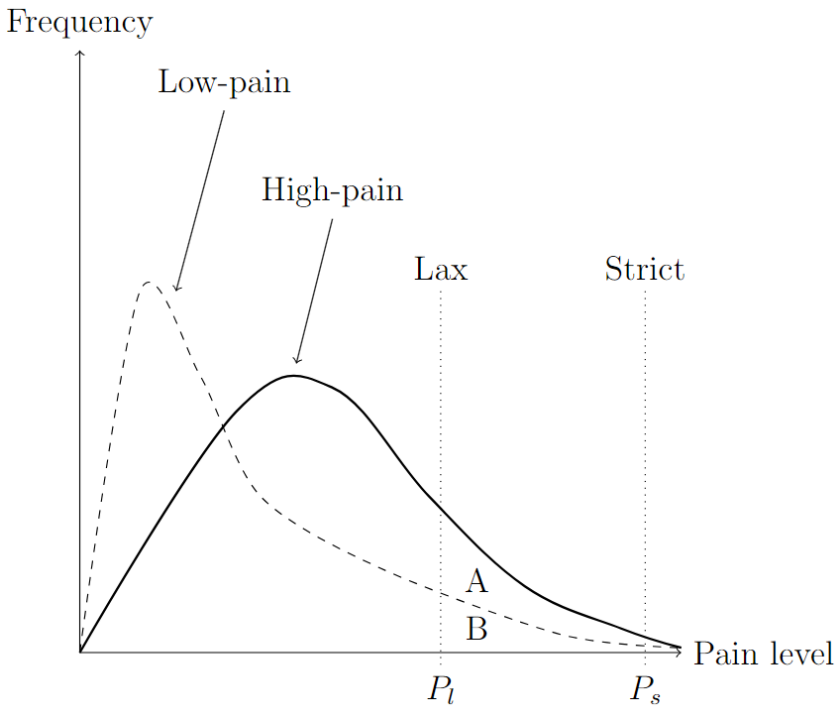


Figure 2
Time Series of the Percent Adults 18-64 Visiting a Physician in the NAMCS Data with Acute or Chronic Pain and Percent with Pain Receiving an Opioid Prescription

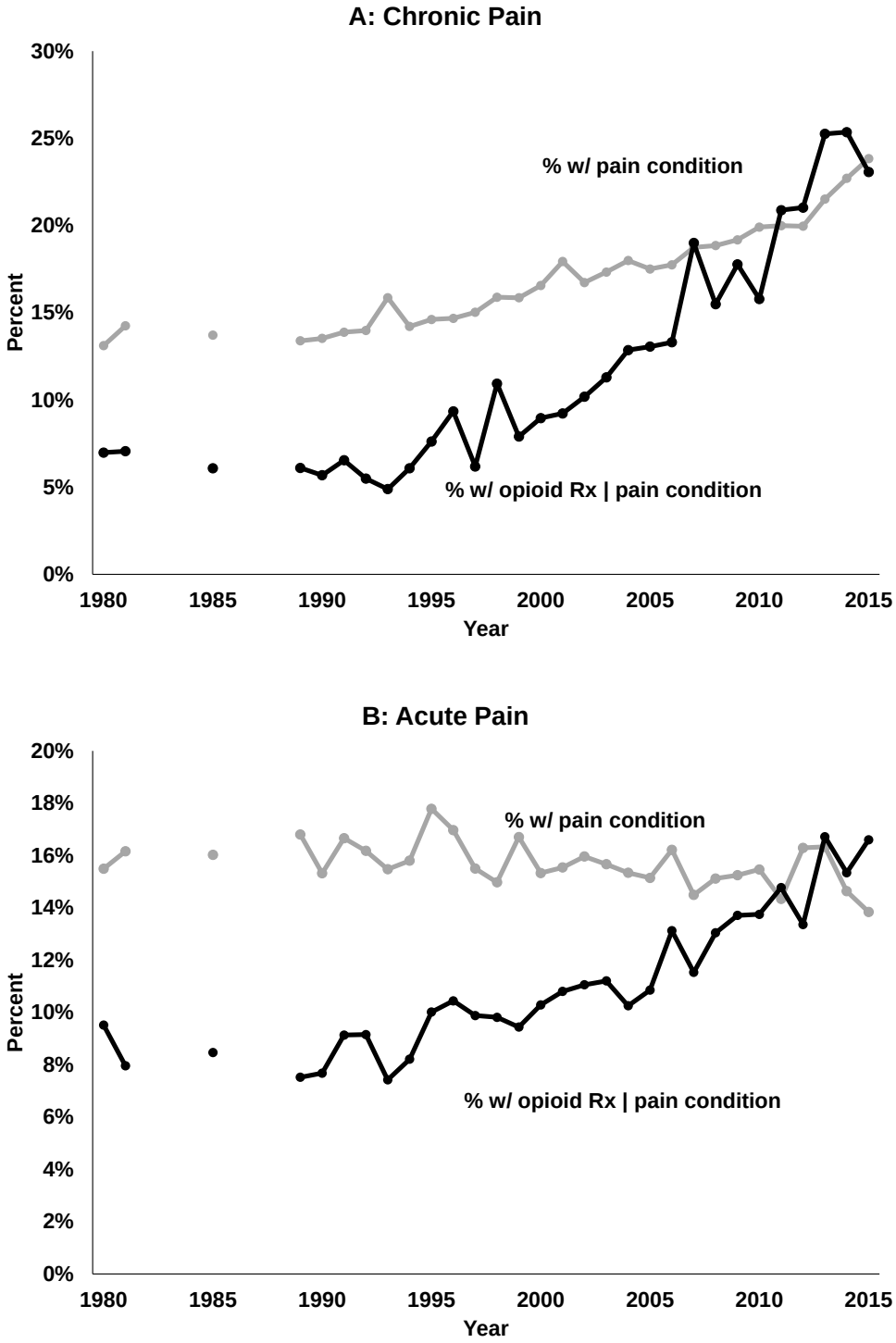


Figure 3
Medication Seeking Behavior by Patients in the NAMCS, 1980 - 2015

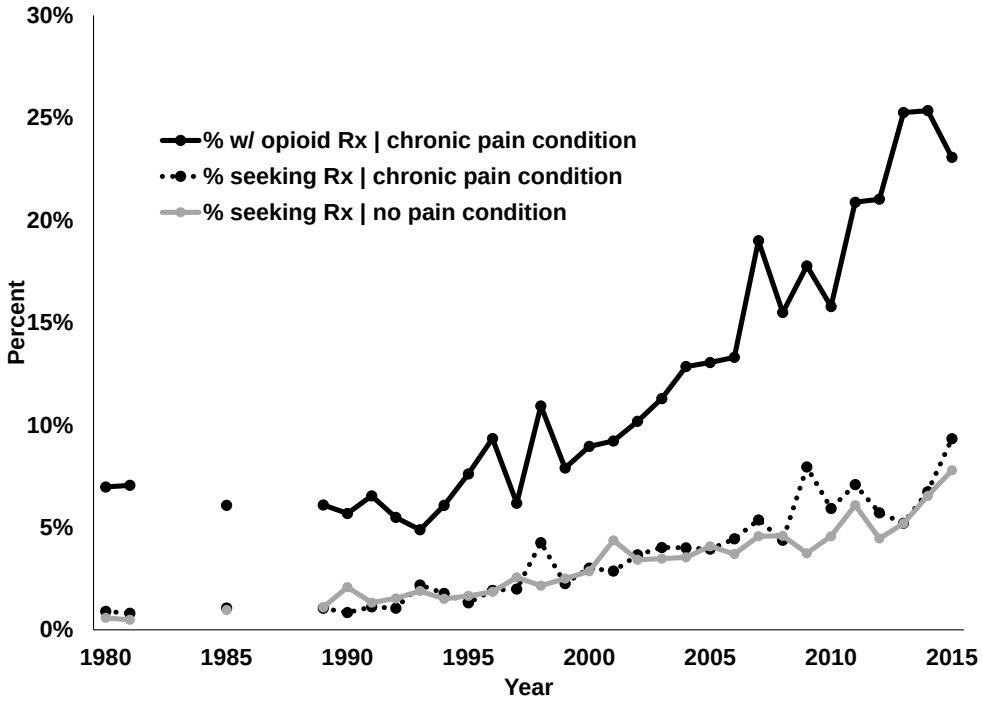


Figure 4
Relationship Between Pain for the non-SSDI Population and the SSDI Rate in the NHIS

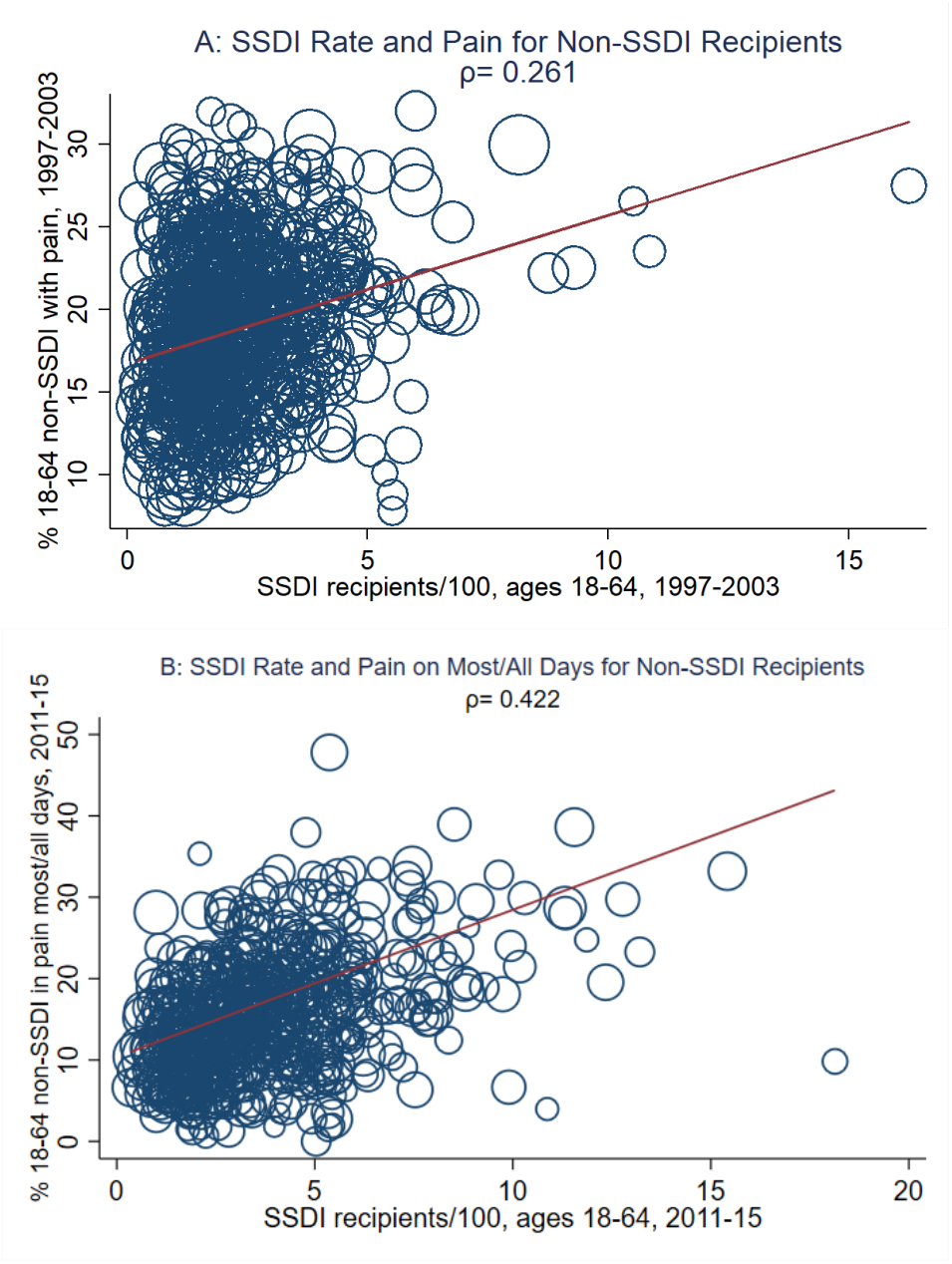


Figure 5
Bubble Plot, Various County-Level Death Rates in 1990 with the 1990 County SSDI Rate

