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Understanding the Demographics of the Opioid Overdose Death Crisis*

David Powell[†]

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Abstract

The United States is enduring the worst drug overdose crisis in its history. Relative to previous overdose epidemics, the opioid crisis is marked by its severity, breadth, and demographics. Some demographic groups have experienced staggering rates of overdose growth. I examine overdose trends during the opioid crisis relative to historical disparities and evaluate the role of OxyContin's launch in explaining these trends. I use geographic variation in OxyContin's launch to understand the impacts of a large opioid shock on the demographics of the crisis. I find that OxyContin's introduction induced substantial and enduring differences in overdose rates across demographic groups.

Keywords: Overdose Deaths, Racial Disparities, OxyContin, Purdue Pharma, Deaths of Despair

JEL Classification: I12, I18, J11, J15

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1 Introduction

In 2019, more than 70,000 individuals in the United States died of drug overdoses, over 70% of which involved opioids (CDC, 2021). The opioid crisis is a national emergency, marked in contrast to previous drug epidemics by its severity, its breadth, and its demographics. All demographic groups have experienced increases in overdose death rates; however, the epidemic has flipped some pre-existing disparities in overdose death rates while exacerbating others. The literature has documented that growth in overdose rates has differed substantially by race/ethnicity,¹ sex (Altekruse et al., 2020; Mack et al., 2013; McCarthy, 2013), and education (Ho, 2017). However, our understanding of these differential impacts is limited, consistent with a lack of research on demographic-specific disparities more broadly in the social sciences (Advani et al., 2021) and despite the importance of examining such disparities in the context of the opioid crisis (Om, 2018).

The heterogenous effects of the opioid crisis on overdose death rates are striking, but little work has explored the underlying determinants of this variation. The literature has debated whether the opioid crisis is a function of demand² (“deaths of despair”) or supply (Maclean et al., 2020; Currie and Schwandt, 2021). However, these forces may not impact and equilibrate all groups equally. For example, the disproportionate burden shouldered by groups with low education is consistent with reduced economic prospects and limited job opportunities driving demand for substance use (Ho, 2017). Yet, as highlighted in Currie and Schwandt (2021), Black Americans have experienced persistently poorer economic outcomes, but historically have not felt the full force of the opioid crisis when compared to non-Hispanic White Americans.³

In this paper, I examine how the opioid crisis has affected overdose rates for specific demographic groups relative to historical trends. While there are small literatures studying overdose rates for some demographic groups in the context of the opioid crisis, this

¹See Shiels et al. (2018); Alexander et al. (2018); SAMHSA (2020); Singh et al. (2019); Stevens-Watkins (2020); Drake et al. (2020); Tipps et al. (2018); Cano (2021).

²Case and Deaton (2015, 2017) argue that rising overdose rates are a function of deteriorating economic and cultural conditions for large segments of the population. A growing literature studies how economic conditions or shocks to labor opportunities relate to overdoses or a broader set of deaths of despair (Hollingsworth et al., 2017; Ruhm, 2019a; Venkataramani et al., 2020; Pierce and Schott, 2020; Betz and Jones, 2018; Charles et al., 2019; Currie et al., 2019). In addition, Ruhm (2019b) documents that non-opioid overdose death rates are also rapidly increasing, suggesting that the ongoing overdose epidemic is not specific to opioids.

³The overdose rates of White and Black Americans have converged in recent years (Alexander et al., 2018; Case and Deaton, 2021).

research tends (with some exceptions) to document overdose rates starting in 1999 or more recently. By extending these analyses further back, I can study how the opioid crisis has impacted overdose rates across demographic groups relative to historical (i.e., before the opioid crisis) differences. This comparison contextualizes how the opioid crisis has exacerbated pre-existing demographic differences in overdoses as well as which demographic groups have been disproportionately harmed relative to historical rates.

In addition, I examine the effect of OxyContin’s launch on overdose death rates by race/ethnicity, sex, and education. I focus on OxyContin because of its pivotal role in the opioid crisis (Alpert et al., 2021; Kolodny et al., 2015). Purdue Pharma introduced OxyContin in 1996. Soon after its launch, OxyContin became a blockbuster drug, and Purdue Pharma aggressively marketed it and the use of strong opioids more broadly.⁴ Within just a few years after its introduction, OxyContin was the most abused opioid in the United States (Cicero et al., 2005).

OxyContin was introduced nationwide; however, there is persistent geographic variation in OxyContin supply (and, as a result, total opioid supply) based on whether a state had a “triplicate prescription program” at the time of OxyContin’s launch. Triplicate programs, which tracked controlled substances from prescriber to pharmacy to a state monitoring agency, were early and especially stringent forms of prescription drug monitoring programs. They led Purdue Pharma to conclude that “The product [OxyContin] should only be positioned to physicians in non-triplicate states...” (Groups Plus, 1995). This differential marketing triggered enduring geographic variation in opioid supply, prescribing, and promotional activities. I leverage this variation as a large differential supply shock to understand how OxyContin and its aggressive promotion affected the demographics of overdose death rates over the long term. I conclude that initial exposure to OxyContin and its enduring effects on opioid access drove large disparities in overdose death rates across demographic groups.

Understanding the differential impacts of the opioid crisis is important for targeting policies and resources. It is equally critical to discern the structural causes of these disparities so that appropriate policies can be matched to the underlying reasons for a group’s exposure to the opioid crisis. This paper considers the role of OxyContin and, more gen-

⁴Purdue Pharma’s stated objective in the early years was: “To convince health care professional (physicians, nurses, pharmacists, and managed health care professionals) to aggressively treat both non-cancer pain and cancer pain. The positive use of opioids, and OxyContin Tablets in particular, will be emphasized” (Purdue Pharma, 1999).

erally, medical access to opioids in explaining long-term differential overdose death rates in the U.S. population.⁵

Due to the severity of the opioid crisis, overdose trends have markedly altered life expectancy (Harper et al., 2021), and there is increasing interest in the changing mortality rates across demographic groups. This paper enhances our understanding of broader mortality trends in the United States. The education gradient of mortality has been of special interest to the literature (e.g., Lleras-Muney (2005); Clark and Royer (2013); Leive and Ruhm (2021a)) as have mortality differences by race and ethnicity (Curtin and Arias, 2019; Satcher et al., 2005; Schwandt et al., 2021) and sex (Geronimus et al., 2019).

The launch of OxyContin and its lasting effects on opioid access had enduring impacts on overdose disparities across demographic groups. White and Native Americans have especially been impacted by this supply shock. Men have been more affected than women, though women also experienced large overdose increases in response to increased opioid supply, especially relative to historical overdose rates. Individuals with no college education were disproportionately impacted; OxyContin exposure is responsible for a large and growing education disparity in overdose deaths. My analyses suggest that growth in drug overdose rates has been supply-driven across most demographics. These effects continued even after the opioid crisis transitioned to illicit opioid markets, consistent with findings in prior work (Alpert et al., 2018).

2 Background

2.1 OxyContin’s Launch

Recent work concludes that the introduction and marketing of OxyContin played a leading and ongoing role in the growth of overdose death rates (Alpert et al., 2021). OxyContin was introduced to the market in January 1996 by Purdue Pharma. OxyContin’s key innovation was its long-acting formula which provided 12 hours of continuous pain relief, significantly improving the quality and ease of pain management compared with previous drugs. However, the timed-release benefit of OxyContin is contingent on taking the pill whole. Crushing or dissolving the pill causes the high dose of oxycodone, intended to be released slowly over 12 hours, to be delivered all at once. This property made OxyContin especially easy to abuse.

⁵This medical access may (and likely does) spill over into illicit markets (Powell et al., 2020).

Purdue Pharma aggressively marketed OxyContin, especially for non-cancer pain (GAO, 2003), and it soon became a blockbuster drug with a high rate of misuse.

To understand the initial marketing of OxyContin, I made Freedom of Information Act (FOIA) requests to Florida, Washington, and West Virginia to obtain recently unsealed documents from investigations and court cases brought against Purdue Pharma in these states (see an example in Figure A.1). These internal Purdue Pharma documents included survey research suggesting that triplicate prescription programs had a chilling effect on the prescribing of strong opioids: physicians were worried about government oversight, and they viewed using the triplicate forms as a major hassle.⁶

The Purdue Pharma documents mention triplicate programs dozens of times, acknowledging that “these regulations create a barrier when positioning OxyContin” (Purdue Pharma, 1995). Since there would be lower returns to promoting OxyContin in triplicate states, the internal Purdue Pharma research recommended that “the product [OxyContin] should only be positioned to physicians in non-triplicate states” (Groups Plus, 1995).

2.2 Triplicate Prescription Programs

Triplicate prescription programs emerged as some of the first programs to monitor the prescribing and diversion of controlled substances. In a triplicate prescription program, the prescriber is mandated to use state-issued triplicate prescription forms when prescribing Schedule II controlled substances. The prescriber keeps one copy of the prescription. The patient provides the remaining two to the pharmacy, which keeps one copy and sends the other to the state monitoring agency.

