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Labor Market Effects of the Oxycodone-Heroin Epidemic*

David Cho, Daniel I. Garcia, Joshua Montes, and Alison Weingarden[†]

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Abstract

We estimate the causal effects of heroin use on labor market outcomes by proxying for heroin use with prior exposure to oxycodone, the largest of the prescription opioids with a well-documented history of abuse. After a nationwide tightening in the supply of oxycodone in 2010, states with greater prior exposure to oxycodone experienced much larger increases in heroin use and mortality. We find increases in heroin use led to declines in employment and labor force participation rates, particularly for white, young, and less educated groups, consistent with the profile of oxycodone misusers. The results show the importance of extending beyond prescriptions when accounting for the labor market effects of the opioid crisis.

Keywords: labor force; participation rate; opioid crisis

JEL: J21, I12, I18

*The views expressed in this paper are our own and do not reflect the views of the Federal Reserve System or its staff.

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Since 2000, the opioid crisis has claimed more than 480 thousand lives in the U.S.¹ While the tragic consequences on health and human life are salient, the labor market effects of the opioid crisis are still under debate. Much of the literature to this point has centered on the *prescription* opioid crisis. Since use of prescription opioids can have multiple causes, the aggregate effects on labor supply are not immediately obvious. Some prescription use could ameliorate pain and help workers remain in the labor force, while misuse could lead to addiction and major life disruptions including labor force exits. Also, some prescription use is transitory and without long-term consequences. Indeed, the literature that links prescription opioid use to labor supply outcomes finds mixed results. For instance, [Harris et al. \(2020\)](#) and [Aliprantis et al. \(2019\)](#) find large negative effects, while [Currie et al. \(2019\)](#) report modest positive effects for women.

We contribute to this literature by estimating the effects on the labor force participation rate (LFPR) and other outcomes of the transition from prescription opioid abuse to illicit opioids, particularly heroin. In contrast to the more ambiguous effects of general prescription opioid use, the labor supply effects of using heroin are likely negative. Still, the aggregate effects could be modest, if the number of heroin users is small and the effects of heroin use on LFPR are also small. However, we find the transition to heroin use has had meaningful, negative effects on the LFPR and the employment-to-population ratio (EPOP), particularly for white, young, and less educated cohorts, who were more likely to abuse prescriptions such as oxycodone.

As a starting point, and to motivate our analysis, we use data from the National Survey of Drug Use and Health (NSDUH) to assess recent increases in heroin users and the employment status of heroin users. According to the self-reported survey estimates, the number of individuals having used heroin increased by about 1.7 million to 5.4 million from 2007 to 2017, but the true figures could have been much larger given the tendency of individuals to underreport illegal drug use in surveys ([Harrell 1997](#); [Harrison 1997](#)). Indeed, the number of deaths from heroin overdoses increased fivefold during this period. We also find that heroin users are substantially less likely to be in the labor force. In particular, we estimate a linear probability model of labor force participation and show that heroin users are about 14 percentage points less likely to be in the labor force, a difference comparable to that between those without a high school degree and college graduates. Though suggestive, the NSDUH estimates are not causal, as unobserved individual characteristics could explain both low participation and heroin use.

To estimate the effects of the transition to heroin on labor market outcomes, we exploit variation from policy changes in 2010 that limited the supply of oxycodone – the largest of the prescription opioids with a well-documented recent history of abuse ([Hays 2004](#); [Cicero et al. 2005](#); [Katz and Hayes 2004](#)). In 2010, Purdue Pharma reformulated OxyContin, a popular brand of oxycodone, which impeded users from crushing or liquefying the pills for consumption ([Severtson et al. 2013b](#);

¹See “[Overdose Death Rates](#)” (National Institute on Drug Abuse; January 29, 2021)

Cicero and Ellis 2015).² Also in 2010, the DEA and local authorities began shutting down Florida “pill mills,” which had been boosting national supply (Surrat et al. 2013; Davis and Carr 2017;).³ Because of these developments, national manufacturer shipments of oxycodone have declined since peaking in 2010, after more than quadrupling in the 2000s. As supply tightened, many who were addicted to oxycodone turned to heroin (Cicero and Ellis 2015; Alpert et al. 2018 and Evans et al. 2019).

To causally estimate the effects of the increase in heroin use on labor market outcomes, we use an event-study design that exploits variation across states prior to 2010 in oxycodone use and the 2010 reduction in oxycodone supply, following the approaches in Alpert et al. (2018), Evans et al. (2019), and Powell and Pacula (2021). As documented by this literature, trends in heroin mortality prior to 2010 were similar across states with high and low oxycodone exposure. However, after 2010, heroin overdoses ramped up more in states with greater prior oxycodone exposure, reflecting an increase in demand for heroin. Also, deaths from fentanyl overdoses (a synthetic opioid), hepatitis-C cases, and other related negative outcomes also increased more in states with greater prior oxycodone exposure (Powell et al. 2019; Powell and Pacula 2021). Given these findings, we proxy for heroin and other illicit drug use after 2010 with oxycodone exposure prior to 2010.

We contribute to this literature by estimating meaningful and negative effects on EPOP and LFPR of the oxycodone-heroin epidemic. In our baseline specifications, we track the prime-age cohort in 2010, and account for differences across states in demographics, industry composition, and cyclical performance during the housing boom and Great Recession.⁴ The baseline estimates show EPOP and LFPR trended similarly in states with high and low oxycodone exposure prior to 2010. However, after 2010, states with previously higher oxycodone exposure experienced significantly larger relative declines in EPOP and LFPR. In areas with one standard deviation higher oxycodone exposure prior, EPOP and LFPR were on average about 30 basis points lower from 2011 to 2019 relative to the pre-period. These effects seem driven by behavioral changes related to greater heroin use rather than compositional effects, as we find little evidence of the latter. The unemployment rate estimates are small and not significant, indicating most of the effect of the increase in heroin use on employment occurs through the participation margin. We also find the increase in heroin use led to an increase in the rate of social security disability insurance (SSDI) beneficiaries – consistent with Park and Powell (2021) – and contributed to a modest change in the composition of employment towards part-time work.