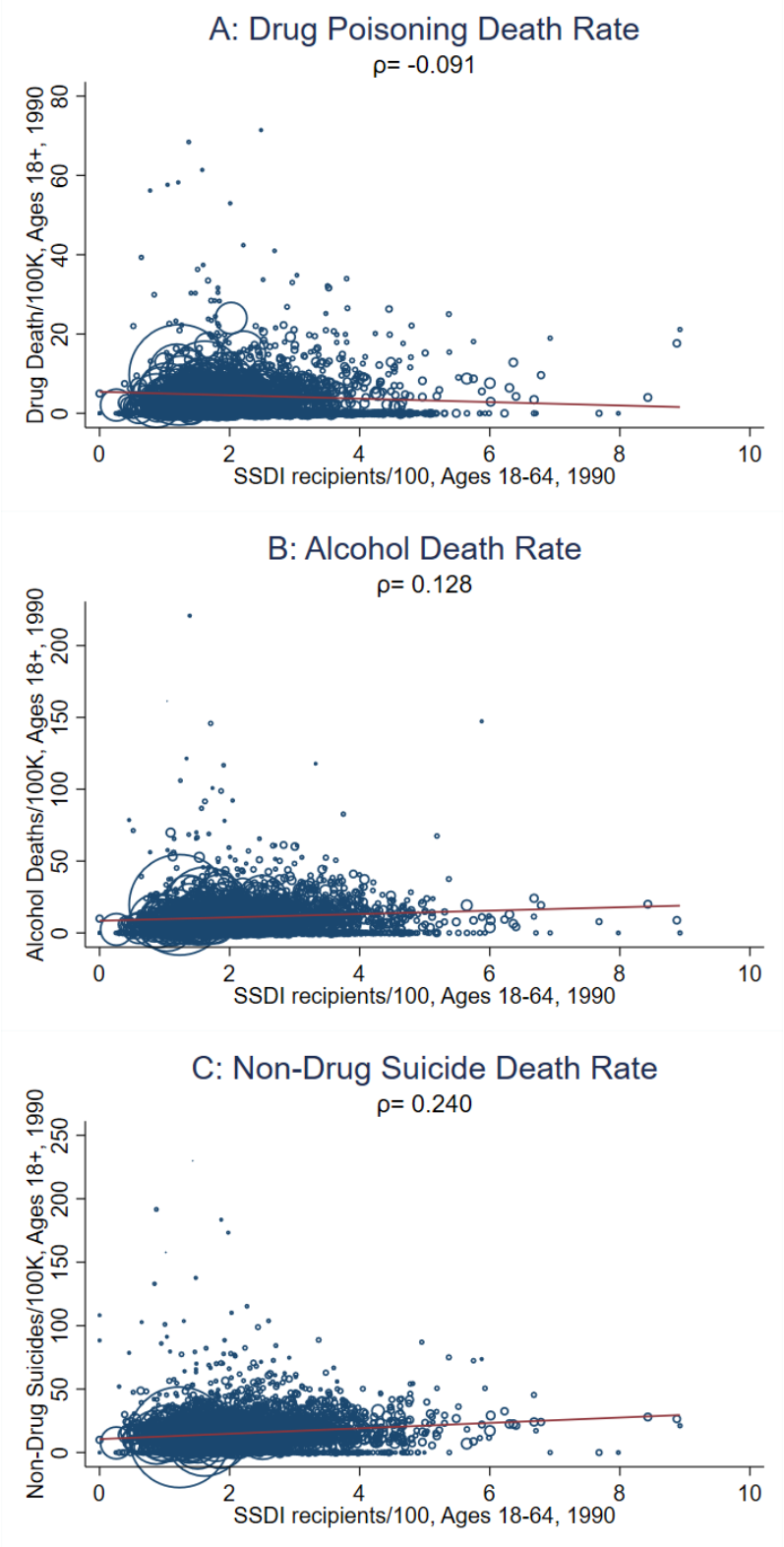


Figure 6
Correlation Coefficient Between 1990 County SSDI Rate and
Adult County Death Rate for Various Years, 1990-2015

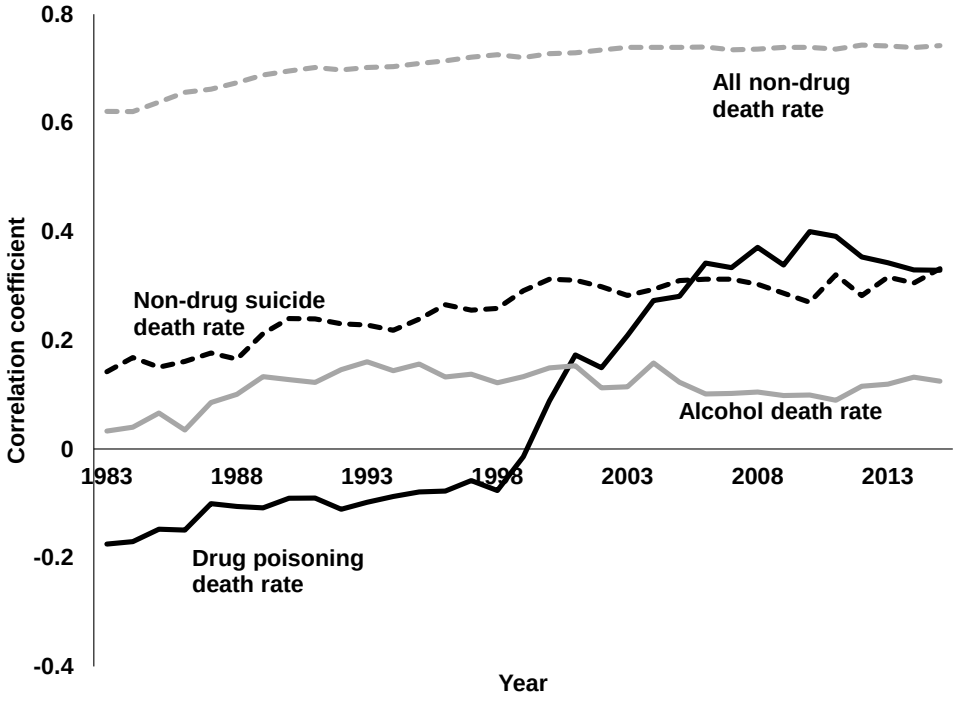


Figure 7
Time Series of Quarterly Morphine Equivalent Grams (MEG) per 100,000 Population for all Opioids
by Quartile of County SSDI Rate in 1990, ARCOS Data, 1997.1 – 2015.4

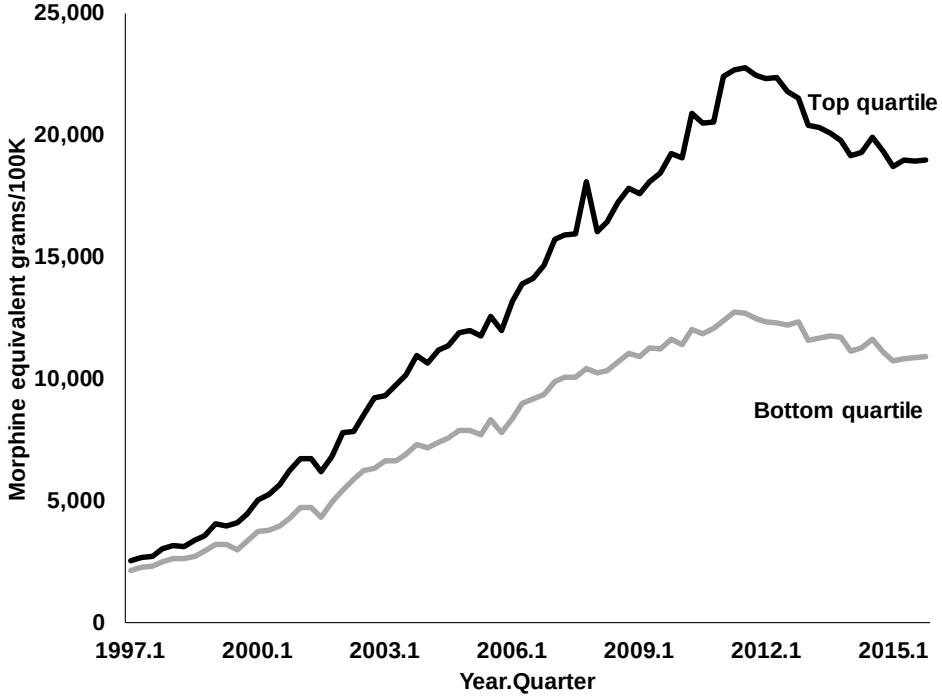


Figure 8
Death Rates for Adults Ages 18+ by Quartile of 1990 County SSDI Rate and Event Study Results, Death Rate at the County Level, 1990-2015, Parameter Estimates and 95% Confidence Intervals