At the time of OxyContin’s launch, five states (“triplicate states”) had triplicate programs – California (enacted 1939), Idaho (1967), Illinois (1961), New York (1972), and Texas (1982). Interestingly, these programs were enacted decades before the opioid crisis and were phased out in the years following OxyContin’s launch. Thus, this study examines the longer-term consequences of the initial targeting and adoption of OxyContin. I use the 1996 launch of OxyContin as a large, differential shock to opioid access to study its long-term

⁶“Writing triplicate prescriptions was more trouble than others, due to the details of the forms and the various people that need to be copied to them. To the extent that they [physicians] can avoid this extra effort, they will try to follow alternative protocols” (Groups Plus, 1995).

consequences on overdose disparities.⁷

Figure 1 displays evidence across multiple data sources that non-triplicate states were more “exposed,” in terms of supply and promotional activity, to the introduction of OxyContin. Panel A shows promotional payments for OxyContin, obtained from the Open Payments data base for August 2013 through the end of 2016 (Centers for Medicare and Medicaid Services, 2018).⁸ Promotional activities for OxyContin differ substantially between triplicate and non-triplicate states. It might be surprising that triplicate status in 1996 could have such long-term effects on marketing strategies, but a defining feature of Purdue Pharma’s detailing strategy was its persistence. Internal documents suggest that a core strategy of the sales force was to call and visit the top OxyContin prescribers, and this behavior continued, and became even more frequent, through 2018.⁹ Thus, contemporary differences in promotional activities reflect variation in initial targeting, amplified by a marketing strategy that targeted high prescribers.

I also examine early and longer-term differences in OxyContin “adoption” as measured in terms of prescriptions and morphine equivalent doses. I provide differences in per capita OxyContin morphine equivalent doses from the Automation of Reports and Consolidated Orders System (ARCOS) data (Drug Enforcement Agency, 2018) for 2000-2016 in Panel B,¹⁰ per beneficiary Medicaid prescriptions from the State Drug Utilization Data (SDUD) data (Centers for Medicare and Medicaid Services, 2015) for 1996-2005 in Panel C,¹¹ and per capita prescriptions in the Medical Expenditure Panel Survey (MEPS) (Agency for Healthcare Research and Quality, 2018) for 1996-2016 in Panel D.¹² In all cases, triplicate states have substantially less access/use of OxyContin, both in the years immediately after

⁷Alpert et al. (2021) dedicate a significant amount of analysis to isolating the underlying mechanisms generating the large overall post-1996 overdose effects, determining that it is primarily driven by a marketing effect and not direct effects of triplicate programs impacting overdose rates even years after repeal of the programs. Relative to Alpert et al. (2021), there is little new evidence in this paper about the underlying mechanisms for *why* differential access to OxyContin after launch caused such long-term mortality effects.

⁸The Open Payment Database collects and lists data on payments – for research, meals, travel, gifts, or speaking fees – from drug companies to physicians and teaching hospitals.

⁹“McKinsey recommended doubling down on Purdue Pharma’s strategy of targeting high prescribers for even more sales calls...” (Commonwealth of Massachusetts, 2018). Purdue Pharma ended promotional activities for OxyContin in 2018.

¹⁰The online ARCOS data are provided by ingredient. I made a FOIA request for data on OxyContin specifically. This request was approved for 2000-2016.

¹¹I end the time series in 2005 since Medicare Part D was enacted in 2006. While the current SDUD available online suppresses numbers of prescriptions less than ten, I use a version downloaded before this suppression policy was implemented.

¹²I accessed these data in the AHRQ Data Facility due to the necessity for geocoded data.

the launch in 1996 and continuing through the most recent years of data.¹³ Moreover, there were substantial spillovers to other strong oxycodone products because Purdue Pharma promoted the use of oxycodone more broadly (Purdue Pharma, 1999). Thus, total oxycodone (and opioid supply) differences between non-triplicate and triplicate states exceeded the differences in OxyContin (see Figure A.2).

3 Data and Empirical Analysis

3.1 Data

Overdose Deaths

I use the restricted geocoded National Vital Statistics System (NVSS) Multiple Cause of Death mortality files – the census of deaths in the United States – to study annual overdose deaths from 1983 to 2018 (Centers for Disease Control and Prevention, 2020).¹⁴ For 1983-1998, I define drug poisonings as deaths involving underlying cause of death ICD-9 codes E850-E858, E950.0-E950.5, E962.0, or E980.0-E980.5, following the CDC.¹⁵ For the 1999-2018 data, I code deaths as drug overdoses using the ICD-10 external cause of injury codes X40-X44, X60-64, X85, or Y10-Y14 (Warner et al., 2011).¹⁶

I focus on all overdose deaths to understand how the demographics of fatal drug overdoses have changed over time; using all overdoses also provides improved consistency over this period.¹⁷ I show supplementary results for overdoses involving opioids; those results tend to be stronger.

The death certificates include demographic information, although the consistency

¹³There is a notable dip in national OxyContin supply in 2005-2006 due to a patent dispute at this time. However, total oxycodone supply was unaffected over this period, presumably as generic OxyContin formulations substituted for the brand-name version (see Figure A.2). This dispute was eventually resolved and the generic versions exited the market. Despite these large shifts in national trends, the relative supply of non-triplicates and triplicates remained approximately the same.

¹⁴I begin in 1983 because the 1981 and 1982 files do not include all deaths.

¹⁵See Table 2 of https://www.cdc.gov/drugoverdose/pdf/pdo_guide_to_icd-9-cm_and_icd-10_codes-a.pdf, last accessed August 18, 2021. I include opioid-involving overdose results in the Appendix. I use deaths involving E850.0, E850.1, E850.2, or N965.0 (Alexander et al., 2018) for these analyses.

¹⁶The CDC reports that the transition from ICD-9 to ICD-10 resulted in a small increase in poisoning-related deaths (not necessarily drug poisonings) of 2% (Warner et al., 2011). I assume year fixed effects adequately account for this transition. When studying opioid-involving overdose deaths in the appendix, I use the T40.0-T40.4, T40.6 codes to limit overdoses to those involving opioids.

¹⁷Ruhm (2018) discusses missing opioid designations on death certificates. Empirical work has often used the broader overdose category for these reasons (e.g., Venkataramani and Chatterjee (2019)).

and inclusion of these variables are somewhat limited. For race, I use the following groups as categorized in the NVSS codebooks: Whites, Blacks, “American Indians (includes Aleuts and Eskimos)” (which I will refer to as “Native Americans”), and an aggregation of multiple categories for Asian-Americans and Pacific Islanders (“AAPIs”).¹⁸ Beginning in 1989, Hispanic origin is also recorded so I include complementary analysis of the non-Hispanic White population given the special attention paid to this demographic in the literature.

Education is reported in the NVSS data beginning in 1989; however, the education coding changed due to a 2003 revision which was not adopted universally so the old coding is used even in subsequent years. I construct two education categories which should be relatively consistent across this coding change: “no college” and “college,” defined as having attended any years of college. This coding is also used in Case and Deaton (2017).¹⁹ A small percentage of death certificates do not include a known education level. I examine this margin explicitly and generally interpret the results under the assumption that OxyContin’s introduction did not differentially affect unknown education status rates on death certificates. I focus on ages 25+ for the education analysis.

To form rates, I scale by population size using Medicare SEER population data.²⁰ They do not include education information, so I use information from the Current Population Study to impute population size by education status.

OxyContin Prescriptions

To measure OxyContin prescriptions, I use a restricted geocoded version of the Medical Panel Expenditure Survey (MEPS), accessed at the AHRQ Data Facility (Agency for Healthcare Research and Quality, 2018). The MEPS is a nationally representative survey of households and their medical providers, including medical and pharmaceutical claims. I constructed per capita OxyContin prescriptions for 1996-2016. For many demographic groups, the MEPS does not provide an adequate sample size, so I evaluate this supporting information for larger demographic groups only.

¹⁸The individual categories change over time. This aggregated category creates a consistent group.

¹⁹I use these categories because I cannot determine high school graduation status prior to the 2003 revision. Case and Deaton (2017) divide the “college” category further. Alternatively, Leive and Ruhm (2021a) and Leive and Ruhm (2021b) construct relative education groups based on within-year placement.

²⁰These data only include ethnicity beginning in 1990 so I use Census ethnicity data for 1989.

3.2 Empirical Analysis

I first show overdose death rates stratified by demographics to illustrate variation in the impact of the opioid crisis. While the literature has documented some of this heterogeneity for overdoses, it presents this information beginning in 1999 or, typically, even more recently. Truncating the data at 1999 or later limits our ability to inspect how relative demographic trends evolved and potentially flipped due to the opioid crisis. Moreover, less attention has been paid in the opioid crisis literature to the historical trends of some demographic groups, such as Native Americans (see Tipps et al. (2018); Whelshula et al. (2021) for important exceptions), who have been disproportionately affected by the growth in overdose death rates. I provide these time series analyses to establish important context when exploring OxyContin’s differential effects.

I also plot the difference in overdose rates between pairwise groups of particular interest in the literature. I provide these “overdose rate gaps” for both non-triplicate and triplicate states. Triplicate states were less exposed to OxyContin’s launch and the opioid crisis, providing evidence of how overdose rate disparities across demographics might have evolved in the absence of OxyContin.²¹ The non-triplicate states show how additional exposure to the opioid crisis altered these differences.

As a complementary exercise, I implement a difference-in-differences design, comparing changes in overdose rates in non-triplicate states to changes in triplicate states. I estimate the following specification:

$$y_{st} = \alpha_s + \gamma_t + \beta_t \mathbf{1}(\text{Non-Triplicate}_s) + \epsilon_{st}, \quad (1)$$

where y_{st} is overdoses per 100,000 people in state s and year t for the population of interest. This “event study” specification includes state and time fixed effects while permitting differential annual growth in the overdose rate for non-triplicate states (as defined in 1996), indexing the coefficient by t . I normalize $\beta_{1995} = 0$. β_t represents the differential overdose rate growth in non-triplicate states in year t (relative to 1995) and can be interpreted as the response resulting from additional exposure to OxyContin’s launch.