²See, for example, “Origins of an Epidemic: Purdue Pharma Knew Its Opioids Were Widely Abused” (New York Times; May 29, 2018)

³See also “Feds Raid Pain-Clinics Suspected of Illegally Distributing Millions of Prescription Drugs” (ABC News; March 5, 2010) and “Florida Shutting ‘Pill Mill’ Clinics” (New York Times; August 31, 2011)

⁴In the baseline, we focus on people who were prime-age (25-54 year olds) in 2010, as this group faced a plausibly exogenous and unexpected shock to oxycodone supply. That said, we show that the estimates for the group of persons 25-54 years old each year are similar to the cohort approach.

We also estimate the employment and participation effects on different demographic groups using the same event-study framework with dependent variables aggregated by race and ethnicity, sex, education, and age cohorts. The EPOP and LFPR estimates are negative and significant for non-Hispanic whites (both male and female), those without a college degree, and younger cohorts. In contrast, the estimates are generally smaller and not significant for nonwhites, those with a college degree or higher, and the cohort of individuals aged 55-64 in 2010. These patterns are consistent with the profile of oxycodone abusers prior to 2010, who according to NSDUH estimates, were predominantly white, less educated, and younger. Despite these differences, we stress that these results do not suggest that nonwhites and those with a college degree have been unaffected. For nonwhites and college graduates, the main estimates are negative and the standard errors are large enough we can neither reject the hypothesis that they are zero nor large and negative. More broadly, death rates related to opioids have recently risen across demographic groups ([Drake et al. 2020](#)). Since 2016, mortality rates from synthetic opioids and heroin have increased more rapidly for Blacks and American Indian or Alaska Natives, reaching comparable levels as for non-Hispanic whites.

To gain a sense of the aggregate implications on LFPR for the prime-age cohort in 2010, we combine the baseline estimates with counterfactual assumptions in which all U.S. states had low oxycodone rates and were thus unaffected by the supply changes in 2010. Using different counterfactual assumptions, we find the oxycodone-heroin shock may have lowered LFPR between 25 and 40 basis points on average from 2010 to 2019. We also relate these magnitudes to the gap between the LFPR and the LFPR predicted by a demographics model. We find the oxycodone-heroin shock could explain between one sixth and one quarter of the gap implied by the demographics LFPR model. In sum, while the drag on LFPR from the increase in heroin use has been sizeable, it is modest compared to other LFPR drivers such as population aging and effects from the Great Recession.

We address various concerns with the identifying assumption that states with different exposure to oxycodone would have experienced similar labor market trends in the absence of the 2010 supply shock. As usual, we cannot directly test for this assumption. However, LFPR and EPOP pre-trends are flat in the baseline estimates, and the main estimates are similar with and without the various controls. Also, we show that the estimates are robust to alternative specifications, including other ways of controlling for differences across states in industry composition, demographics, and the severity of the Great Recession. We also address the possibility that the cyclical nature of drug use could explain some of the results, both by controlling for geographic variation in other painkillers as well as measuring oxycodone exposure in the early 2000s rather than at the end of the decade. In addition, the main findings are not likely related to the secular decline in manufacturing, as we show state-level manufacturing employment shares and oxycodone rates are uncorrelated. Finally, we show the labor market findings are similar when using alternative specifications and sample

restrictions.

There is a large literature on trends in LFPR and EPOP following the Great Recession. Many causes, both structural and cyclical, have exerted downward pressure on the LFPR (Aaronson et al. (2014), Council of Economic Advisers (2016), Montes (2018), Abraham and Kearney (2020)). The aging of the Baby Boom generation was an important structural factor while the depth of the financial crisis and falling demand for less-skilled workers also contributed to declining participation rates, particularly for prime-age, less-educated men. As posited by Krueger (2017), the opioid crisis could be an additional contributing factor. Most of the research on this front has estimated LFPR elasticities with respect to prescription opioid use, including Harris et al. (2020), Aliprantis et al. (2019), and Currie et al. (2019). Our main contribution is to instead estimate the effects from the oxycodone-heroin epidemic, similar to concurrent work by Park and Powell (2021). In addition, we present new NSDUH analysis and new findings on the heterogeneous labor market effects. We find the labor supply effects are stronger for white, less educated, and younger cohorts, in line with the profile of oxycodone misusers prior to 2010. Also, we report negative effects for both men and women, with the latter suggesting the Currie et al. (2019) findings of a positive effect on female LFPR from prescriptions may not take into account the oxycodone-heroin link.⁵

This paper is also related to the literature on policy interventions in the opioids market. Supply restrictions such as the 2010 OxyContin reformulation and some prescription drug monitoring programs (PDMPs) appear successful in reducing prescriptions (Buchmueller and Carey 2018; Grecu et al. 2019).⁶ These restrictions prevent some new prescription opioid use disorders from starting (Sacks et al. 2019), but can lead to higher demand for heroin and other drugs for those with existing disorders (Cicero and Ellis 2015; Currie and Schwandt 2020). For the latter, treatment via medications such as methadone and buprenorphine is helpful, but these treatments are underutilized (Fullerton et al. 2014; NIDA 2020; Jones et al. 2015). Rees et al. (2019) finds expanding access to overdose-reversing drugs such as naloxone lowers mortality rates, though Doleac and Mukherjee (2018) finds the moral hazard effects could outweigh the benefits.