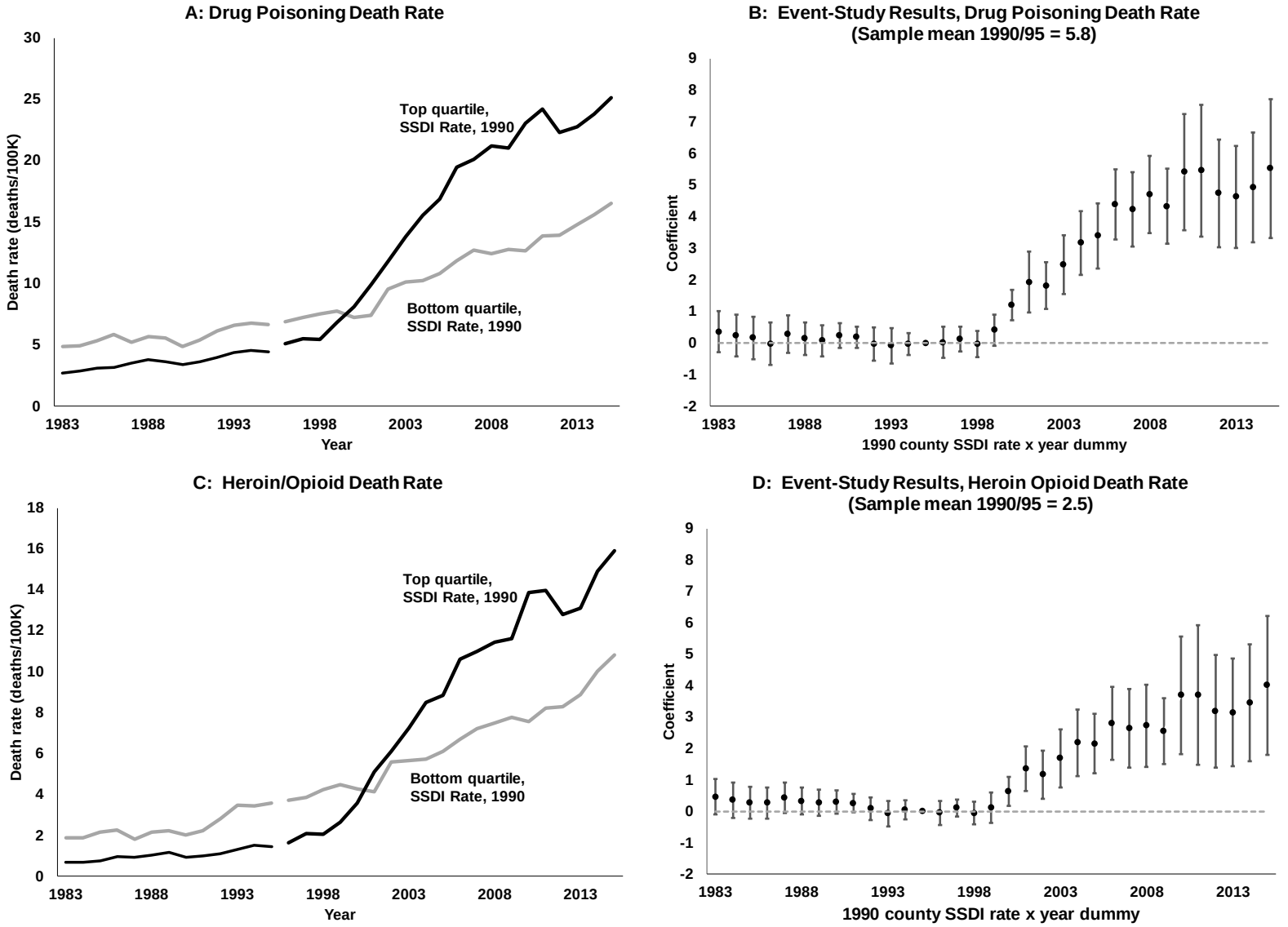
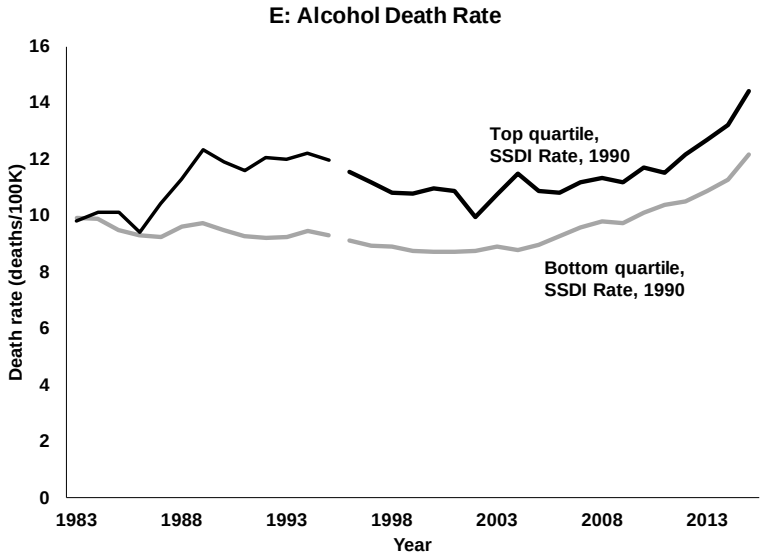
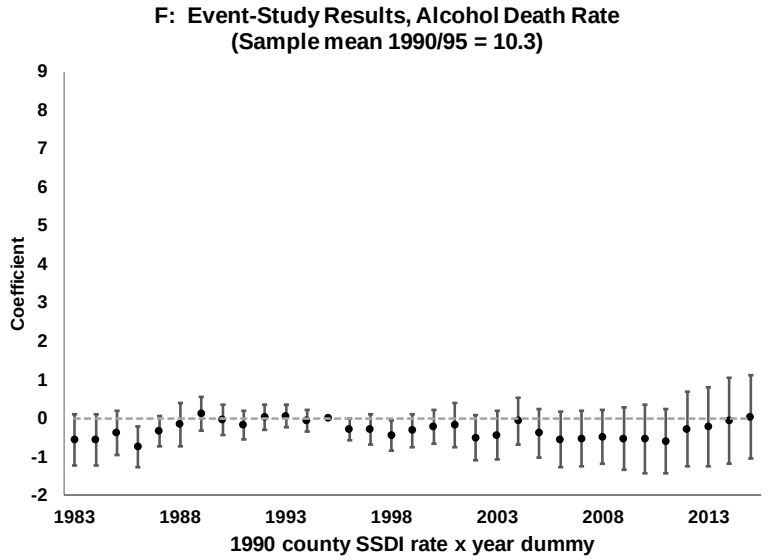
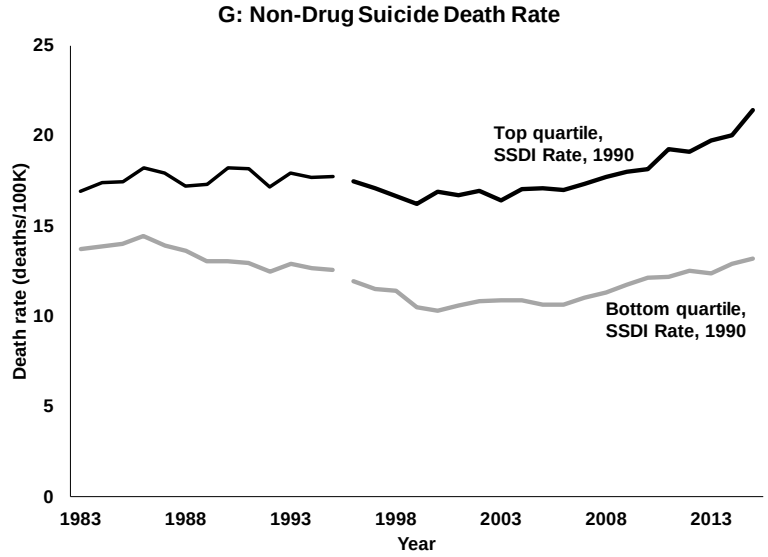


Figure 8 (continued)
Death Rates for Adults Ages 18+ by Quartile of 1990 County SSDI Rate and Event Study Results, Death Rate at the County Level, 1983-2015, Parameter Estimates and 95% Confidence Intervals







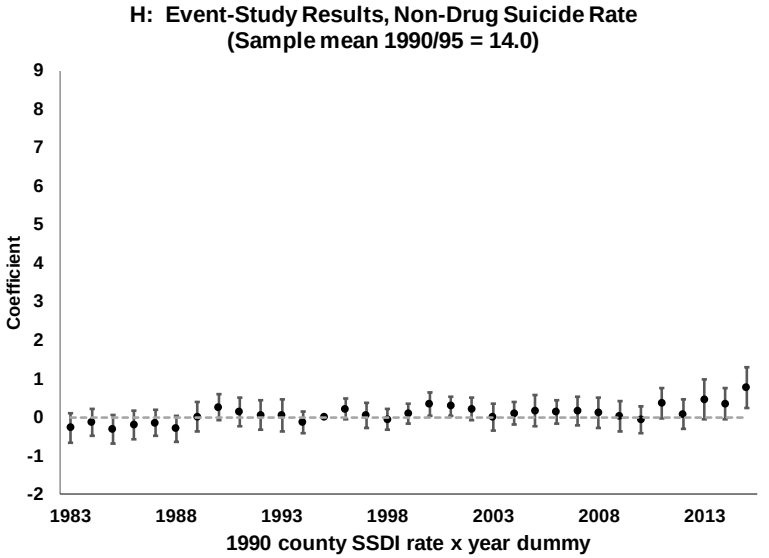


Figure 9
Regression-Adjusted Differences in Quarterly Morphine Equivalent Grams (MEG) per 100,000 Population for all Opioids Between Top and Bottom Quartile of County SSDI Rate in 1990
For Oxycodone and All Opioids Minus Oxycodone ARCOS Data, 1997.1 – 2015.4

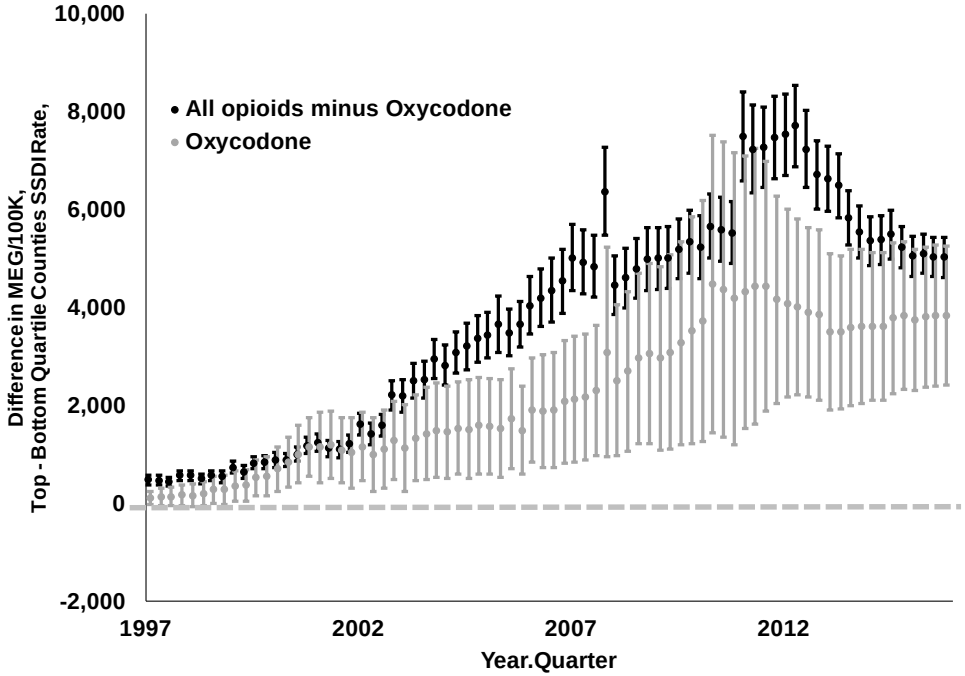


Table 1
Difference-in-Difference Estimates for Drug Poisoning Death Rates, 1983-2015

	Baseline	No demographic controls	PDMPs	Linear county trends	State x year f.e.	Employment rate	China trade shock
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

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.