The pre-1996 coefficient estimates provide evidence about the existence (or non-

²¹In the absence of OxyContin, I cannot rule out that another addictive opioid might have been aggressively marketed at some point in time and generated large overdose effects. In this respect, this paper is a partial equilibrium analysis, using the launch of OxyContin as a proxy for exposure to the existing opioid crisis given the large differences in supply generated by its introduction.

existence) of pre-existing trends. In some cases, there is some evidence of systematic pre-1996 movements between non-triplicate and triplicate states. However, these rather small shifts rarely can explain the large post-1996 growth in overdoses. In addition, they are generally explained by differences in cocaine overdoses in the early-1990s due to the geography of the crack epidemic. I provide event studies excluding cocaine overdoses in the Appendix to illustrate that these pre-trend concerns typically disappear when cocaine is excluded, while the post-OxyContin effects are generally unaffected.

To summarize the event study results, I also present averages of the β_t estimates relative to 1991-1995 in Table A.1.²² I provide these aggregates for 1996-2000, representing the introduction of OxyContin and ramp up of marketing by Purdue Pharma; 2001-2010, representing the “first wave” of the opioid crisis; and 2011-2018, representing the second and third waves of the opioid crisis. I expect early OxyContin exposure to potentially have effects even as the opioid crisis transitioned to illicit opioids given that previous research has shown that reformulation, through geographic exposure to OxyContin, induced the transition to heroin (Alpert et al., 2018) and, later, to fentanyl (Powell and Pacula, 2021), increasing overdose rates. I also include averages for the entire 1996-2018 post-period.

I estimate equation (1) using weighted (by demographic population size) least squares. I use a wild bootstrap to construct 95% confidence intervals (Roodman et al., 2018). Since there are only five triplicate states, I use Webb et al. (2014) weights. This approach tests a series of null hypotheses and constructs confidence intervals based on the hypotheses that are not rejected. Given this process, the confidence intervals are not symmetric.

4 Results

4.1 Overdose Death Rates

Race/Ethnicity. In Panel A of Figure 2, I present overdose death rates by race: White Americans, Black Americans, Native Americans, and AAPIs. Overdose rates were generally highest for Black Americans before 1996, but White Americans experienced the sharpest growth since OxyContin’s launch. The Black population had a relatively flat overdose death

²²For the averages between period t_0 and t_1 , I use $\frac{1}{t_1-t_0+1} \sum_{t_0}^{t_1} \hat{\beta}_t - \frac{1}{5} \sum_{t=1991}^{1995} \hat{\beta}_t$. As should be evident from the event study results, the choice of pre-period does not meaningfully impact the results. I use 1991-1995 instead of 1995 since, in principle, a longer pre-period will make the estimates less susceptible to transitory pre-period shocks. Alternatively, I could use the full pre-period (1983-1995) and results are similar.

rate through the first two waves of the opioid crisis and appear to be generally less affected by the opioid epidemic until the fentanyl crisis, beginning in 2014. Native Americans have experienced a steep increase in overdose deaths throughout the opioid crisis. AAPIs had smaller levels of overdose rate growth.

In Panel B of Figure 2, I plot the differences in the overdose rates between White and Black Americans for non-triplicate states and triplicate states. Non-triplicate states experienced a substantially larger increase in this gap than triplicate states after 1996.²³ In fact, the White-Black overdose rate gap is *negative* for most of the post-period in triplicate states, which were less exposed to OxyContin’s launch, suggesting that OxyContin played a significant role in shifting racial disparities in overdose rates.

In the rest of Figure 2, I examine the differential trends in non-triplicate states relative to triplicate states by race and summarize the results in Table A.1. White and Native Americans were heavily impacted by the introduction of OxyContin through the entire sample period. I estimate statistically significant effects for AAPIs in some years, though the effect sizes are small when compared to other racial groups. The effect sizes for Black individuals are larger than those for AAPIs, but they are generally not statistically different from zero (the 1996-2018 average estimate is statistically significant at the 10% level). The annual estimates increase after 2013 for Black Americans. After 2013, I find large (though noisy) overdose rate increases in non-triplicate states relative to triplicate states.

I provide event study estimates when excluding overdoses involving cocaine in Figure A.3. There is much less evidence of any systematic pre-1996 movements, but the post-1996 estimates are generally similar (at least until 2014 when cocaine and fentanyl were often mixed). I typically observe stronger effects when focusing on overdoses involving opioids (see Figure A.4).

As a complementary analysis, I study non-Hispanic White Americans compared to the rest of the population, focusing on non-Hispanic White Americans given their prominence in the “deaths of despair” literature. This analysis is limited to 1989-2018 since ethnicity is not listed in the data before 1989. Event study estimates in Figure A.5 suggest large overdose death effects for non-Hispanic White Americans. There is less evidence for the rest

²³As with the event study estimates below, there is some evidence of small but systematic movements prior to 1996 when comparing triplicate to non-triplicate states, but these effectively disappear when cocaine overdoses are removed (while the post-1996 differences are generally unaffected). See Figure A.3, discussed below.

of the population (ignoring heterogeneity within this aggregate) until 2014, when we observe a large and statistically significant increase in non-triplicate states relative to triplicate states (consistent with non-triplicate states being more exposed to the fentanyl crisis). Difference-in-differences estimates are provided in Table A.1.

The permeation of fentanyl into illicit drug markets has increased overdoses involving non-opioid substances, and states more exposed to OxyContin were then more impacted by the heroin crisis, the fentanyl crisis, and the polysubstance crisis (Powell and Pacula, 2021). Thus, while some groups were impacted by the opioid crisis due to early medical access to OxyContin, other groups were less impacted until the crisis began to bleed into non-opioid drug markets. Overall, non-triplicate status, as a proxy for exposure to OxyContin’s launch, generated new and large overdose rate gaps based on race and ethnicity.

Sex. From 1999 to 2010, women experienced faster growth – proportional to their prior overdose rate – in overdose death rates than men (Mack et al., 2013). Panel A of Figure 3 shows that the overdose rate for women was flat prior to 1996 before steadily increasing since. Men also experienced a sharp and steady increase in overdose death rates that deviates from the pre-1996 trend.

Panel B of Figure 3 provides the male-female overdose rate gap by triplicate status. This evidence shows that the introduction of OxyContin exacerbated the gap over time. Triplicate states experienced little change in the male-female overdose death rate gap from 1993 through 2014; non-triplicate states experienced sharp (though uneven) growth.²⁴ Beginning in 2014, we observe evidence of large long-term effects as non-triplicate states were more exposed to the fentanyl crisis. While both men and women responded to the introduction of OxyContin, the evidence suggests that its launch had substantial effects on the male-female overdose gap, especially as illicit markets developed.

In Panels C and D of Figure 3, I present event study estimates stratified by sex. I observe a much stronger relationship between exposure to OxyContin’s launch and overdose rate growth among men. For the entire 1996-2018 period (see Table A.1), the annual overdose rate grew by an additional 6.7 overdoses per 100,000 for men in non-triplicate states relative to triplicate states, compared to an additional 3.4 for women. The event study patterns are similar when studying examining overdoses excluding cocaine (Figure A.6) or when limited to those involving opioids (Figure A.7).

²⁴As before, the trends between non-triplicate and triplicate states deviate somewhat in the pre-period, but this is primarily due to overdoses involving cocaine. See Figure A.6 for related evidence.

Education. The changing education gradient of mortality is a central theme of the “deaths of despair” analysis. There have been substantial differences in overdose rate growth between the “no college” and “some college” population, as shown in Panel A of Figure 4. Panel B shows how the difference in overdose death rates between these education groups has evolved in non-triplicate and triplicate states. Triplicate states have experienced a modest increase in this differential; however, the gap has increased by an order of magnitude more in non-triplicate states. The large differences in the education overdose rate gap between non-triplicate and triplicate states suggests that OxyContin’s launch – and opioid supply more generally – has played a substantial role in the growing life expectancy disparities by education status.

I present the corresponding event studies estimates stratified by education levels in Panels C and D of Figure 4. I observe much larger effects for the population without any college education. Over the 1996-2018 period, the additional exposure to OxyContin’s introduction due to non-triplicate status increased overdose rates by 10.4 deaths per 100,000 for those with no college education, compared to 2.5 for those with some college education or more (see Table A.1). This differential response generated substantial changes to the education gap in overdose death rates and the mortality rate gap more generally. Results are similar if I exclude overdoses involve cocaine or only study overdoses involving opioids (Figures A.8 and A.9).²⁵

4.2 Prescribing

Exposure to OxyContin’s launch substantially affected long-term overdose death rates across demographic groups. In this section, I test whether there are corresponding differences in prescribing for those same groups, which would further highlight the role of access and supply in explaining overdose death disparities. I limit this analysis to groups that have adequate sample sizes in the MEPS, though sample sizes in this section tend to be small even for large demographic groups so the results are noisier than those presented above. Figure 5 presents the results.

In the top portion of Figure 5, I show OxyContin prescriptions by triplicate status for White (Panel A) and Black (Panel B) Americans. There is a substantially larger gap

²⁵To test for the importance of missing education information in the NVSS, I study the relationship between triplicate status and the rate of missing education information in Figure A.10 and find no evidence of any systematic differences between triplicate and non-triplicate states over time.

between non-triplicate and triplicate states among the White population, which is consistent with the overdose rate results above.

I examine OxyContin prescribing by sex in Panels C and D of Figure 5. In most years and especially during the early years after OxyContin’s introduction, the differences between non-triplicate and triplicate states in prescribing was much larger for men.²⁶ This result is again consistent with the overdose death rate differences shown previously.

Finally, in Panels E and F of Figure 5, I examine OxyContin prescriptions by education level. The difference between non-triplicate and triplicate states is substantially larger for the no college group (note differences in y-axes), consistent with the overdose death rate findings above. Overall, groups that experienced larger responses in terms of overdose death rates to OxyContin’s launch also received higher rates of OxyContin prescribing (especially prior to reformulation). This finding is consistent with the importance of supply-side effects in generating the overdose rate gaps observed throughout this paper.