1 Data and Descriptive Analysis

We use data from several sources. Our primary dataset that tracks opioid use is the Drug Enforcement Agency’s (DEA) Automation of Reports and Consolidated Orders System (ARCOS) for

⁵A growing literature examines the effects of the opioid crisis on broad outcomes including consumer finance (Jansen 2019), house prices (D’Lima and Thibodeau 2019), and firm value and investment (Ouimet et al. 2019).

⁶While some studies find PDMPs are generally effective in reducing opioid abuse, others find the average effects are modest (Meara et al. (2016); Moyo et al. (2017); Davis 2017). In addition, Horwitz et al. (2018) discuss data issues with PDMP studies that may mixed findings in the literature.

purchases of prescription opioids by pharmacies and other buyers from manufacturers. These data allow us to measure opioid distribution to retail pharmacies by state and over time, and we use these shipments of opioids to pharmacies as a proxy for opioid use. To measure deaths related to opioid use, we use of cause-of-death data from the National Vitality Statistics System managed by the Center for Disease Control and Prevention. The Current Population Survey (CPS) is our primary dataset that measures labor market outcomes, such as labor force participation, employment, and part-time work status by various demographic groups. In addition, we use American Community Survey data via the Census to measure geographic variation in demographic characteristics.

To supplement our analysis and to provide a descriptive motivation for our work, we use health-survey data to look at patterns of heroin use and the labor market attachment of those with and without nonmedical use of prescription pain medications and heroin. To track those patterns, we use the National Survey of Drug Use and Health, NSDUH, which has been conducted by the federal government since 1971 to measure drug use, addiction, and mental health issues. NSDUH is an annual, nationally representative survey of persons aged 12 and over in the civilian non-institutionalized population of the U.S.⁷ The survey contains information on various individual characteristics, including labor force status, education, and demographics such as age and race and ethnicity. Among other questions, respondents are asked if they have ever used drugs such as heroin and if they have done so recently. Though individual survey responses are anonymized and confidential, population estimates of illegal drug use from NSDUH are likely undercounts, given the well-known tendency of individuals to underreport drug use especially heroin and other illegal substances (Harrell 1997; Harrison 1997; Colón et al. 2001; Morral et al. 2000). Moreover, the survey misses some high-risk groups, such as those imprisoned and without a home or shelter.

We begin by measuring the number of heroin users based on NSDUH data. As shown in the left panel of figure 1, the number of adults who recently used heroin increased from about 350 thousand to 900 thousand between 2007 and 2017. Over the same period, the number of adults with prior heroin use increased from about 3.7 million to 5.4 million. Although these are notable increases, they are likely conservative, given the noted measurement issues.⁸ Indeed, as the right panel of figure 1 shows, the dramatic rise in deaths from heroin overdoses – which increased fivefold from 2007 to 2017 – suggests the increase in users could have been much larger.⁹ That said, some of the rise in mortality could also reflect riskier consumption practices.

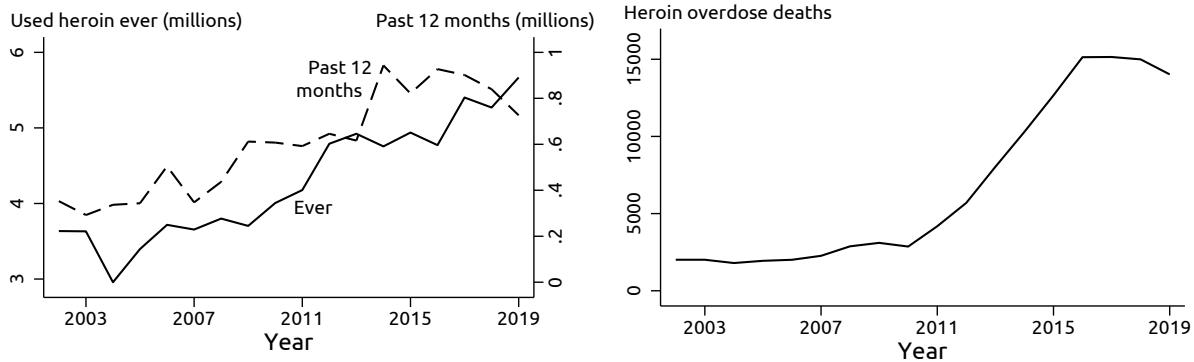
Our analysis using the NSDUH data suggest that heroin users are much less likely to be in the labor force. Table 1 reports selected coefficient estimates from a linear probability model of labor

⁷See <https://www.samhsa.gov/data/data-we-collect/nsduh-national-survey-drug-use-and-health> for more information.

⁸See also “The federal government is systematically undercounting heroin users” (Washington Post; August 22, 2017)

⁹Ruhm (2018) argues deaths from heroin overdoses are also likely understated.

Figure 1: Trends in heroin use and mortality



Source: National Survey of Drug Use and Health (left) and the National Vital Statistics System (right) for heroin overdose deaths (T40.1).

force participation, which controls for various demographic, education, and location characteristics. The first column estimates a baseline model without considering drug use, and shows that those with a college degree or higher are about 16 percentage points more likely to be in the labor force than those without a high school degree (the omitted group).¹⁰ The second column estimates show that those who have abused pain medication within the past year, including prescription opioids, are about one percentage point less likely to be in the labor force. More strikingly, the model estimates in the third column show that recent heroin users are about 14 percentage points less likely to be in the labor force—a magnitude comparable to the coefficient on the college degree or higher indicator. Given the likely undercount of heroin use, the coefficient estimate on heroin use could be attenuated. That said, we stress that these results, while suggestive, are not causal for many reasons, including the possibility that some omitted variables may help explain both heroin use and labor force status.