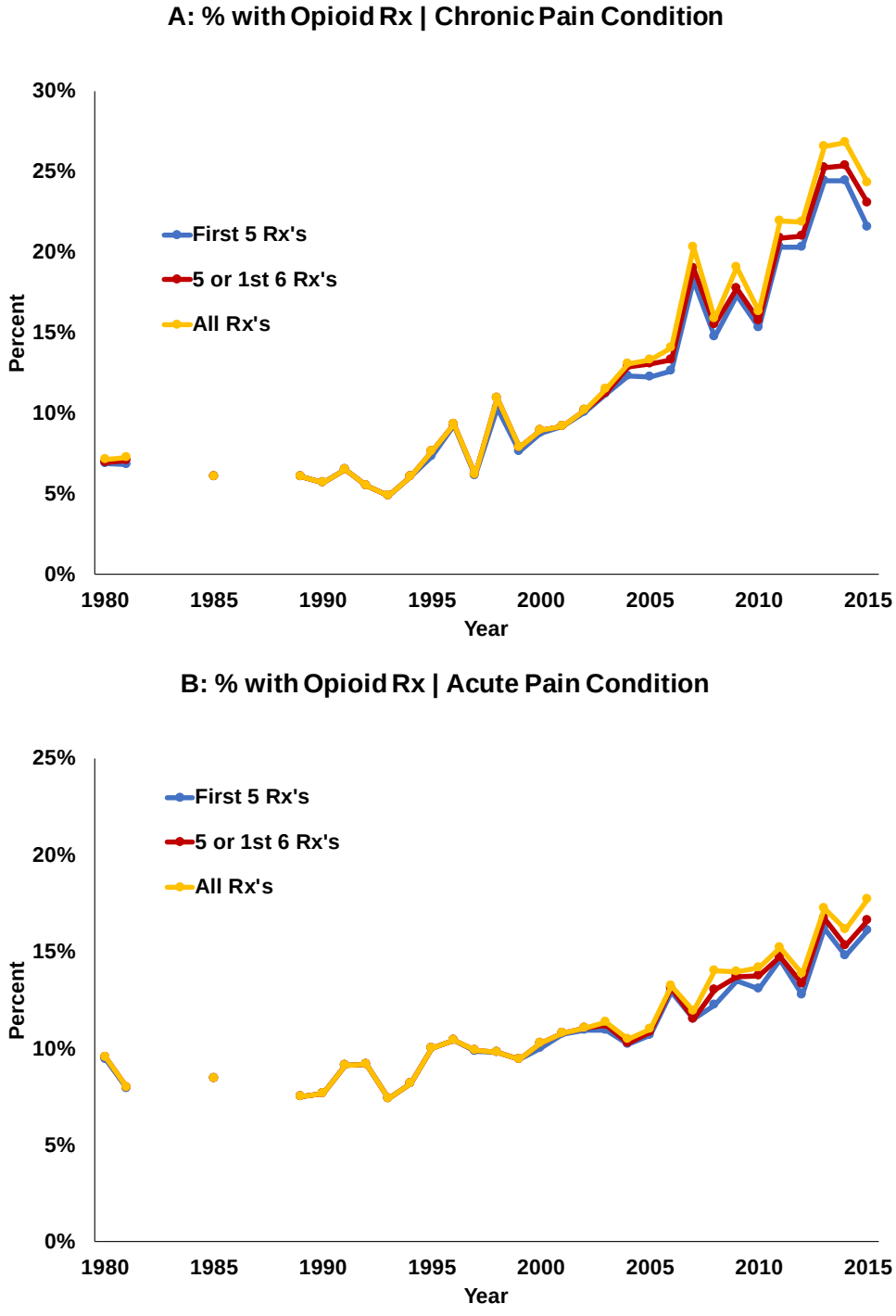
Table 2
Difference-in-Difference Estimates for Drug Death Poisoning Rates and the Role of Marketing, 1983-2015

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 zero	<0.001		<0.001
F-test, all Non-triplicate zero		<0.001	<0.001

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 10,498 observations in total.

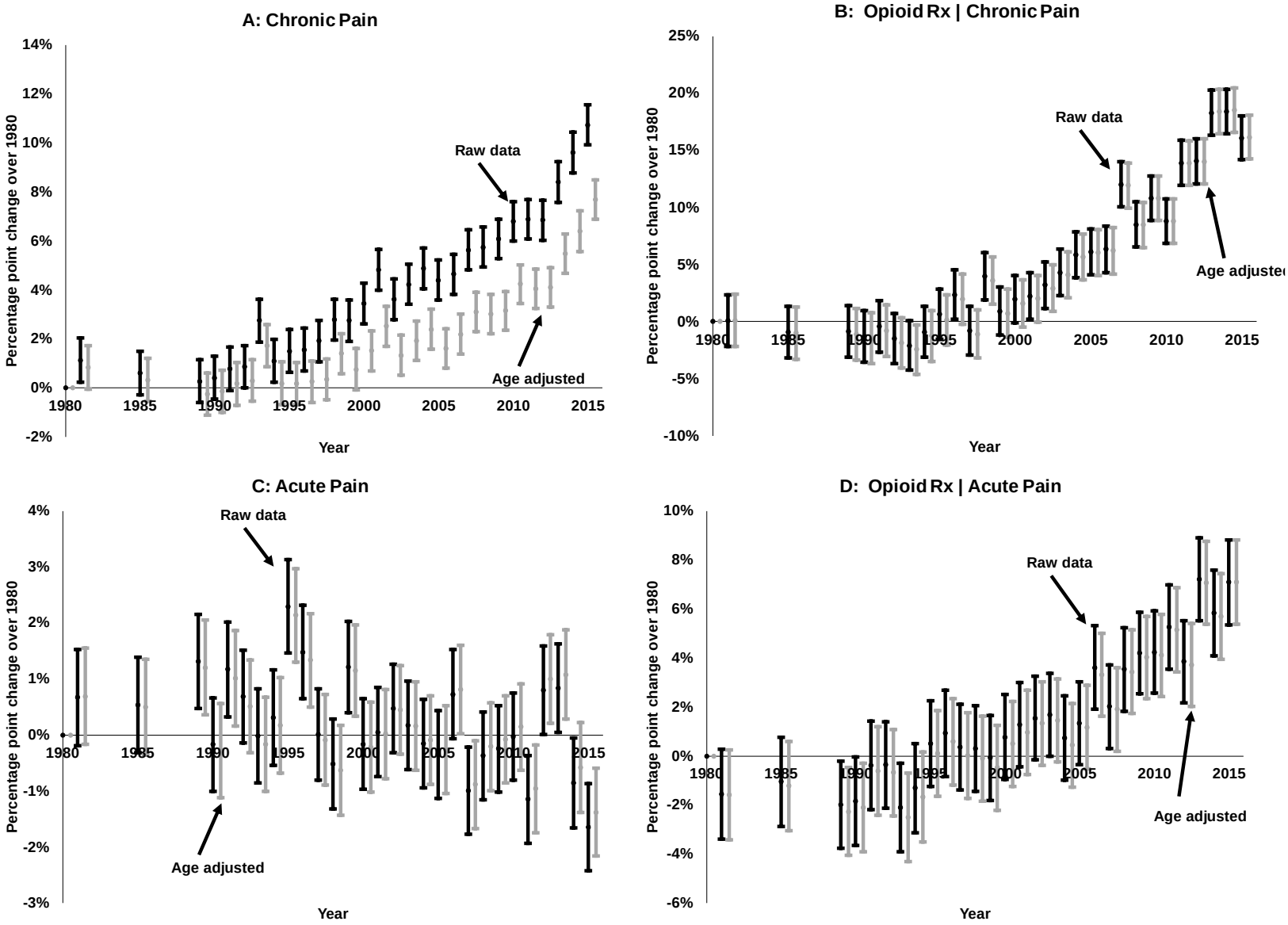
Appendix A: Additional Tables and Figures

Appendix Figure A1
Fraction of Patients Presenting with a Chronic or Acute Pain Condition Receiving an Opioid Prescription Using Different Number of Prescriptions, Adults 18-64, NAMCS Data