5 Discussion

The opioid crisis has engendered staggering demographic differentials in overdose rates. This paper provides evidence that these overdose differentials are supply-driven: we observe much larger overdose gaps between demographic groups in states more exposed to the opioid crisis due to an early and persistent supply shock. Consistent with this relationship, we observe similar differential patterns in OxyContin prescriptions. The overdose differentials persist even beyond the introduction of an abuse-deterrent version of OxyContin as states more exposed to OxyContin experienced sharper growth in their illicit opioid markets (Powell and Pacula, 2021). This transition to illicit markets has also begun to alter the demographics of the opioid crisis. While there is some evidence of differential trends prior to 1996, these are explained by eliminating overdoses involving cocaine or by just focusing on overdoses involving opioids.

The evidence that overdose differentials across demographic groups are substantially supply-driven does not rule out the importance of demand-side factors. It is possible that different populations have varying demands for substance use. In this case, an opioid supply shock would differentially impact these populations, suggesting that the interaction of

²⁶As discussed above in footnote 13, there is a notable dip in OxyContin prescribing in 2005-2006 due to a patent dispute at that time.

supply and demand factors is critical. However, the results do suggest that OxyContin, and opioid access more generally, played an independent and critical role in the development and persistence of demographic-specific trends. The literature provides limited evidence to explain the reasons why some demographic groups have been more impacted by the opioid crisis than others. This paper finds that OxyContin’s launch – and its enduring impacts – played a central role in the magnitudes of the overdose rate growth and the differences in that growth across demographic groups.

The results suggest that the increase in opioid access through OxyContin’s launch and general promotion of opioids generated new overdose deaths across demographic groups, rather than simply crowding out other types of drug overdoses. Thus, while “despair” due to poor economic and cultural conditions may have played a synergistic role, this supply-side shock was a necessary factor in inducing the additional drug overdose deaths that have defined the opioid crisis. These supply-side impacts appear to have affected nearly all demographic groups. There were some exceptions. I estimate only small increases for AAPIs; the estimates are large for Black individuals, but they are generally not statistically different from zero.

Future work should consider why some demographic groups were more strongly affected by the supply shock than others. This paper provides evidence of differential exposure to OxyContin since its introduction, but it is an open question why – for example – White Americans received higher rates of OxyContin prescribing than Black Americans, though the literature has suggested hypotheses (Om, 2018). However, given that there are such different responses to this major supply shock, this research suggests that studying the opioid crisis – especially the evaluation of supply-side interventions – merits separate analyses based on demographics.

Overall, few demographic groups escaped the supply-driven forces of the opioid crisis. This result is fundamental to understanding the underlying structural factors driving the opioid crisis across the population. Those structural factors may differ across groups, but the literature has generally not considered the role of supply-side shocks on overdose rate disparities. The launch of OxyContin and the accompanying aggressive promotion of strong opioids had extensive but disparate impacts across the population. These disparate impacts, in many cases, can explain existing differences in overdose rates across demographic groups. For example, in areas less exposed to OxyContin’s introduction, White Americans have typically had *lower* overdose rates than Black Americans since OxyContin’s launch

despite much larger overdose rates nationally (especially during the first wave of the opioid crisis). This result suggests that OxyContin flipped pre-existing racial overdose patterns. Similarly, the overdose rates of men relative to women grew faster in states more exposed to OxyContin's launch; the overdose rates of those with no college education were substantially higher relative to those with some college education in those same states. This evidence implies that OxyContin's launch and its enduring effects can largely explain the demographics of the overdose death rates in the United States.

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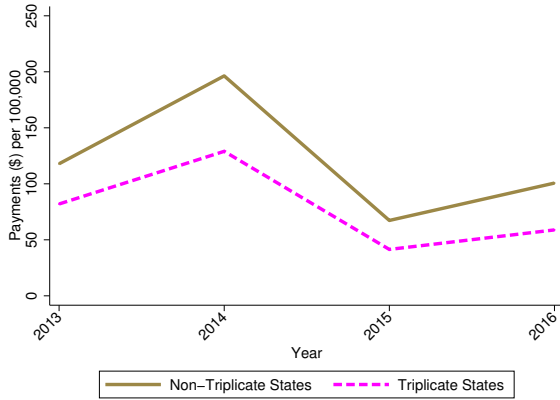
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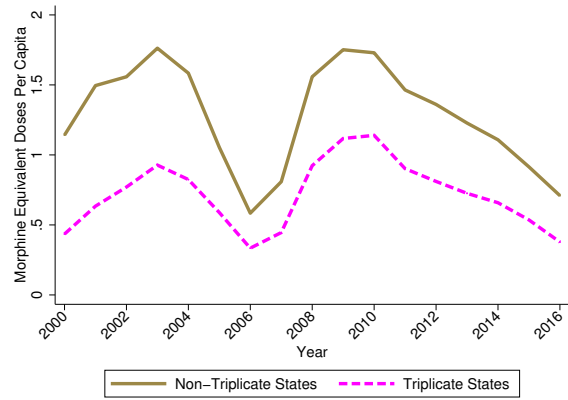
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Figures

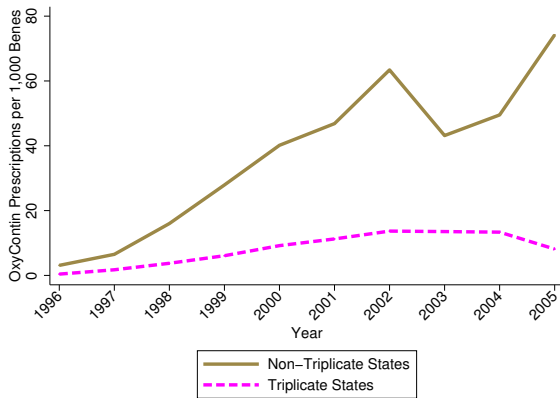
Figure 1: OxyContin Promotional Activity, Distribution, and Prescriptions by Triplicate State Status



A: OxyContin Promotional Payments



B: OxyContin Distribution (ARCOS)



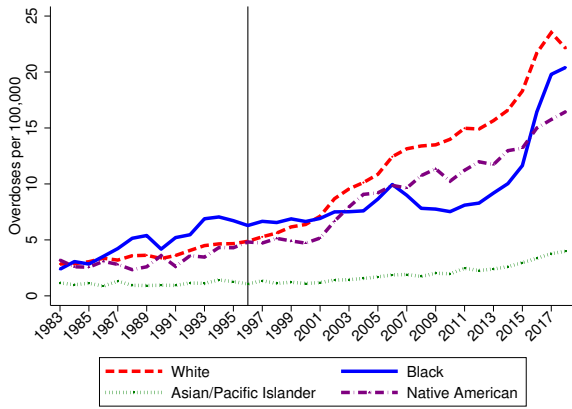
C: OxyContin Prescriptions (Medicaid)



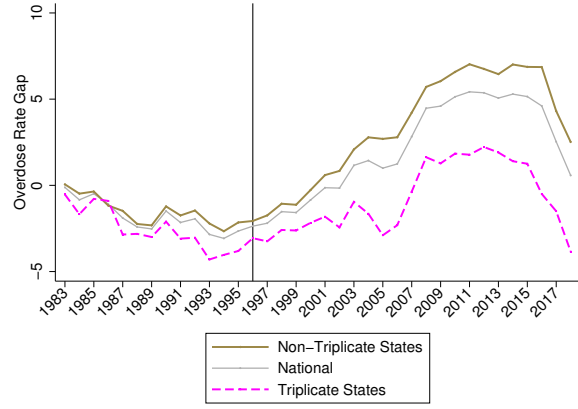
D: OxyContin Prescriptions (MEPS)

Notes: In Panel A, I use CMS Open Payments Data to calculate total payments and gifts made to physicians regarding OxyContin for the available years. I scaled this measure by population. The outcomes correspond to August 2013 – December 2016. Because the 2013 data only cover a partial year, I annualize the rate in that year. In Panel B, I use ARCOS data to construct OxyContin morphine equivalent doses per capita. I define a morphine equivalent dose as 60 morphine milligram equivalents. OxyContin data are only available for 2000-2016. In Panel C, I report the number of prescriptions per 1,000 beneficiaries from the Medicaid SDUD. I end this time series in 2005 due to the introduction of Medicare Part D. In Panel D, I report the number of prescriptions per 1,000 people in the MEPS using MEPS survey weights.