¹⁰Estimating a similar linear probability model in column 1 of table 1 in the CPS yields similar coefficients estimates.

Table 1: Probability of being in the labor force for people ages 24 to 49, 2002-2014

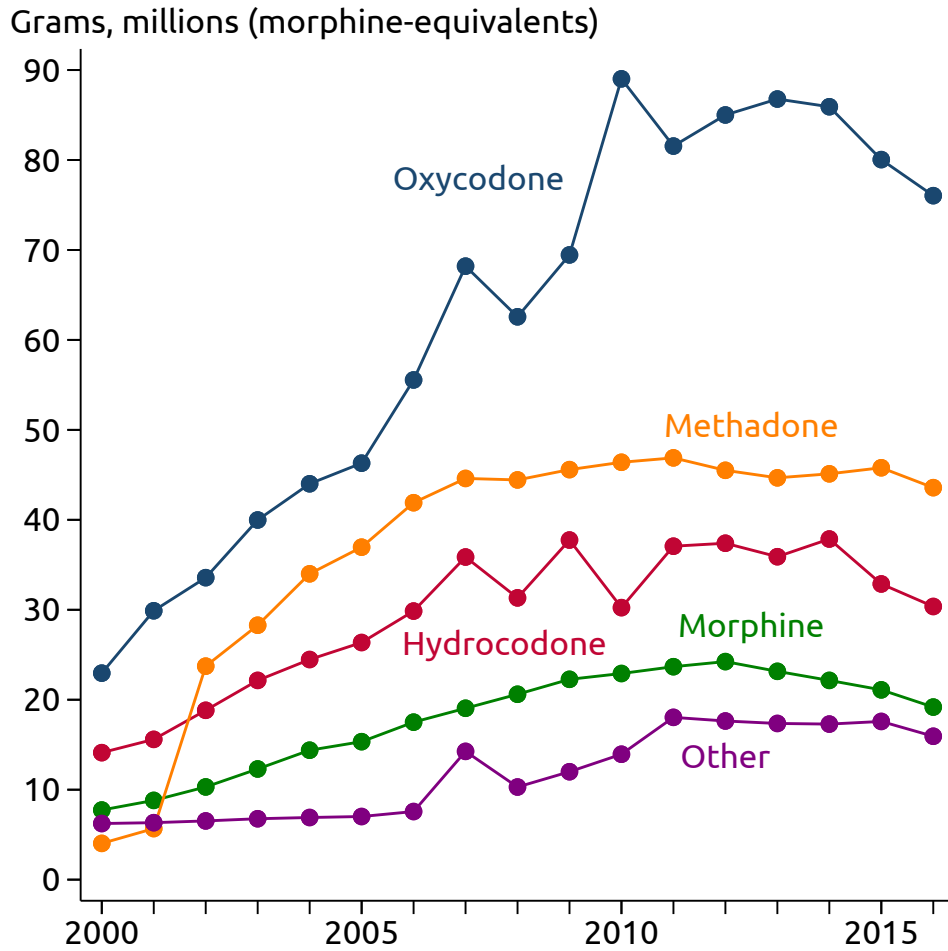
LF=100 or 0	Baseline Coef./SE	Pain Meds Coef./SE	Heroin Coef./SE
High school	9.93*** (0.41)	9.92*** (0.41)	9.89*** (0.41)
Some college	13.48*** (0.41)	13.46*** (0.41)	13.43*** (0.41)
College plus	15.93*** (0.41)	15.89*** (0.41)	15.84*** (0.41)
Abuse pain meds		-1.00** (0.48)	
Heroin user			-14.30*** (2.32)
All other controls	Yes	Yes	Yes
R-squared	0.06	0.06	0.06
Observations	238267	238267	238267

Note: The explanatory variables include education, race and ethnicity, sex, age, marriage, year, and metro area location fixed effects.

Source: National Survey of Drug Use and Health and authors' calculations.

To isolate plausibly exogenous variation in heroin use, we exploit 2010 policy changes that limited the supply of oxycodone and which had the unintended consequence of expanding demand for heroin (Cicero and Ellis 2015; Alpert et al. 2018 and Evans et al. 2019). Oxycodone is the powerful primary ingredient in painkillers such as OxyContin, Percocet, and Roxicodone. Before 2010, shipments and use of oxycodone varied significantly by state. To measure oxycodone and other opioids, we use data from the DEA's ARCOS database, which tracks shipments of controlled substances (by weight of the active ingredient) from manufacturers to buyers such as pharmacies, practitioners, and universities. Figure 2 shows oxycodone is the largest of the prescription opioids and contributed considerably to the 2000s expansion in prescription opioids.

Figure 2: Shipments of oxycodone and other opioids



Source: ARCOS data.

Table 2 provides basic characteristics of persons who self-report in NSDUH having used OxyContin for nonmedical reasons within the past year. Recent nonmedical users were much more likely to be white, younger, and without a college degree relative to those without nonmedical use. There are also differences by sex and location, though those are less stark.¹¹

¹¹Note that, in the 2000s, NSDUH did not include an analogous question of recent nonmedical use for broader oxycodone products.