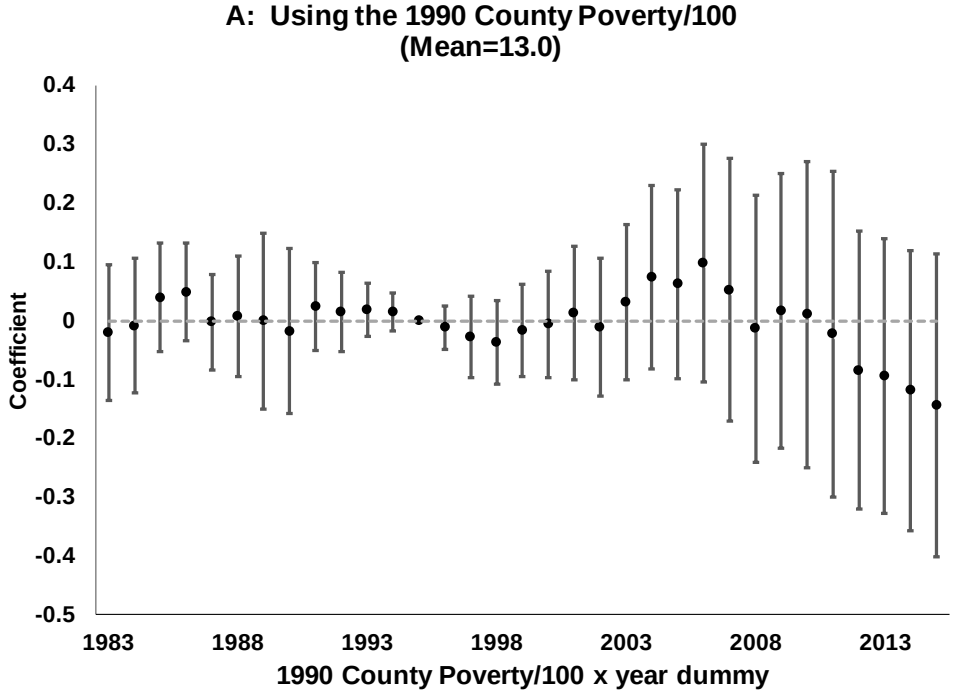


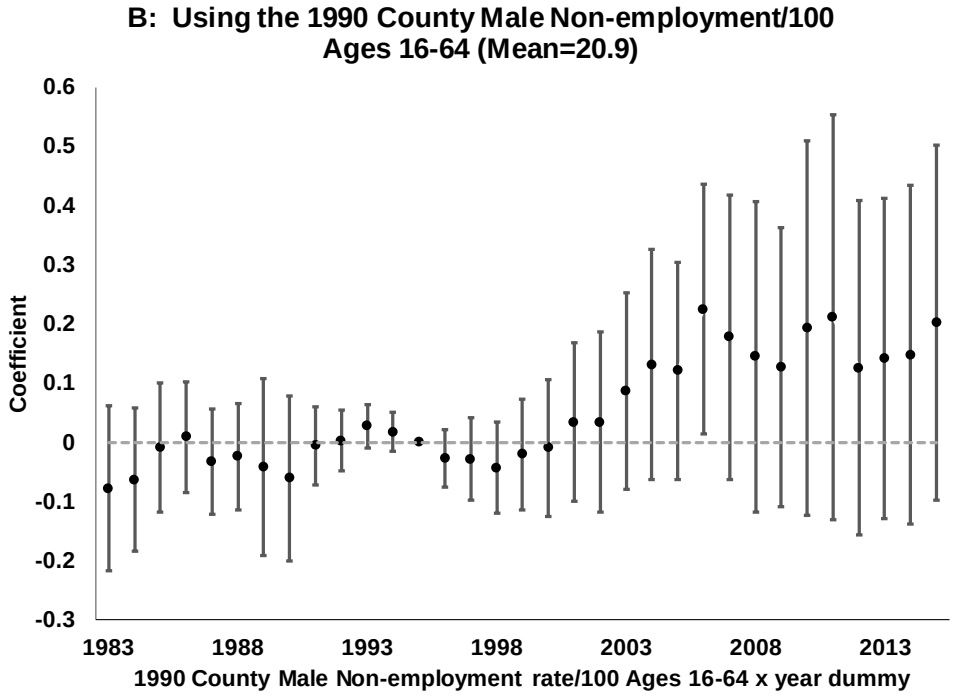
Appendix Figure A2

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

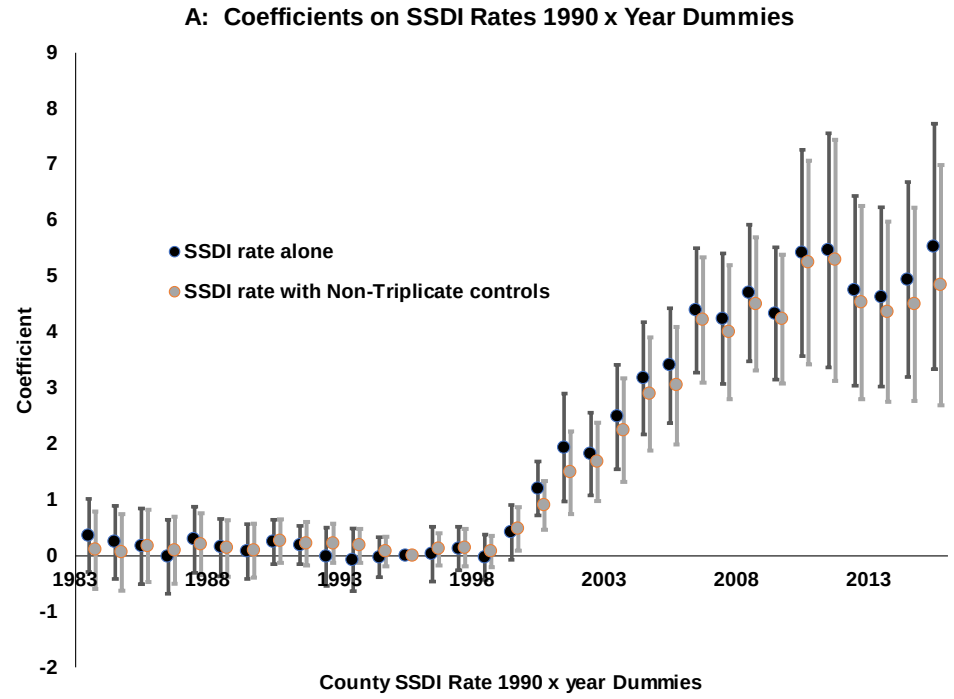


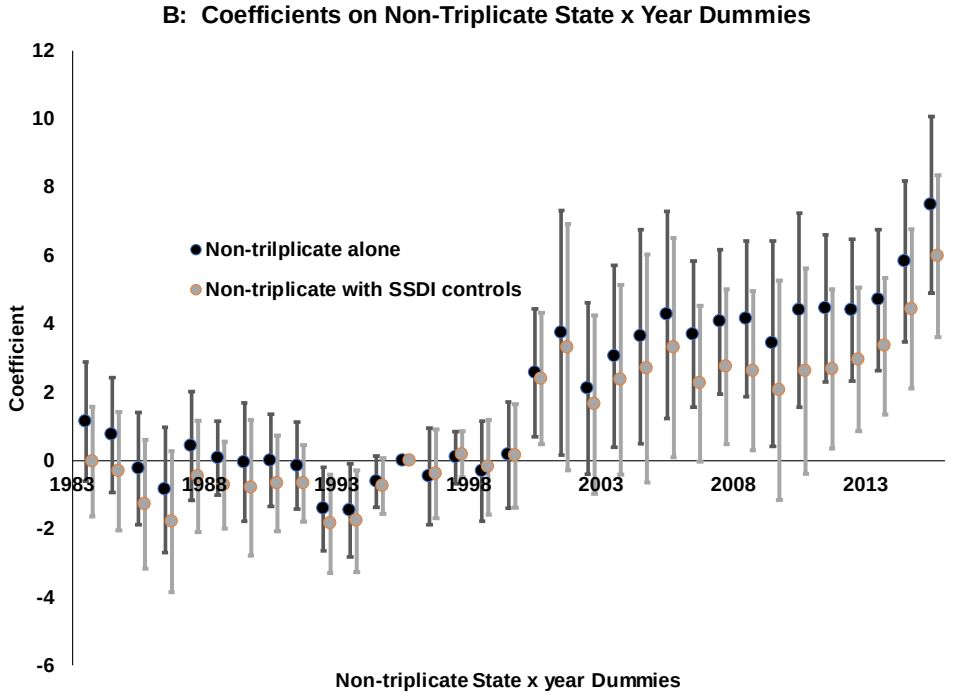
Appendix Figure A3
Event Study Results, Drug Death Rate at the County Level, 1983-2015,
Changing the County Measure in 1990
Parameter Estimates and 95% Confidence Intervals



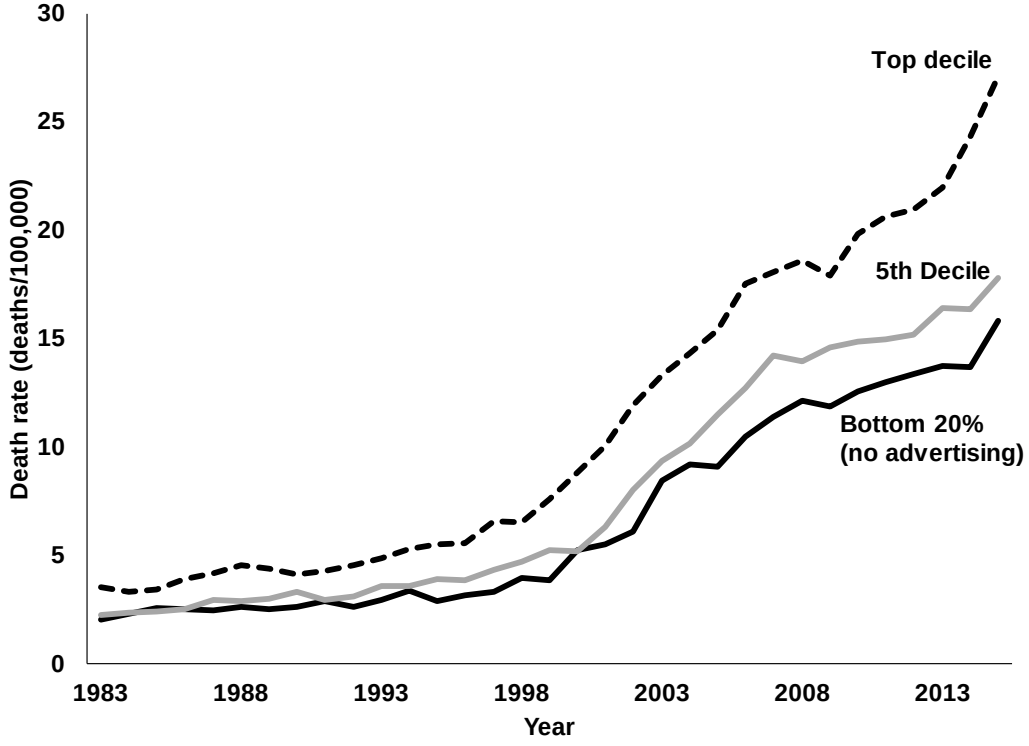


Appendix Figure A4
Event Study Results, Drug Death Rate at the County Level, 1983-2015,
SSDI and Non-Triplicate Treatments
Parameter Estimates and 95% Confidence Intervals





Appendix Figure A5
Drug Poisoning Deaths Rates Over Time Based on the Decile of Advertising Visits/1,000
In the Open Payments Data Set in 2016-2019



Appendix B:

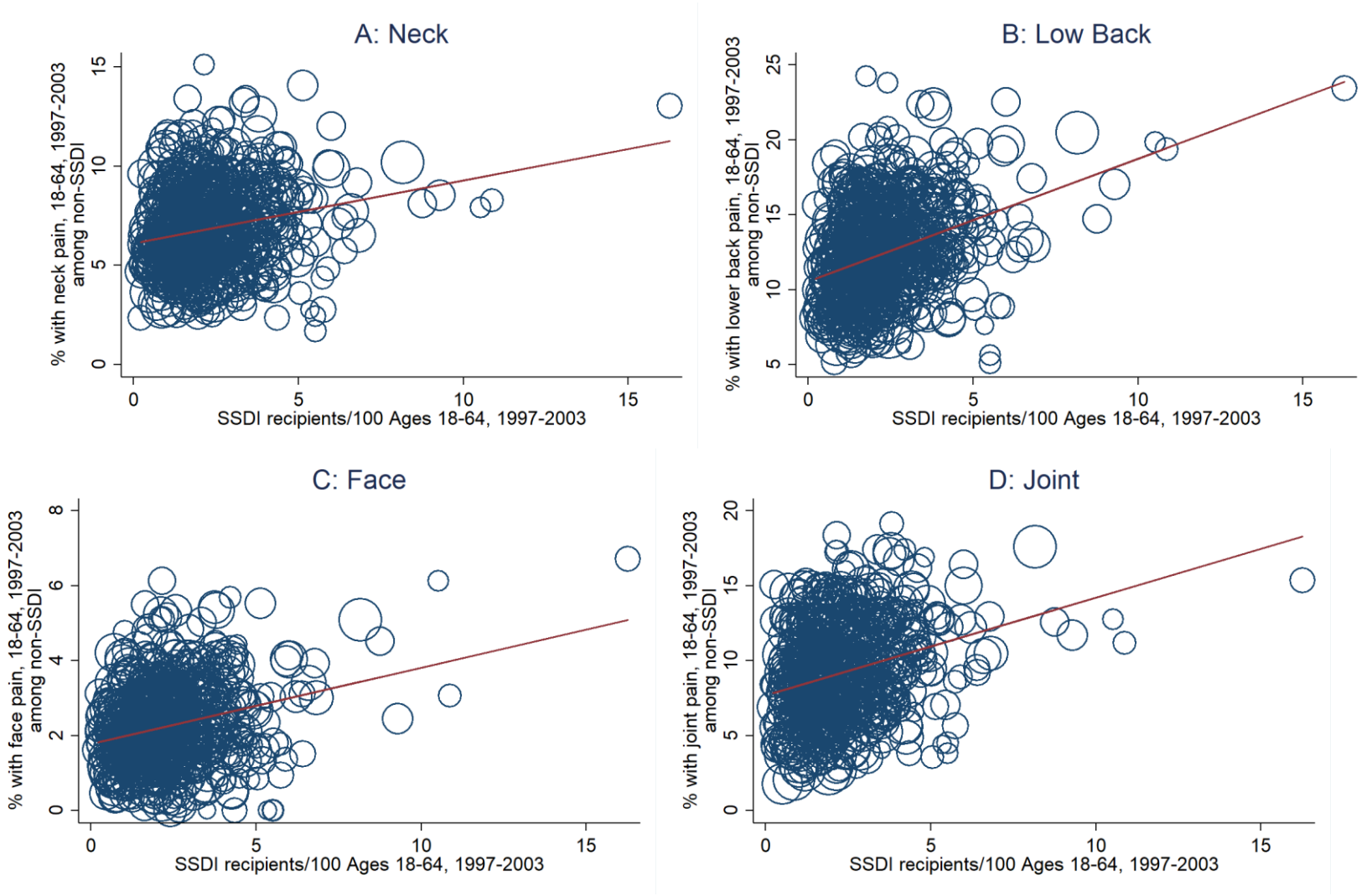
Relating SSDI Rates to Pain, Additional Information