Figure 2: Overdose Death Rates by Race

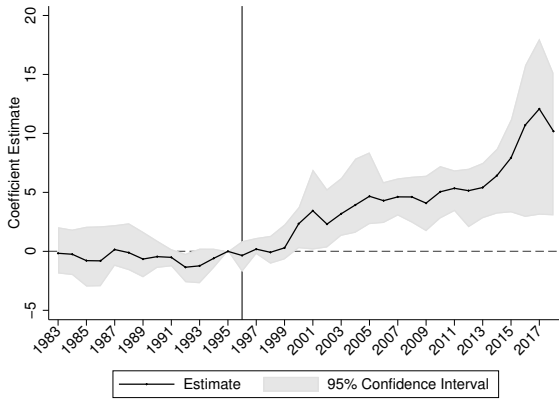


A: Time Series of Overdose Rates

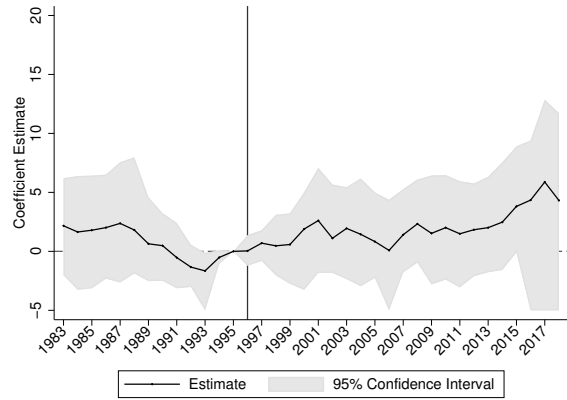


B: White-Black Overdose Rate Gap

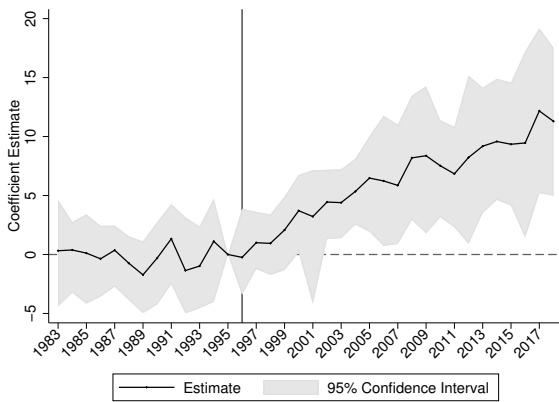
Event Study Estimates



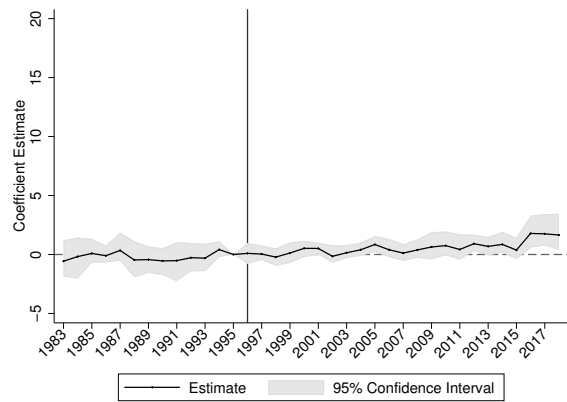
C: White



D: Black



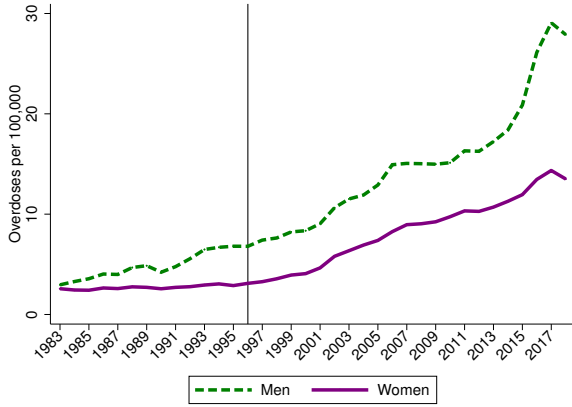
E: Native American



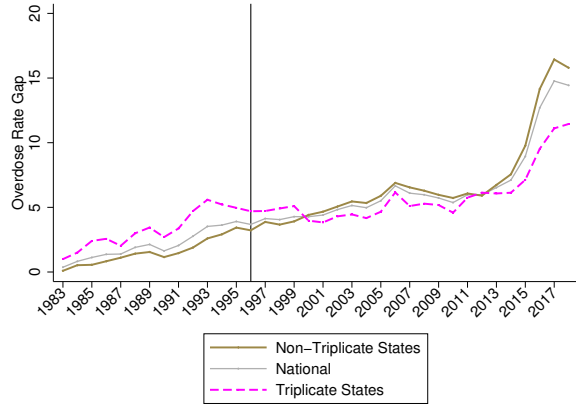
F: AAPI

Notes: I plot drug overdose deaths per 100,000 for 1983-2018 using NVSS data in Panel A. Panel B shows the difference between the overdose rates for White and Black Americans in triplicate and non-triplicate states. Panels C-F provide event study estimates from estimation of equation (1) in the paper. The model includes state and year fixed effects. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated using a clustered (by state) wild bootstrap.

Figure 3: Overdose Death Rates by Sex

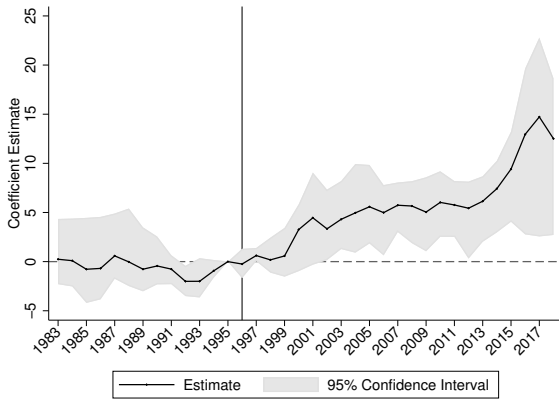


A: Time Series of Overdose Rates

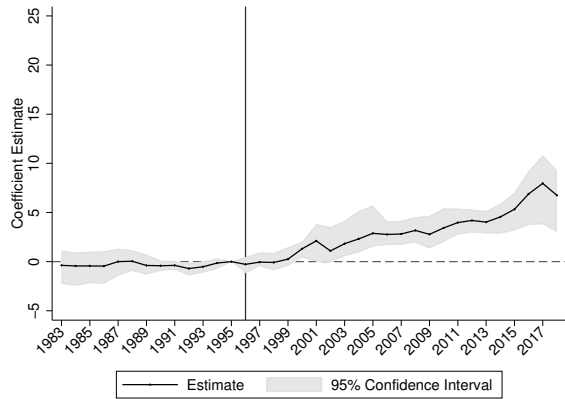


B: Men-Women Overdose Rate Gap

Event Study Estimates



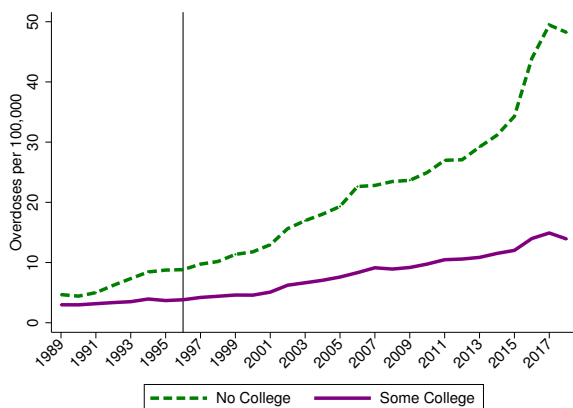
C: Men



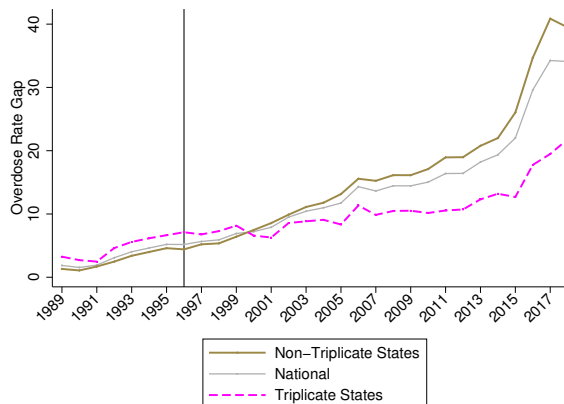
D: Women

Notes: I plot drug overdose deaths per 100,000 for 1983-2018 using NVSS data in Panel A. Panel B shows the difference between the overdose rates for men and women in triplicate and non-triplicate states. Panels C-D provide event study estimates from estimation of equation (1) in the paper. The model includes state and year fixed effects. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated using a clustered (by state) wild bootstrap.