Table 2: Characteristics of persons self-reporting nonmedical use of OxyContin, 2006-2009

Percent who are:	Nonmedical use within past year?	
	Yes	No
White (non-Hispanic)	87.15%	68.84%
Younger than 50	91.19%	59.34%
College degree or higher	10.53%	27.80%
High school degree or less	58.04%	46.78%
Male	61.27%	48.18%
Live in metro area	81.88%	83.30%
Live in large metro area	47.97%	53.53%

Source: NSDUH, for persons 18 years of age and older.

In the face of the growing oxycodone crisis, two key developments curbed the supply of oxycodone in 2010. First, Purdue Pharma reformulated OxyContin, a popular brand of oxycodone that had been commonly abused. The new abuse-deterrent formulation successfully impeded many users from crushing and dissolving the pill for non-oral consumption (Severtson et al. 2013b; Severtson et al. 2013a; Cicero and Ellis (2015)). OxyContin had been designed to gradually release large quantities of oxycodone to treat pain over an extended period. However, users crushed or solubilized the pills for a quick and potent release of the oxycodone (Hays 2004; Cicero et al. 2005; Cone et al. 2003).

In addition to the reformulation of OxyContin, the DEA and local enforcement began to shut down Florida ‘pill mills’ –clinics with lax standards where physicians prescribed large quantities of oxycodone and other prescription drugs often to out-of-state buyers in 2010. (Surrat et al. 2013; Davis and Carr 2017; Temple 2015). Returning to figure 2, national oxycodone shipments more than quadrupled in the 2000s, and have declined since peaking in 2010.

As the supply of oxycodone tightened, many who were addicted to oxycodone turned to heroin as a substitute (Cicero and Ellis 2015; Alpert et al. 2018 and Evans et al. 2019).¹² Indeed, deaths from heroin overdoses began to step up shortly after 2010, as documented by Evans et al. (2019) and Alpert et al. (2018). Similar to their approach, we measure state-level oxycodone exposure as the ratio of the oxycodone grams shipped to the state’s chain and retail pharmacies to the state’s

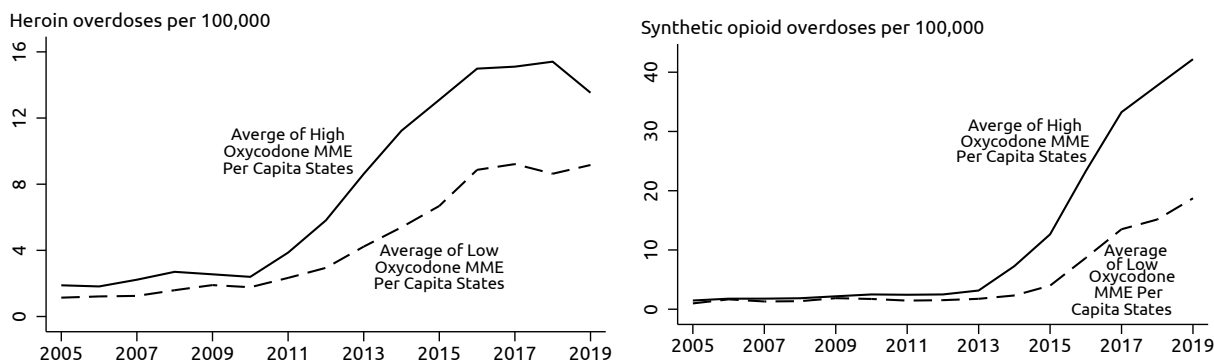
¹²Cicero et al. (2012) report little substitution specifically on the OxyContin reformulation into other prescriptions such as hydrocodone. At the time, hydrocodone brands including Vicodin contained other ingredients such as acetaminophen, which deters some abuse as high doses of acetaminophen are highly toxic.

prime-age population, both averaged over 2006 to 2009.¹³

$$OxyRate_s^{pre} = \frac{Oxycodone_s^{2006-2009}}{Prime\text{-}age\ population_s^{2006-2009}} \quad (1)$$

Figure 3 shows that trends in heroin and synthetic opioids (largely fentanyl) death rates prior to 2010 were similar across states with high and low oxycodone exposure. After 2010, heroin death rates rose nationally, but had a stronger increase in states with high oxycodone exposure (left panel). Throughout the 2010s, deaths from heroin overdoses have been higher in states with higher oxycodone rates, though the gap appears to be closing somewhat in recent years. For synthetic opioids, overdoses trended similarly in both groups and increased more in states with greater oxycodone exposure prior to 2010. Fentanyl deaths began rising a few years after 2010 likely as it took some time for suppliers to innovate (Park and Powell 2021; Pardo et al. 2019). These results reaffirm previous findings by Alpert et al. (2018) and Evans et al. (2019) for heroin overdoses and Powell and Pacula (2021) for fentanyl and cocaine overdoses.¹⁴