IPUMS processed and harmonized data from the National Health Interview Series. We use those data and refer to them as the NHIS. The public use NHIS does not include geographic identifiers. However, part of the sampling process breaks the United States up into groups of counties and large metropolitan areas. These form the basis of the PSU and strata variables and over certain sets of years (1997-2004; 2005-2014; 2015-2018) the PSU and strata variables are consistent over time. For example if a certain cluster of counties were represented by PSU 1 and strata 3 in the year 1997, then those same counties would be coded as PSU 1 and strata 3 until 2004. While this does not allow us to merge on the geographic area's SSDI rate in 1990, the NHIS contains enough information for us to estimate the SSDI rate in that given year and geographic area. We have 678 unique PSU by strata combinations in each year of the NHIS that we use.

In Figure 4, we provided the correlation between SSDI rates and an aggregate measure of pain in the geographic region. That aggregate measure is based on four questions that were consistently asked in the NHIS between 1997 and 2003. There were other years in which these questions were asked, but the geographic units (strata by PSU) were not consistent. Three of the questions asked whether an individual had a specific type of pain that lasted for at least a whole day in the past three months. These types of pain were: neck pain, low back pain, and facial pain. The fourth question asked whether the person had had any pain, aching, or stiffness in or around a joint, excluding the neck and back, in the past thirty days. Between 1997 and 2003, the average fraction of individuals who responded yes to these types of pain were 0.07 (neck), 0.13 (low back), 0.02 (facial), and 0.09 (joint).

In Figure B1, we show the relationship between SSDI rates and each of the four measures of pain from 1997-2003. There is a positive relationship in each subfigure ranging from 0.21 (neck pain) to 0.35 (low back pain). The red line in each figure represents the regression line; the results are presented in Table B1 below. Because there are some values of the SSDI rate that could be seen as outliers, we have checked whether the regression estimates are sensitive to omitting the top 5 or top 10 percent of SSDI rates. As seen in Table B1, the point estimates actually increase when we omit the highest SSDI rate observations. This suggests that the potential outliers are not artificially creating the positive relationship we observed in the full sample.

Appendix Figure B1
Relationship Between 1997-2003 Measures of Pain for those not on SSDI and SSDI Rates, NHIS



Appendix Table B1
Simple Regressions of Pain Measures for those not on SSDI
Against SSDI Rates, NHIS 1997-2003

	Neck	Low Back	Face	Joint
SSDI/100 18-64 year olds				
Full sample	0.317	0.818	0.204	0.651
	(0.052)	(0.081)	(0.031)	(0.086)
Drop top 5 pct of SSDI	0.353	0.942	0.203	0.734
	(0.080)	(0.119)	(0.042)	(0.129)
Drop top 10 pct of SSDI	0.379	1.026	0.231	0.741
	(0.095)	(0.141)	(0.048)	(0.153)

Regressions are at the strata by PSU unit of observation and are weighted by the number of 18-64 year olds in the cell. Robust standard errors are reported.

Appendix C: The Multiple Cause of Death Data

Our analysis requires data on underlying cause of death with geography defined at the county. 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 the, 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) as one county. We do not use 1982 because in that year, there was a 505 sample for 19 states. As a result, we start our analysis with 1983. 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. Fir this reason, we end our analysis in 2015, we could have easily ended it in 2013 as well when fentanyl first came to prominence. In all years in our sample except 1982, the MCOD is a census of deaths in the US. In 1982, there is a 50 percent sample from 19 states.

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). To identify deaths by cause, we use the coding from 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 code 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 C1 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.[17] 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 C1.

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

[17] 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.

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.

Table C1
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. We do this for two reasons. First, these are 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. Second, at the end of the paper, we examine deaths by level of education and this only makes sense for adults. To make the samples as consistent as possible, we focus solely on adults aged 18 and above.

In 1989 there was a redesign of the MCOD data and one variable that was added was the education of the deceased. In the early years of this variable, the variable measured completed years of education but in 2003, mirroring the changes that occurred in data sets such as the Current Population Survey, the coding was changed to reflect credentials obtained. The codes used in the two versions are reported in Table C2 below. After 2002, counties could report either the pre-2003 or the newer coding.

We construct three education groups in the analysis: high school or less, some college but no 4-year degree, and a 4-year degree or more. In Table C3, we show the codes used to construct these three groupings. We are implicitly assuming that someone that went to four years of college received a bachelor's degree in the early period.

One problem with the education variable is that it was poorly reported the first few years. Among deceased adults aged 18 and up, the variable was not reported in 29 percent of cases in 1989. This fell to 10 percent in 1994 and 7 percent by 2000. In analyses based on education, adding the early years of the sample may not be comparable to later years. However, we argue in the paper that there was a structural shift in prescribing opioids starting around 1996 so we need as many pre-years as possible. In Figure C1 below, we report the fraction of counties that have less than or equal to 10, 15, and 20 percent non-response to the education question by year. This non-response is really high in 1989 but there are a large number of counties that report at least 80 percent by 1990. As a compromise, we use a sample of counties that have less than or equal to 20 percent non-reporting for 26 years, 1990-2015. This gives us a sample of 2101 counties from 43 states. We refer to this as our balanced sample. In Figure C2, we provide the fraction of adult drug poisoning deaths and the fraction of the adult population from these counties in this balanced sample. It is roughly three quarters in both samples. More importantly, in Figure C3 we report the adult drug poisoning death rate for the entire country and our balanced sample. These two series lie on top of each other.

Table C2
Education Codes in the MCOD Data

1989-2002	2003 and on
0. No formal education	
1. 1 year of education	1. 8th grade or less
2. 2 years of education	2. 9-12th grades
.	3. High school graduate or GED recipient
.	4. Some college, no degree
12. 12 years of education	5. Associate's degree
13. 1 year of college	6. Bachelor's degree
.	7. Master's degree
.	8. Doctorate/professional degree
16. 4 years of college	
17. 5+ years of college	

Table C3
Coding of Education Levels

Education group	1989-2002	2003-on
High school or less	0-12	1-3
Some college but no 4-year degree	1-15	4-5
4-year college degree	16-17	6-8

Figure C1
Percent of the Adult Population in Counties Based on the Fraction of Death Certificates Not Reporting Education

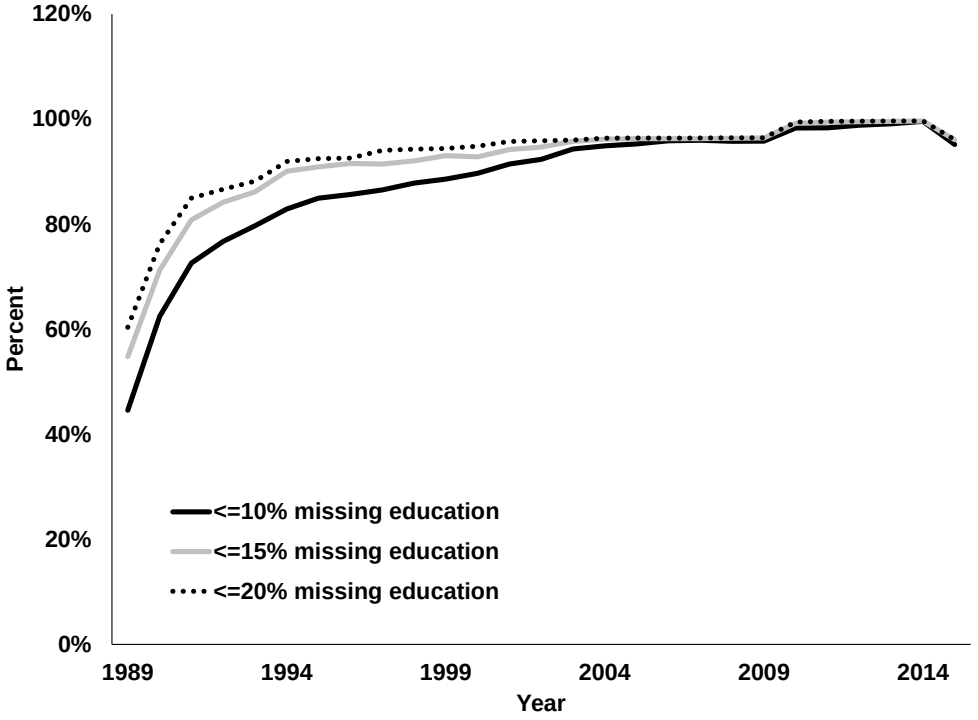


Figure C2
Percent of the Adult Population in and Drug Deaths in the Balanced Panel of Reporting Counties