Figure 4: Overdose Death Rates by Education

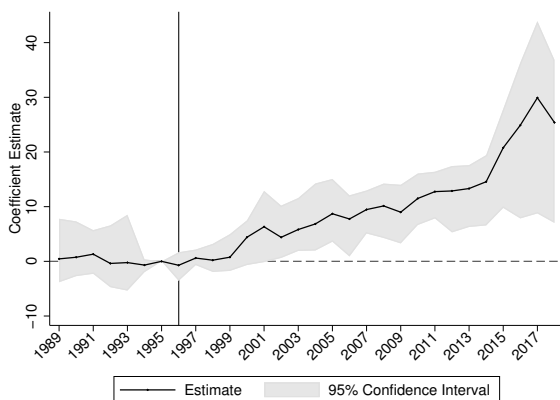


A: Time Series of Overdose Rates

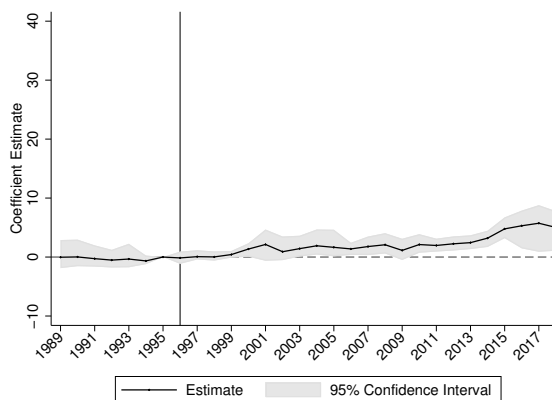


B: No College-College Overdose Rate Gap

Event Study Estimates



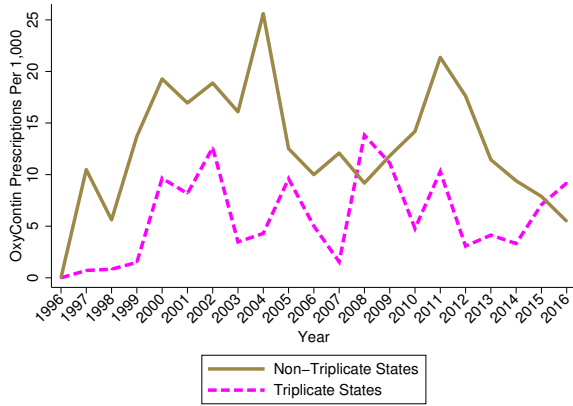
C: No College



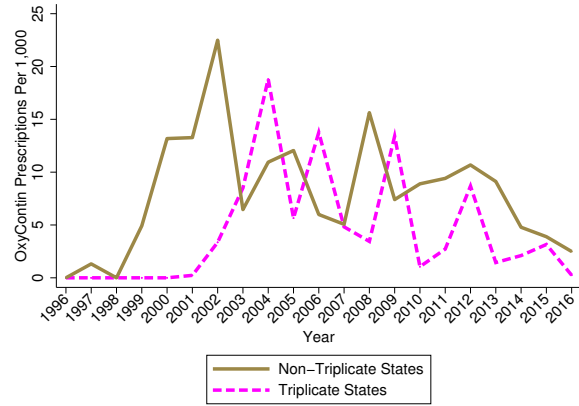
D: Some College

Notes: I plot drug overdose deaths per 100,000 for 1983-2018 using NVSS data in Panel A. Panel B shows the difference between the overdose rates for those with no college and those with some college in triplicate and non-triplicate states. Panels C-D provide event study estimates from estimation of equation (1) in the paper. The model includes state and year fixed effects. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated using a clustered (by state) wild bootstrap. Education information is only available beginning in 1989. “Some college” means at least some college education.

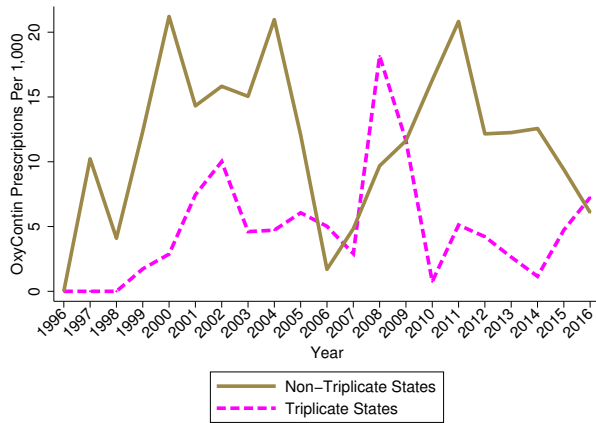
Figure 5: OxyContin Prescriptions by Triplicate Status



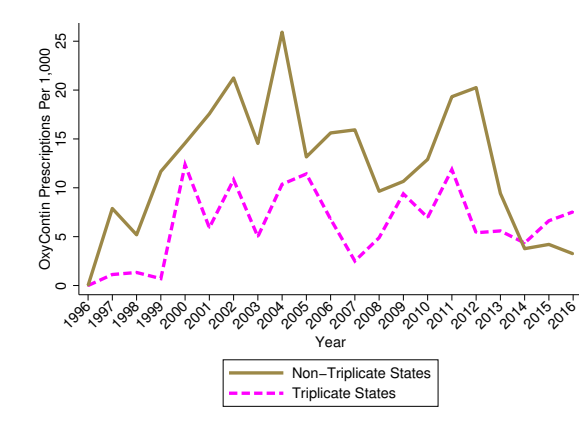
A: White



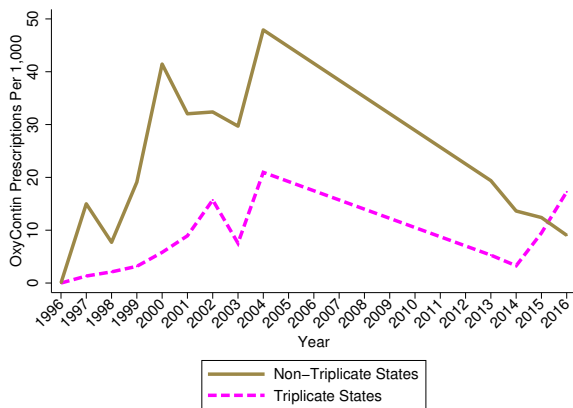
B: Black



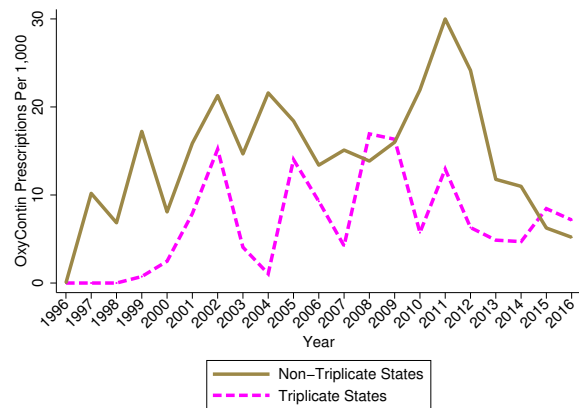
C: Men



D: Women



E: No College



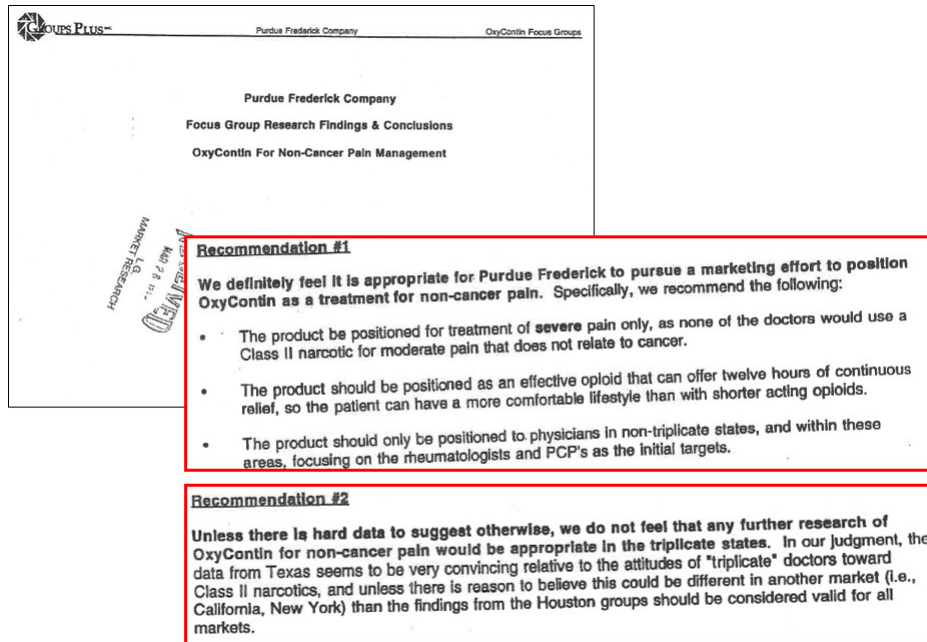
F: Some College

Notes: I report the number of prescriptions per 1,000 people in the MEPS using MEPS survey weights by triplicate status. I do not perform this analysis for all demographics discussed in the paper given small sample sizes.

A Appendix

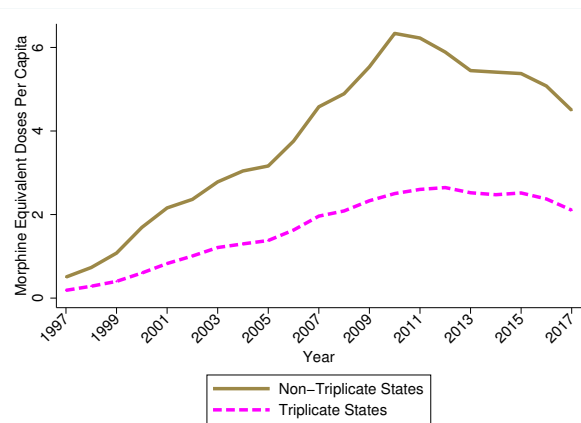
Figures

Figure A.1: Example of Purdue Pharma Focus Group Recommendations



Notes: This figure shows a copy of and relevant passage from Groups Plus (1995).