Figure 3: Overdose death rates by pre-2010 oxycodone exposure

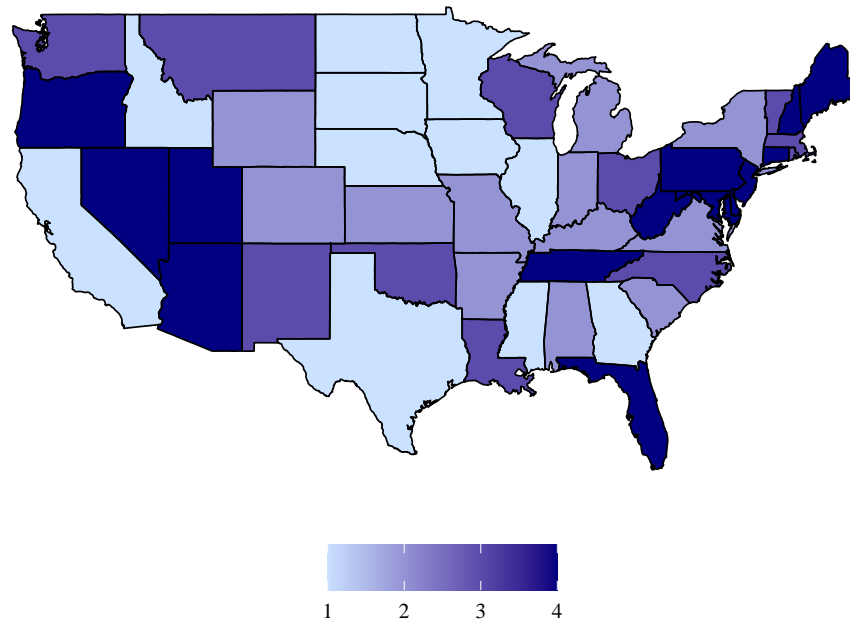


Note: Death rates relative to the prime-age population. Source: ARCOS for state oxycodone rates; National Vital Statistics System for heroin (T40.1) and synthetic opioid (T40.4) overdose deaths.

¹³Evans et al. (2019) measure is based on the public state-level aggregates of ARCOS data, while our measure is based on the DEA’s ARCOS microdata released by the Washington Post and the Charleston Gazette-Mail of West Virginia. Though in practice the measures are similar, the microdata allow us to exclude manufacturer shipments to buyers such as mail-order pharmacies, practitioners, and universities, which are less likely to use the shipments to fill prescriptions for in-state residents. Alpert et al. (2018) measure is based on state variation in NSDUH estimates of the fraction of OxyContin nonmedical users. They find the NSDUH measure is highly correlated with oxycodone supply measure from ARCOS.

¹⁴In addition, Powell et al. (2019) and Beheshti (2019) find evidence of other negative health effects: hepatitis-C infection rates have increased rapidly since 2010, and the increase has been stronger in states with greater pre-2010 exposure to oxycodone.

Figure 4: Geographic variation in oxycodone rates, 2006-2009



Note: Quartiles of oxycodone rates (mme per capita) with 4 indicating the top quartile. Source: ARCOS.

Figure 4 maps quartiles of oxycodone rates prior to 2010. On average, oxycodone rates were highest in the East, comparable in the South and West, and lowest in the Midwest, though there is significant dispersion within regions as well. States in the top quintile of exposure include states with very high heroin mortality rates, such as CT, WV, and NJ.

What explains the geographic dispersion in oxycodone rates? [Alpert et al. \(2019\)](#) find that early differences in the supply and marketing of oxycodone have had persistent implications. Indeed, the aggressive marketing tactics used by oxycodone manufacturers such as Purdue Pharma and Mallinckrodt are well-documented ([Van Zee 2009](#); [Quinones 2015](#); [Kolodny et al. 2015](#)).¹⁵ Relatedly, differences in physician incentives and quality could explain some of the geographic dispersion ([Schnell and Currie \(2018\)](#)).

Another possibility is that poor economic conditions may have contributed to poor health outcomes including both higher deaths ([Case and Deaton 2017](#); [Pierce and Schott 2020](#)) and the prescription crisis ([Hollingsworth et al. 2017](#); [Charles et al. 2018](#)). Specifically, the positive correlation between manufacturing employment shares and CDC opioid prescription rates suggests lower economic opportunities could partly explain the prescription crisis. However, oxycodone rates are weakly (and negatively) correlated with manufacturing employment shares. Hence, the oxycodone-heroin link

¹⁵See also “[Purdue Pharma Pleads Guilty to Criminal Charges for Opioid Sales](#)” (NYT; Dec 17, 2020) and [Mallinckrodt Reaches 1.6 Billion Deal to Settle Opioid Lawsuits](#) (NYT; Feb 25, 2010)

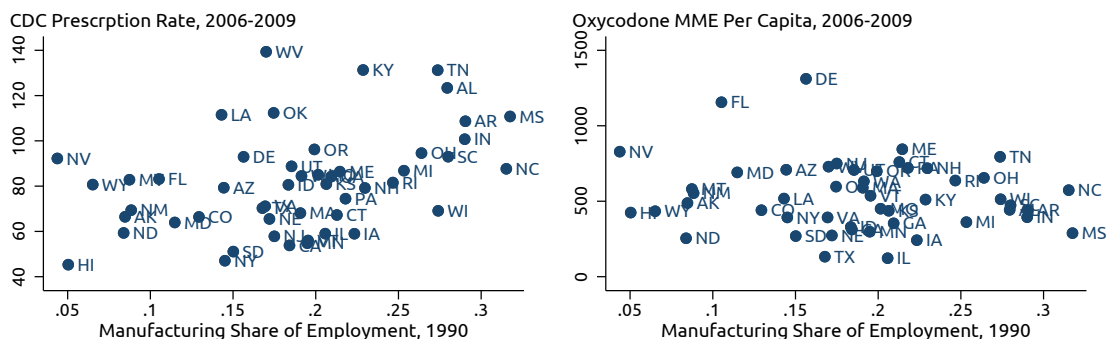
is unlikely driven by manufacturing exposure. The positive association between CDC prescription rates and manufacturing seems to be driven by other prescription opioids such as hydrocodone, as shown in 3 and figure 5.¹⁶

Table 3: Correlations of state manufacturing shares and prescription opioid indicators

	Manufacturing share 1990	CDC RX rate 2006-2009	Hydrocodone rate 2006-2009	Oxycodone rate 2006-2009
Manufacturing Share 1990	1.000			
CDC RX Rate 2006-2009	0.402	1.000		
Hydrocodone Rate 2006-2009	0.294	0.842	1.000	
Oxycodone Rate 2006-2009	-.115	0.301	-.0425	1.000

Source: QCEW for manufacturing employment shares; CDC for prescription rates; and ARCOS for hydrocodone and oxycodone rates.