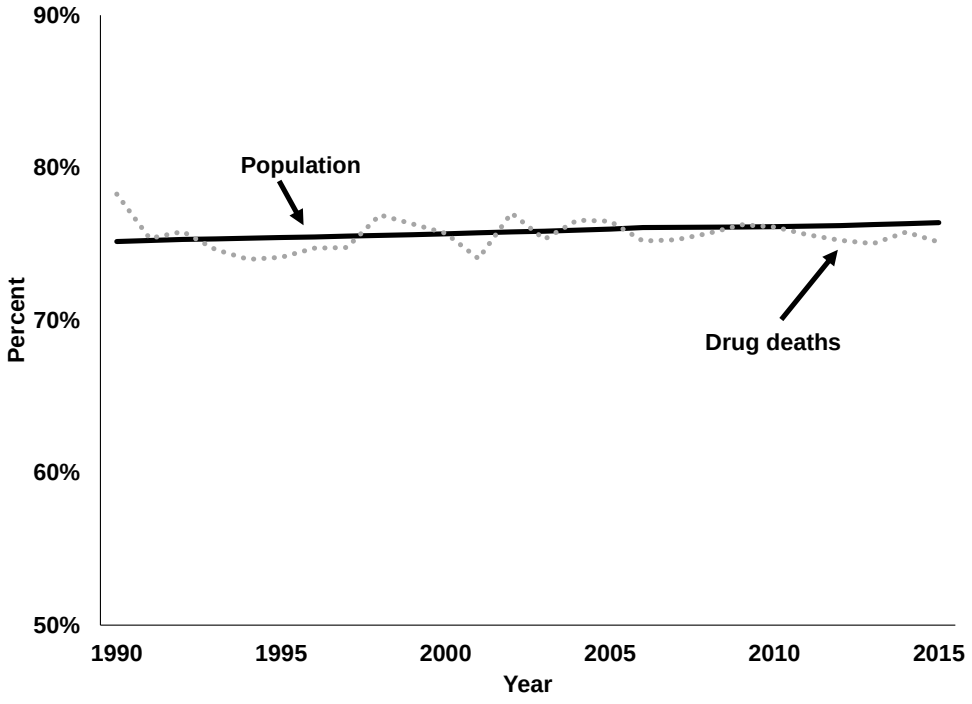
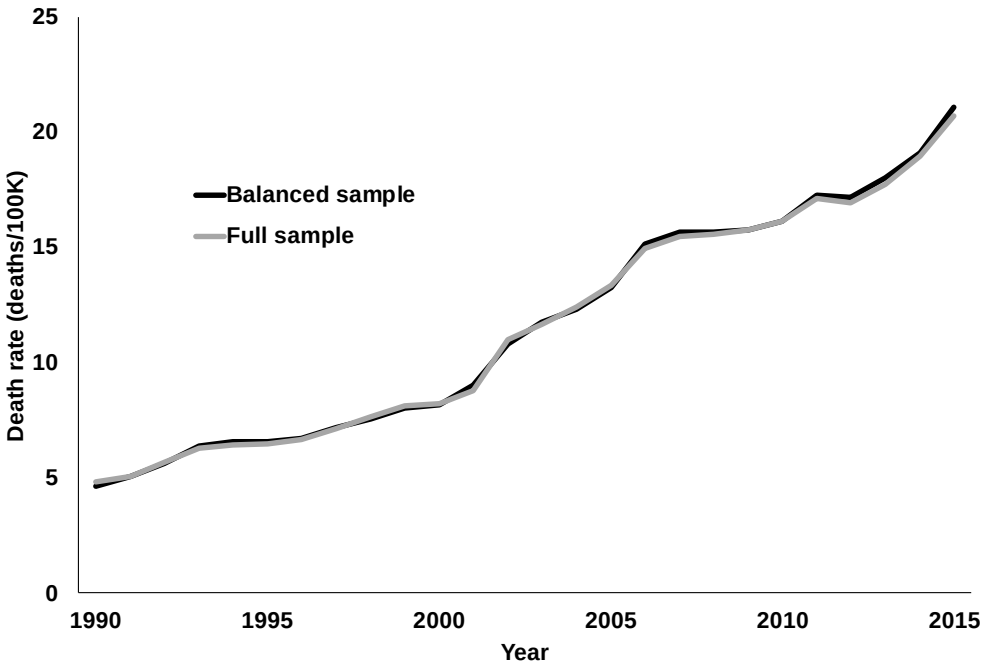


Figure C3
Comparison of Drug Poisoning Death Rate for All Counties
and in the Balanced Sample



Appendix D: 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.[18] SEER has a number of data sets and in this case we use two. First, we use population counts for single age breakdown by race and sex are available back to 1969. 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 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 county by age are not reported in Census tables for all counties. We were able to get complete series from 1982 on 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

Appendix E: 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 count, 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.

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

Appendix F: An Alternative Proxy for Aggregate Pain: Work Limitations

The SSDI rate in 1990 is but one proxy for the amount of aggregate pain in a county. In this appendix, we present basic results using an alternative metric of aggregate pain: the share of working-age adults with a condition that limits their ability to work. In the 1990 Census, long-form respondents aged 16 and above were asked a whether a physical, mental, or other health condition limits the kind and amount of work they can do. Using data from the NHGIS system (Manson et al., 2022), we construct the share of individuals aged 16-64 that answered yes to this question and call this the share with a work limitation.

This is a potential proxy for aggregate pain in that individuals with a work limitation have substantially higher rates of pain. Since 1997, the National Health Interview Survey (NHIS) has asked adults aged 18 and above whether they have a mental, physical or emotional condition that limits the kind or amount of work they can do. We define someone as having a work limitation if they report the condition makes it unable for them to work or limits the amount or type of work they can do. Using the 2011 through 2015 NHIS data from above, we can calculate the fraction of people that have experienced pain on most or all days over the past three months. Of adults aged 18-64 from 2011-2015, 10.1 percent reported they had a work limitation and among those in this group, 59.2 percent said they experience pain on most or all days. This same number for those without a work limitation is 12.1 percent, or one fifth the rate for those with a limitation.

The share of adults with a work limitation in 1990 and the SSDI rate in the same year are highly correlated. In Appendix Figure F1, we present a bubble plot at the county level between these two metrics with the size of the bubble being the number of adults aged 18-64. The correlation between these two measures is very high at 0.86. The share with a work limitation is a much broader measure in that the population-weighted average across counties (7.4 percent) is more than four times the SSDI rate (1.8 percent).

Next, we demonstrate that the share with a work limitation is a predictor or aggregate pain across geographic area for those without a limitation. Similar to Figure 4, in Appendix Figure F2, we use data from the 2011-2015 NHIS and produce a bubble plot at the PSU x strata level of the fraction of adults 18-64 with a work limitation and the fraction of the same aged adults without a work limitation that have pain on most or all days over the past three months. The correlation coefficient, weighted by adult population in the PSU x strata cell is large at 0.42.

In Figure F3, we reproduce Figure 5 where we correlate the share of adults with a work limitation in 1990 from the Census at the county level with various death rates. The patterns in this graph look very similar to the patterns in Figure 5. First, the share with a work limitation is highly predictive of aggregate non-drug death rates as this correlation is always in excess of 0.60. Next, the share with a work limitation is also predictive of two deaths of despair categories: alcohol death rates and non-drug suicide death rates. As with the SSDI rate, the share with a work limitation is actually negatively correlated with the drug death rate in the pre-2000 period but this correlation increases dramatically afterwards, peaking at about 0.39 in 2010.

In Figure F4A, we graph the drug poisoning death rate for the counties in the top and bottom quartile of the share with a work limitation in 1990. These graphs are very similar to those in Figure 8 in that death rates are higher in the counties with lower work limitations in the pre-1996 period. This changes dramatically post-1995 with deaths increasing much more rapidly in higher-share counties afterwards. The pre-1996 trends in both groups of counties are quite similar. We then re-estimate the event-study estimates from equation (1) replacing the year x SSDI rate interactions with year x share work limitations, again using 1996 as the reference year. These results are reported in Figure C4B with the coefficients and 95% confidence intervals. In these models, there is no pre-1996 trends in the coefficients, but in the post-1995 period here is a rapid increase in the coefficients.

To put some magnitudes on these coefficients, note that between 1996 and 2015, drug death rates increased by 10.6 in the counties with the lowest quartile of the share work limitation while the same number is 20.0 in the highest quartile counties, a difference of 9.4. The average share with a work limitation in the top and bottom quartile counties is 12.2 and 5.8 percent, a difference of 6.4. The 2015 coefficient in the event study is 1.89 so we expect the increase in top quartile counties relative to bottom quartile counties to be 1.89*6.4=11.8 which is 129 percent of the actual change. The corresponding number we produced when the SSDI rate was used in the event study was 125 percent. These two measures of aggregate pain are producing essentially the same results.

In the first column of results for Appendix Table F1, we display the basic difference-in-difference estimates analogous to Table 1 but using the share with a work limitation as the key interactor. The interaction term in the 2011 to 2015 period is 1.8 which means that moving from the average lowest to average higher quartile county increased drug death rates by 11.5 (1.8*6.4) which is very close to the simulation we get using the SSDI rate. More importantly, in column (2) where we remove the year effects and replace them with state x year fixed effects that capture any state regulatory or economic effect that is common to all counties within a state, in a given year, the results do not change much.

Finally, in Appendix Table F2, we add year group interactions with the non-triplicate status of the states to see if there is an independent impact of Purdue Pharma advertising once we control for work limitation x year group effects. The 1st column of results has only the work limitation x year group effects, the second column adds the non-triplicate x year interactions, and the final column adds both sets of coefficients. We allow the difference-in-difference estimates to vary based on triplicate status of the states. Adding year group x non-triplicate effects does not change the work limitation x year group effects by an appreciated amount. That said, the final two-year group x non-triplicate dummies are smaller once we control for the change in prescribing behaviors, but the coefficients are still large and statistically significant.

Figure F1
Bubble Plot, 1990 Share Adults 16-64 With a Work Disability vs.
1990 County SSDI Rate for Adults Ages 18-64

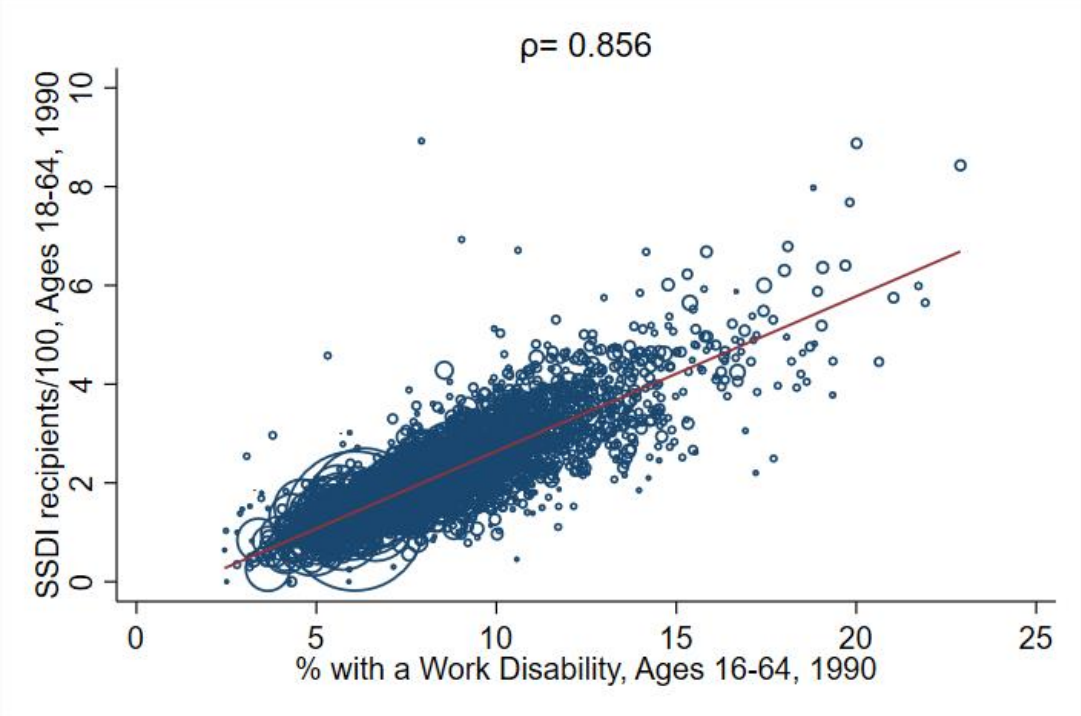


Figure F2
Bubble Plot, Share of Adults 18-64 with a Work Limitation and the Share of
Adults without a Work Limitation in Pain Most or all Days, 2011-2015 NHIS