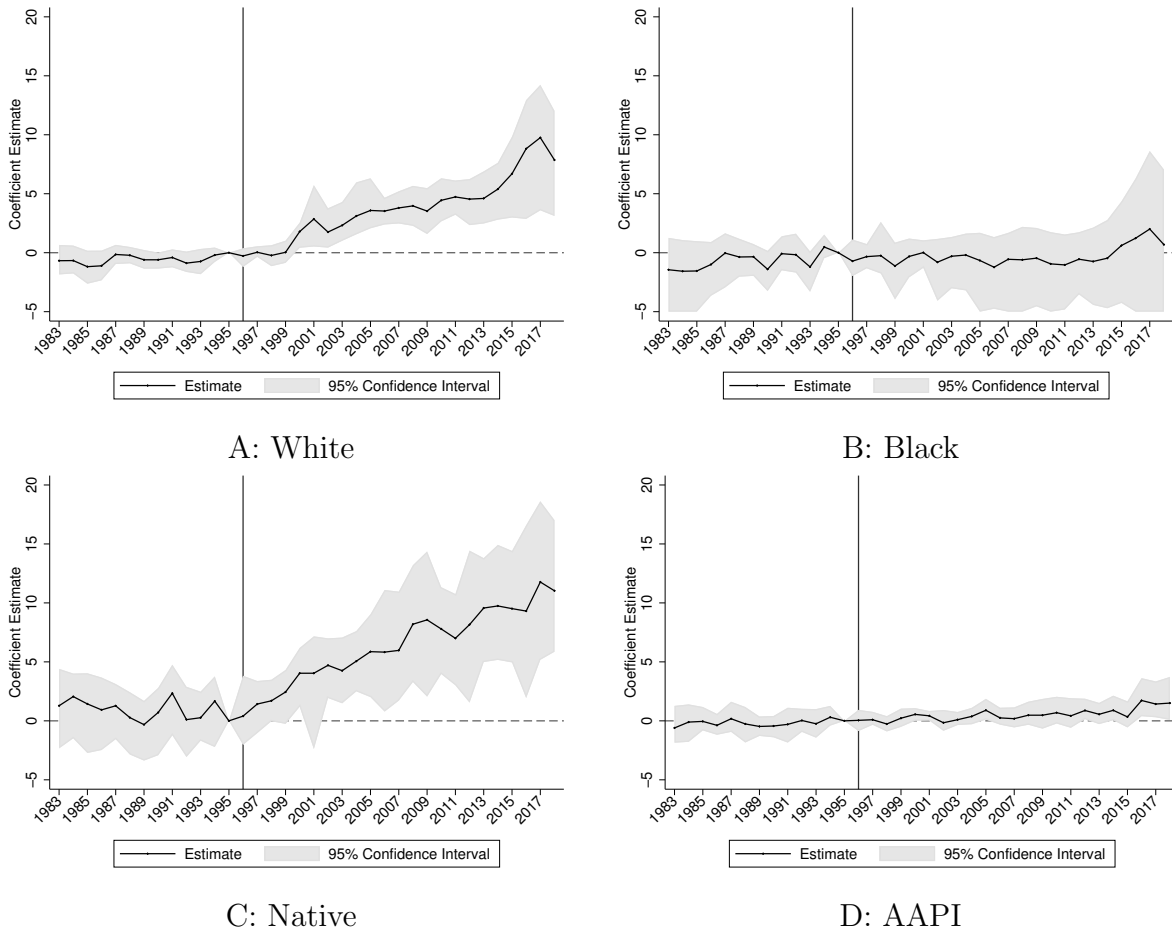
Figure A.2: Oxycodone Distribution Triplicate State Status



Oxycodone Distribution (ARCOS)

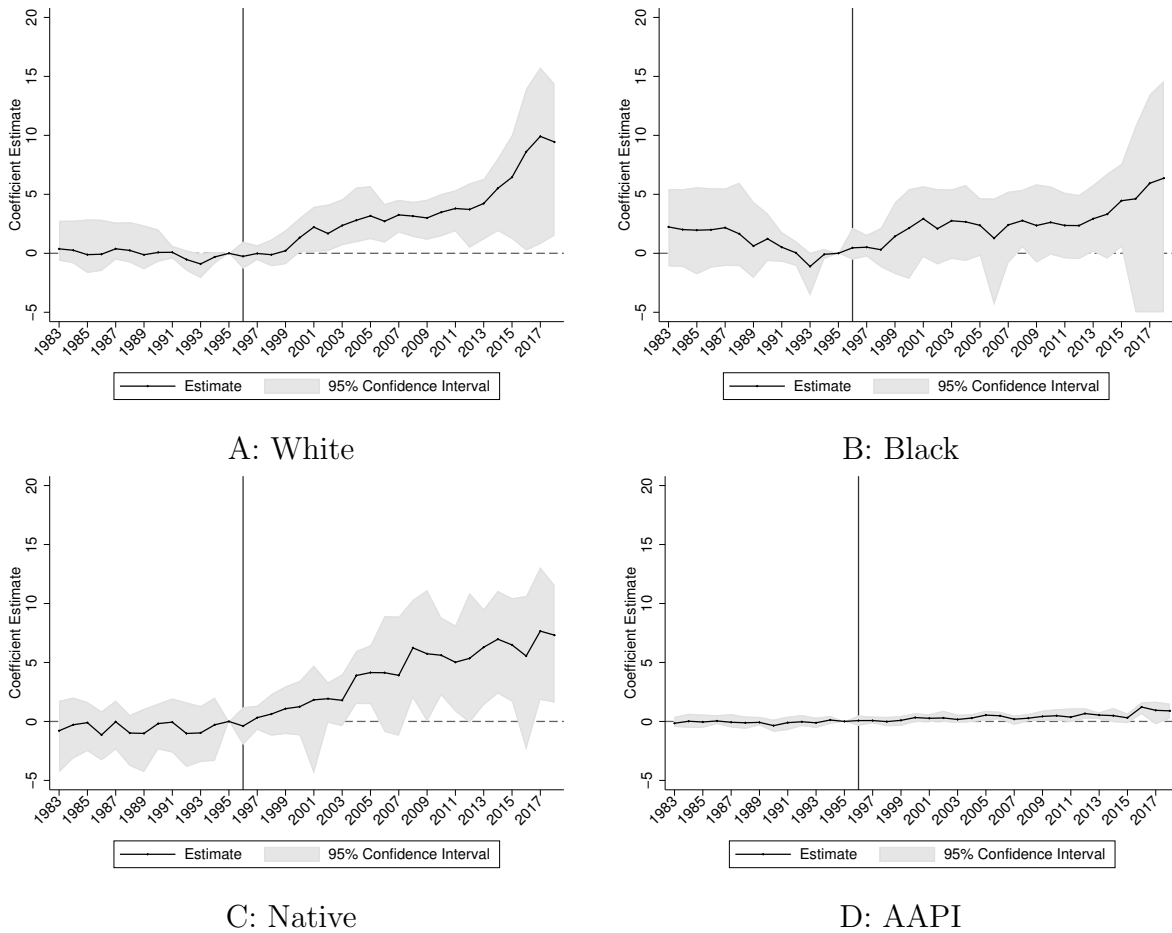
Notes: I use ARCOS data to construct oxycodone morphine equivalent doses per capita. I define a morphine equivalent dose as 60 morphine milligram equivalents. Oxycodone data are only available starting in 1997.

Figure A.3: Event Study Estimates by Race, Excluding Overdoses Involving Cocaine



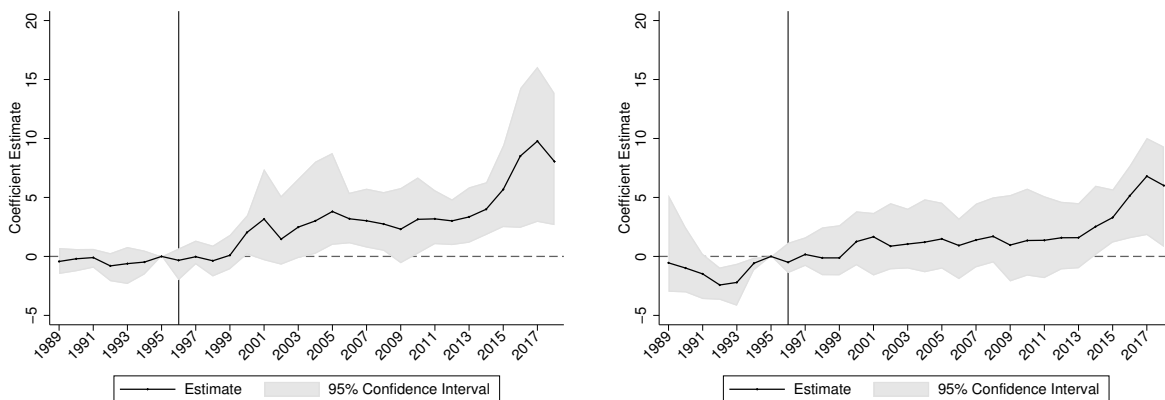
Notes: Each figure presents event study estimates from a regression of overdose deaths, excluding overdoses involving cocaine, per 100,000 for the listed demographic group on state fixed effects, year fixed effects, and Non-Triplicate \times Year interactions. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated by clustered (by state) wild bootstrap.

Figure A.4: Event Study Estimates by Race – Opioid Overdose Deaths



Notes: Each figure presents event study estimates from a regression of opioid-involving overdose deaths per 100,000 for the listed demographic group on state fixed effects, year fixed effects, and Non-Triplicate \times Year interactions. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated by clustered (by state) wild bootstrap.

Figure A.5: Event Studies: Overdose Death Rates for Non-Hispanic White Americans and the rest of the population

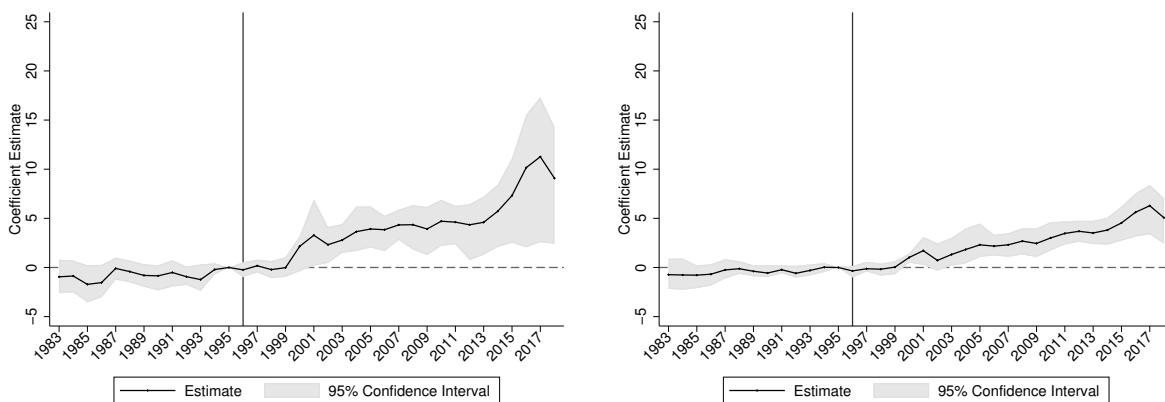


A: Non-Hispanic White

B: Rest of Population

Notes: Panels A and B provide event study estimates from estimation of equation (1) in the paper. The model includes state and year fixed effects. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the stated demographic group. 95% confidence intervals are estimated using a clustered (by state) wild bootstrap.