Figure 5



Source: QCEW for manufacturing employment shares; CDC for prescription rates; and ARCOS for hydrocodone and oxycodone rates.

2 Empirical Framework

We estimate the effects of the increase in heroin use on labor market outcomes by proxying for heroin use with exposure to oxycodone prior to 2010. We use this proxy since reliable measures of heroin use are not available, and previous research has shown prior exposure to oxycodone is

¹⁶For more discussion on the extent to which changes in drug supply or economic conditions have contributed to the increase in mortality, see [Ruhm \(2018\)](#) and [Currie and Schwandt \(2020\)](#)

strongly predictive of increases in mortality from opioids such as heroin and fentanyl. Moreover, oxycodone exposure is also predictive of other related public health issues, including higher incidence of hepatitis-C (Powell et al. 2019) and drug overdoses from cocaine, often fentanyl-laced (Powell and Pacula 2021). Thus, exposure to oxycodone proxies for both increases in heroin use and other related negative public health outcomes caused by the decline in oxycodone supply.

Specifically, we employ a difference-in-difference framework exploiting variation in states’ exposure to oxycodone prior to 2010 interacted with year effects, according to equation 2. Our estimates show differences in how labor markets evolved as explained by variation in their pre-2010 exposure to oxycodone. This approach is similar to that of Alpert et al. (2018), Evans et al. (2019), Powell and Pacula (2021), and Park and Powell (2021) who show the 2010 shock to oxycodone supply led to higher demand for heroin and heroin overdoses.

$$Y_{st} = \alpha_t + \gamma_s + \delta_t \times OxyRate_s^{pre} + \omega X_{st} + \epsilon_{st} \quad (2)$$

State-level dependent variables Y_{st} include EPOP, LFPR, and other indicators including death rates from heroin and synthetic opioids (mainly fentanyl). We interact $OxyRate_s^{pre}$, the average oxycodone rate over 2006-2009, scaled to have mean zero and standard deviation of 1, with a full set of year fixed effects. The model includes year and state fixed effects, α_t and γ_s , respectively, to account for both national trends and fixed differences across states. X_{st} include different cyclical and structural labor market characteristics: the 2010 white, male, college graduate, and foreign-born population shares, each interacted with a full set of year fixed effects; the lagged employment share in the service industry; and house price and unemployment rate changes from 2003 to 2006 and 2006 to 2009, each interacted with a full set of year fixed effects.¹⁷ Standard errors are clustered at the state-level to account for serial correlation. In the baseline, we include the 50 U.S. states and the District of Columbia and use regressions weighted by population.

The main coefficients of interest are estimates of δ_t , which are shown graphically with the 2009 coefficient normalized to zero. These estimates show the causal effect of the oxycodone-heroin shift on the labor market, conditional on the identifying assumption that labor markets would have evolved similarly across states absent the oxycodone supply change in 2010. As usual, this assumption is not testable. However, pre-trends in our baseline estimates are flat, and the main estimates with and without the various controls are similar. In addition, the estimates are robust to different specifications including other ways of accounting for state-level variation in the severity of the Great Recession, industry composition, other drug use, region trends, and other controls.

¹⁷Data come from the American Community Survey via the Census Bureau; the Quarterly Census of Employment and Wages via Bureau of Labor Statistics for industry composition; CoreLogic, Inc., Private-Label Loan, Home Equity Servicing and HPI Data for house prices; and the Current Population Survey for the labor market indicators used throughout the paper.

Also, we show throughout the paper that the labor market estimates are similar in different specifications, including unweighted regressions and sample restrictions. Furthermore, as noted above, manufacturing employment shares and oxycodone rates pre-2010 are essentially uncorrelated, hence our labor market estimates are unlikely to reflect manufacturing exposure.

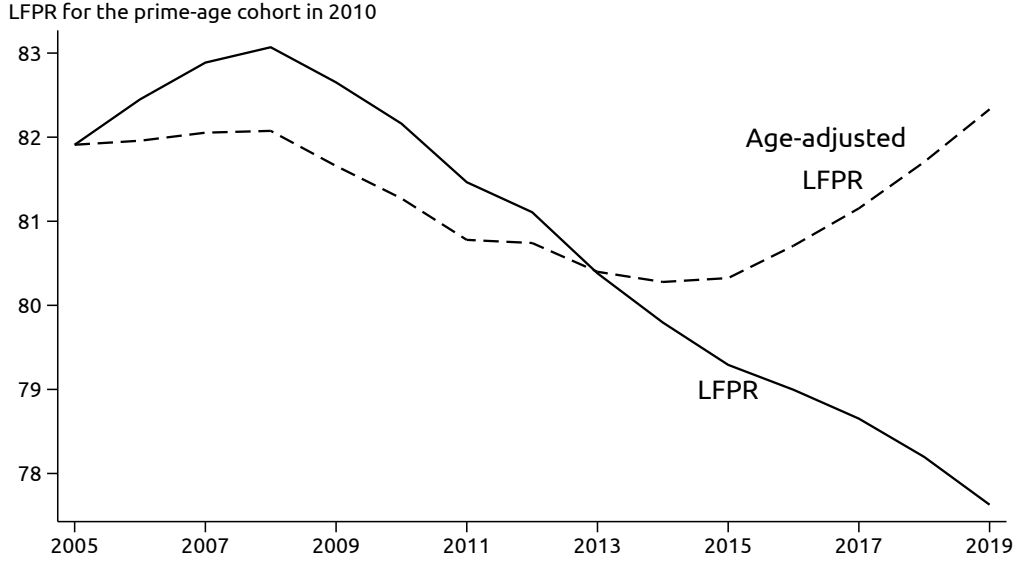
We also estimate the following related specification, where β estimates the conditional difference in the level of the outcome variable by oxycodone exposure between the pre and post-periods:

$$Y_{st} = \alpha_t + \gamma_s + \beta[Post_t \times OxyRate_s^{pre}] + \omega X_{st} + \epsilon_{st} \quad (3)$$