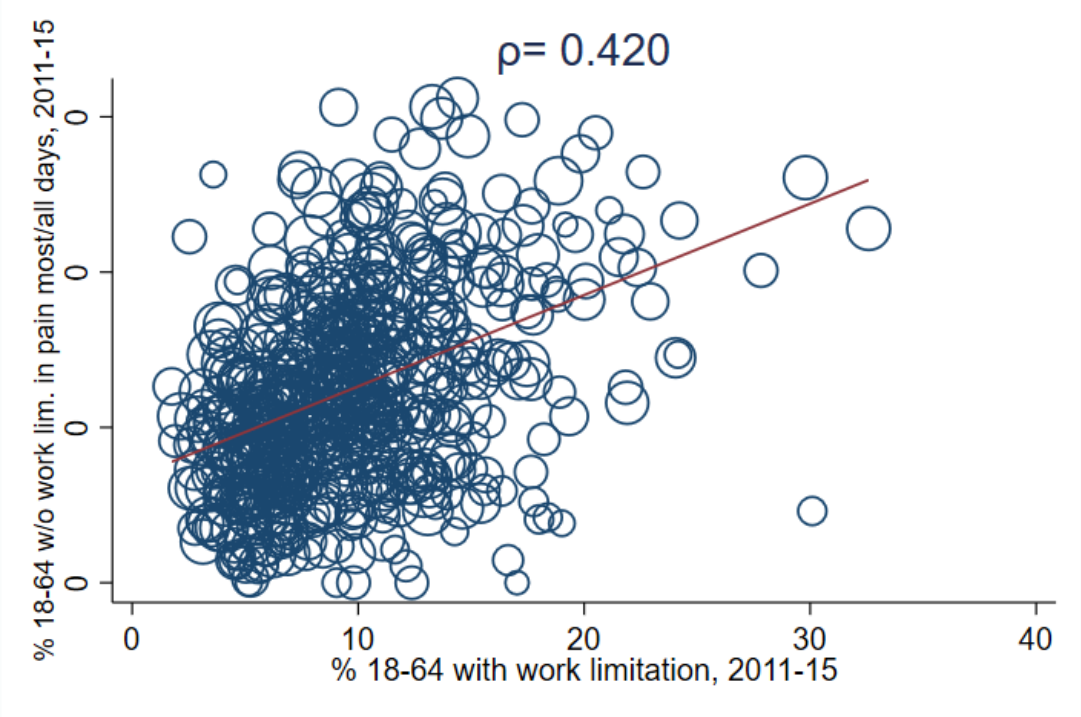


Figure F3
Correlation Coefficient Between 1990 Share of Adults with a Work Limitation and Adult County Death Rate for Various Years, 1990-2015

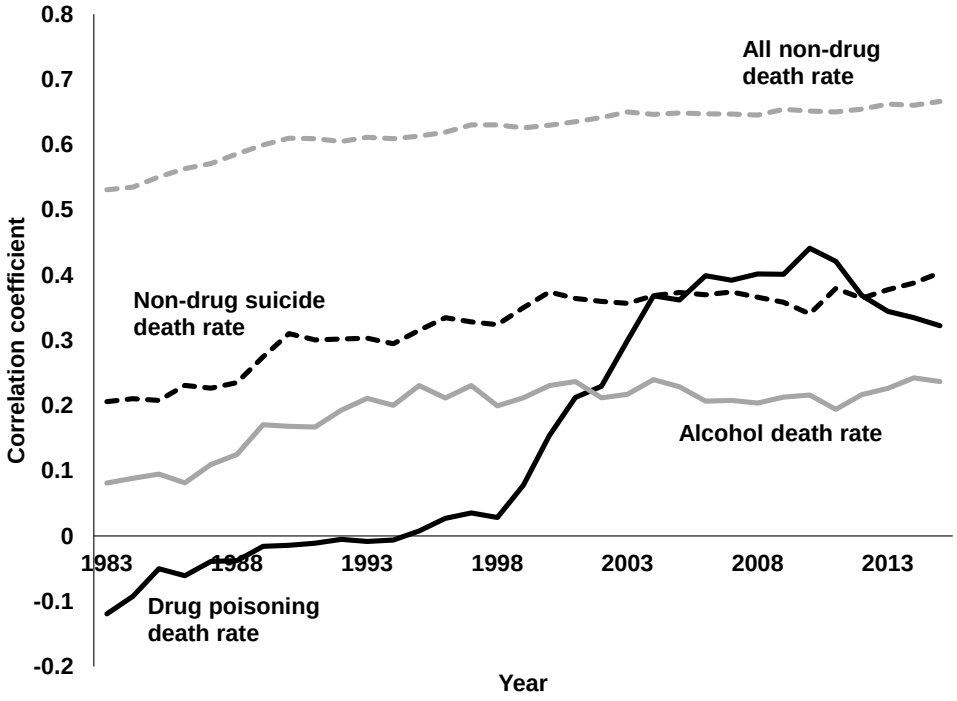


Figure F4
Drug Death Rates for Adults Ages 18+ by Quartile of 1990 Share with a Work Limitation, and Event Study Results, Drug Death Rate at the County Level, 1983-2015, Parameter Estimates and 95% Confidence Intervals

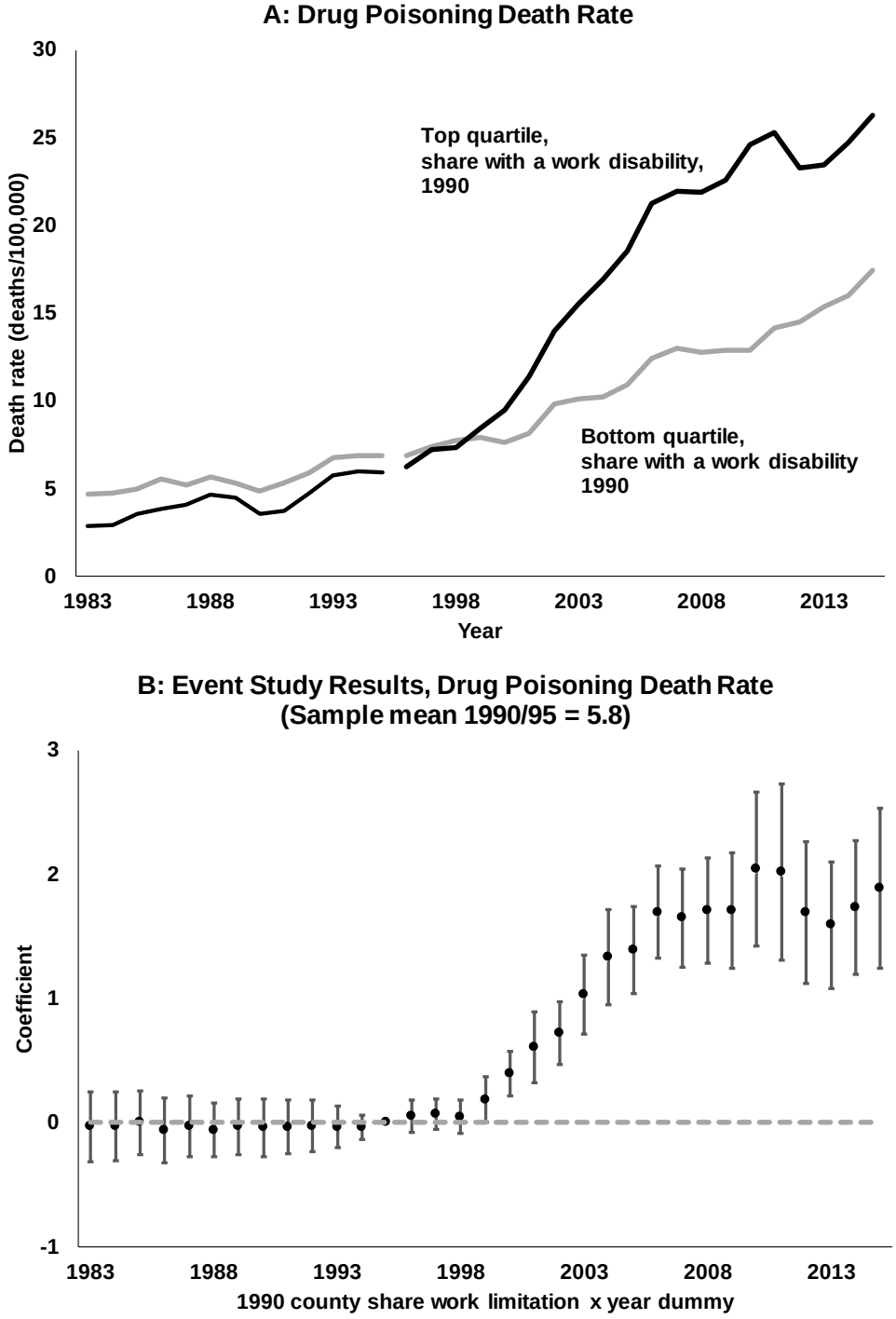


Table F1
Difference-in-Difference Estimates for Drug Poisoning Death Rates, 1983-2015

Covariates	Baseline	State x year fixed effects
1990 Share with a work limitation x		
1996 – 2000	0.18	0.43
	(0.10)	(0.10)
2001 – 2005	1.04	1.13
	(0.17)	(0.13)
2006 – 2010	1.77	1.74
	(0.20)	(0.17)
2011 – 2015	1.80	1.86
	(0.29)	(0.22)
F-test, all zero	<0.001	<0.001
Mean, 1990-1994	5.8	5.8

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.

Table F2
Difference-in-Difference Estimates for Drug Death Poisoning Rates
and the Role of Marketing, 1983-2015

Covariate	Work limitation only	Non-triplicate only	Both
1990 share with a work limitation x			
1996 – 2000	0.18		0.18
	(0.10)		(0.90)
2001 – 2005	1.04		0.96
	(0.17)		(0.16)
2006 – 2010	1.78		1.72
	(0.20)		(0.21)
2011 – 2015	1.80		1.67
	(0.29)		(0.30)
Non-triplicate x			
		0.66	1.10
1996 - 2000		(0.66)	(0.65)
		3.59	3.36
2001 - 2005		(1.31)	(1.37)
		5.61	3.37
2006 - 2010		(0.98)	(1.12)
		5.61	4.92
2011 - 2015		(0.98)	(1.13)
F-test, all work lim. zero	<0.001		<0.001
F-test, all non-triplicate zero		<0.001	<0.001

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 10,498 observations in total.