Figure A.6: Event Study Estimates by Sex, Excluding Overdoses Involving Cocaine

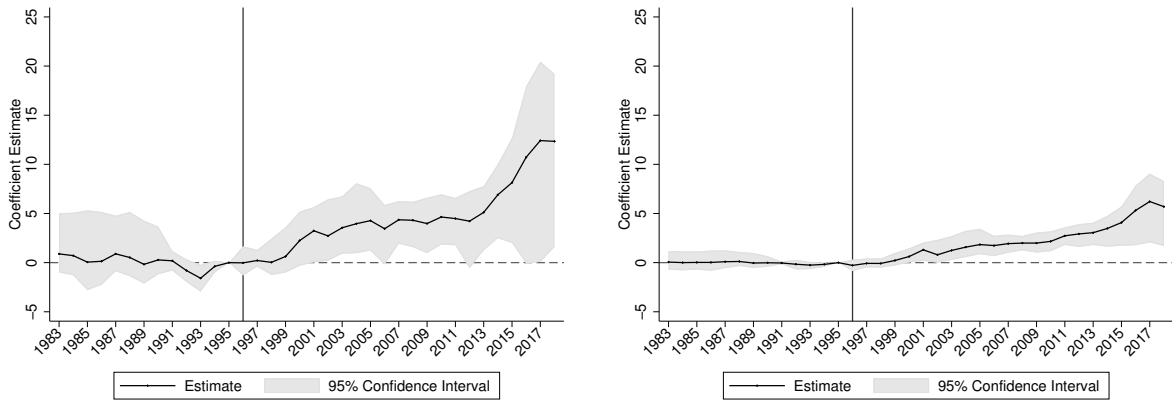


A: Men

B: Women

Notes: Each figure presents event study estimates from a regression of overdose deaths, excluding overdoses involving cocaine, per 100,000 for the listed demographic group on state fixed effects, year fixed effects, and Non-Triplicate \times Year interactions. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated by clustered (by state) wild bootstrap.

Figure A.7: Event Study Estimates by Sex – Opioid Overdose Deaths

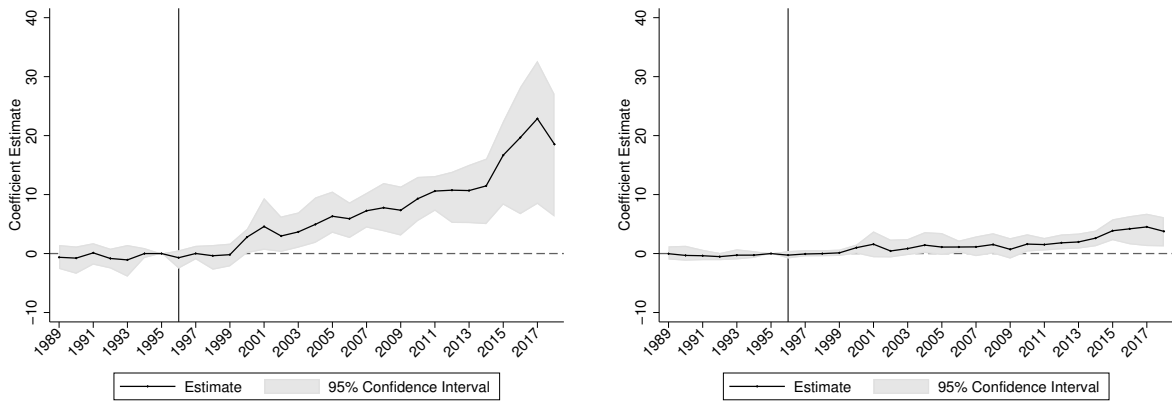


A: Men

B: Women

Notes: Each figure presents event study estimates from a regression of opioid-involving overdose deaths per 100,000 for the listed demographic group on state fixed effects, year fixed effects, and Non-Triplicate \times Year interactions. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated by clustered (by state) wild bootstrap.

Figure A.8: Event Study Estimates by Education, Excluding Overdoses Involving Cocaine

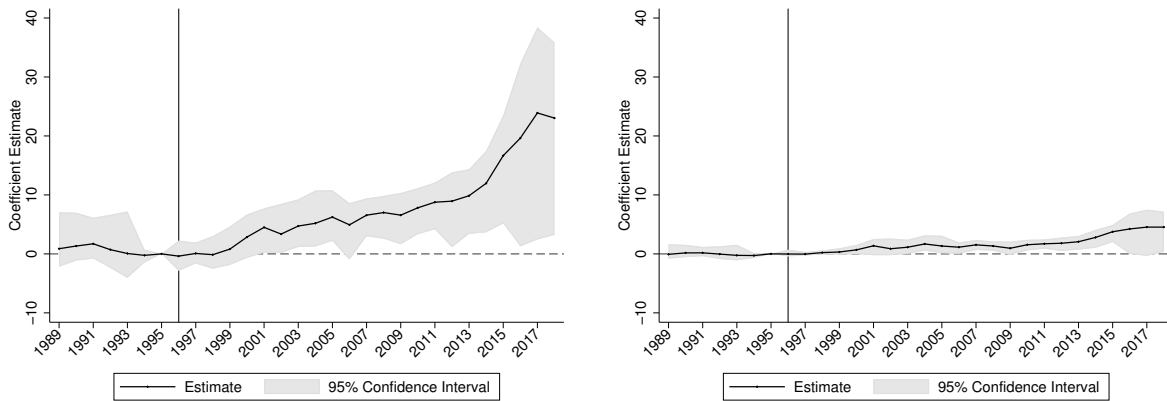


A: No College

B: Some College

Notes: Each figure presents event study estimates from a regression of overdose deaths, excluding overdoses involving cocaine, per 100,000 for the listed demographic group on state fixed effects, year fixed effects, and Non-Triplicate \times Year interactions. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated by clustered (by state) wild bootstrap.

Figure A.9: Event Study Estimates by Education – Opioid Overdose Deaths



A: No College

B: Some College

Notes: Each figure presents event study estimates from a regression of opioid-involving overdose deaths per 100,000 for the listed demographic group on state fixed effects, year fixed effects, and Non-Triplicate \times Year interactions. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the population size for the demographic group. 95% confidence intervals are estimated by clustered wild bootstrap. Ethnicity and education are not reported until 1989 so the sample period for those groups begins in that year. All others begin in 1983. The sample period ends in 2018.

Figure A.10: Overdose Death Event Study Estimates for Missing Education (1989-2018)



Notes: The outcome is the number of overdose deaths for the 25+ population which is missing education information in the NVSS scaled by the total 25+ population (multiplied by 100,000). I regress this outcome on state fixed effects, year fixed effects, and Non-Triplicate \times Year interactions. The estimates represent the differential change in the overdose rate in non-triplicate states. The 1995 coefficient is normalized to zero. Regressions are weighted by the size of the 25+ population. 95% confidence intervals are estimated by clustered (by state) wild bootstrap. Education is not reported until 1989 so the sample period begins in that year and ends in 2018.

Table

Table A.1: Difference-in-Differences Estimates

Non-Triplicate ×	Race				Race / Ethnicity	
	White	Black	Native	AAPI	White and Non-Hispanic	Rest of Population
1996-2000	1.213*** [0.460, 2.181]	1.542* [-0.144, 3.522]	1.476** [0.306, 2.990]	0.250 [-0.102, 0.486]	0.679** [0.082, 1.306]	1.474** [0.150, 4.842]
2001-2010	4.754*** [2.590, 6.494]	2.336 [-0.679, 6.312]	5.987** [2.534, 9.395]	0.543 [-0.392, 1.747]	3.234** [0.769, 5.860]	2.598* [-0.114, 6.629]
2011-2018	8.636*** [3.851, 11.858]	4.077 [-1.524, 8.573]	9.493** [4.557, 14.694]	1.194* [-0.101, 2.959]	6.092*** [2.811, 9.258]	4.876*** [2.154, 7.151]
1996-2018	5.335*** [3.284, 6.796]	2.769* [-0.212, 6.417]	6.226** [2.910, 9.558]	0.705 [-0.160, 1.897]	3.673*** [2.341, 4.947]	3.146*** [0.943, 6.165]
	Sex			Education		
	Non-Triplicate ×	Men	Women	No College	Some College	
	1996-2000	2.018*** [0.640, 3.666]	0.580*** [0.175, 1.177]	1.050 [-0.486, 2.302]	0.702** [0.007, 1.252]	
	2001-2010	6.149*** [2.557, 8.697]	2.868*** [1.640, 4.467]	7.989*** [4.132, 11.262]	2.010*** [0.800, 2.919]	
	2011-2018	10.435*** [4.044, 14.589]	5.806*** [3.600, 7.215]	19.306*** [8.288, 27.424]	4.194*** [1.539, 5.845]	
	1996-2018	6.742*** [3.258, 8.939]	3.393*** [2.454, 4.171]	10.417*** [5.427, 14.524]	2.485*** [1.326, 3.379]	

Notes: ***Significance 1%, **Significance 5%, *Significance 10%. Outcome is all drug overdose deaths per 100,000 for the listed demographic. I estimate an event study, conditioning on state and year fixed effects. The event study estimates refer to the additional overdoses experienced in non-triplicate states by year. I average these estimates for the years listed in the first column. Estimates are relative to the pre-period, 1991-1995. 95% confidence intervals reported in brackets are estimated by clustered (by state) wild bootstrap.