$Post_t$ is an indicator that is equal to 1 for years 2011 to 2019 and is zero for years 2005-2010. Similarly to [Alpert et al. \(2018\)](#) and [Park and Powell \(2021\)](#), we conservatively treat 2010 as part of the pre-period. We rely on both specifications, as the model in equation 2 is more flexible, but the model in equation 3 allows for more concise reporting.

For our labor market dependent variables, we construct local area aggregates from the CPS. For the baseline specifications, we focus on people who were of prime-age (25-54 year olds) in 2010, the year that the supply of oxycodone was tightened significantly and the shift to heroin began. We choose to track this specific prime-age cohort, as this group faced a plausibly exogenous and unexpected shock to oxycodone supply. The cohort approach is a tractable way of omitting prime-age workers who were teenagers in 2010 and thus less attached to the labor force at that time. However, in the robustness section we also include estimates for persons who were 25-54 years old each year and find the results are similar.

Figure 6: LFPR for the prime-age cohort in 2010



Source: CPS and authors' calculations

Figure 6 plots the aggregate LFPR for this cohort of individuals who were 25 to 54 years old in 2010. As the cohort has aged, participation has trended down, since this cohort started to age into its retirement ages after 2010, and those ages are associated with significantly lower LFPRs compared to the prime-age. Of course, we want to avoid picking up the effects of aging for this cohort in our estimates of heroin use on labor market outcomes. Thus, in all of our baseline estimates, we age-adjust each of our labor market outcome variables. Specifically, we use the CPS sample from 1995 on to estimate equation 4 at the individual i level, and aggregate the residuals:

$$Y_{ist} = \theta_{age(i)} + v_{ist} \quad (4)$$

where $\theta_{age(i)}$ are a full set of age fixed effects. Figure 6 plots the aggregate age-adjusted LFPR, normalized to start in 2005 at the same level as the unadjusted series. The age-adjustment leads to an appreciable difference in the aggregate LFPR time series. After declining after the Great Recession, the age-adjusted LFPR has recovered gradually as the labor market has strengthened. In our baseline estimates, we use the age-adjusted measures. That said, we show the age-adjustment has little effect on our labor market estimates, which are cross-sectional. More broadly, we find little evidence of a change post-2010 in the age and other demographic structure of states with greater oxycodone exposure.

Table 4: Effects on heroin and synthetic opioids deaths per 100,000

Dependent variables:								
	Heroin	Heroin	Heroin	Heroin	Synth.	Synth.	Synth.	Synth.
	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE
Oxy Rate pre X								
2011 – 2019	1.717** (0.81)	2.120*** (0.78)	0.771 (0.96)	2.312** (1.11)	4.978*** (1.31)	4.477*** (1.11)	3.138** (1.33)	5.274*** (1.65)
Other controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes
Weighted	No	No	Yes	Yes	No	No	Yes	Yes
FL included	Yes	Yes	Yes	No	Yes	Yes	Yes	No
R-squared	0.36	0.70	0.78	0.80	0.18	0.79	0.84	0.84
Observations	765	765	765	750	765	765	765	750

The table shows estimates of β from equation 3. The models in columns 1 and 5 include only the levels and interaction of $Post_t$ and $OxyRate_s^{pre}$. The remaining columns include all baseline controls. Standard errors clustered by state. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

3 Results

First, we briefly review and update the evidence that states with greater exposure to oxycodone prior to 2010 experienced larger increases in death rates from heroin and other illicit opioids after 2010. Second, we show that the oxycodone-heroin shock led to persistently lower EPOP and LFPR since 2010 in more affected states, particularly for non-Hispanic whites, both male and female, younger cohorts, and persons without a college degree. These effects are likely driven by behavioral changes rather than demographic changes in composition caused by the increase in heroin demand and mortality. Next, we discuss the aggregate implications of the baseline findings, before concluding with a discussion about the sensitivity of these estimates to alternative specifications.

3.1 Effects on heroin and synthetic opioid death rates

First, we update and review the evidence presented in [Alpert et al. \(2018\)](#), [Evans et al. \(2019\)](#), [Park and Powell \(2021\)](#), [Powell et al. \(2019\)](#), [Powell and Pacula \(2021\)](#), and others, that areas with greater exposure to oxycodone pre-2010 experienced subsequent relative increases in heroin and synthetic opioid (mainly fentanyl) mortality. Relative to the cited papers, the data we use (through 2019) is more recent.¹⁸

Table 4 reports estimates from equation 3 for the heroin and synthetic opioid death rate models with

¹⁸[Alpert et al. \(2018\)](#) report data through 2013; [Evans et al. \(2019\)](#) through 2014; [Park and Powell \(2021\)](#) and [Powell et al. \(2019\)](#) through 2015; and [Powell and Pacula \(2021\)](#) through 2017.

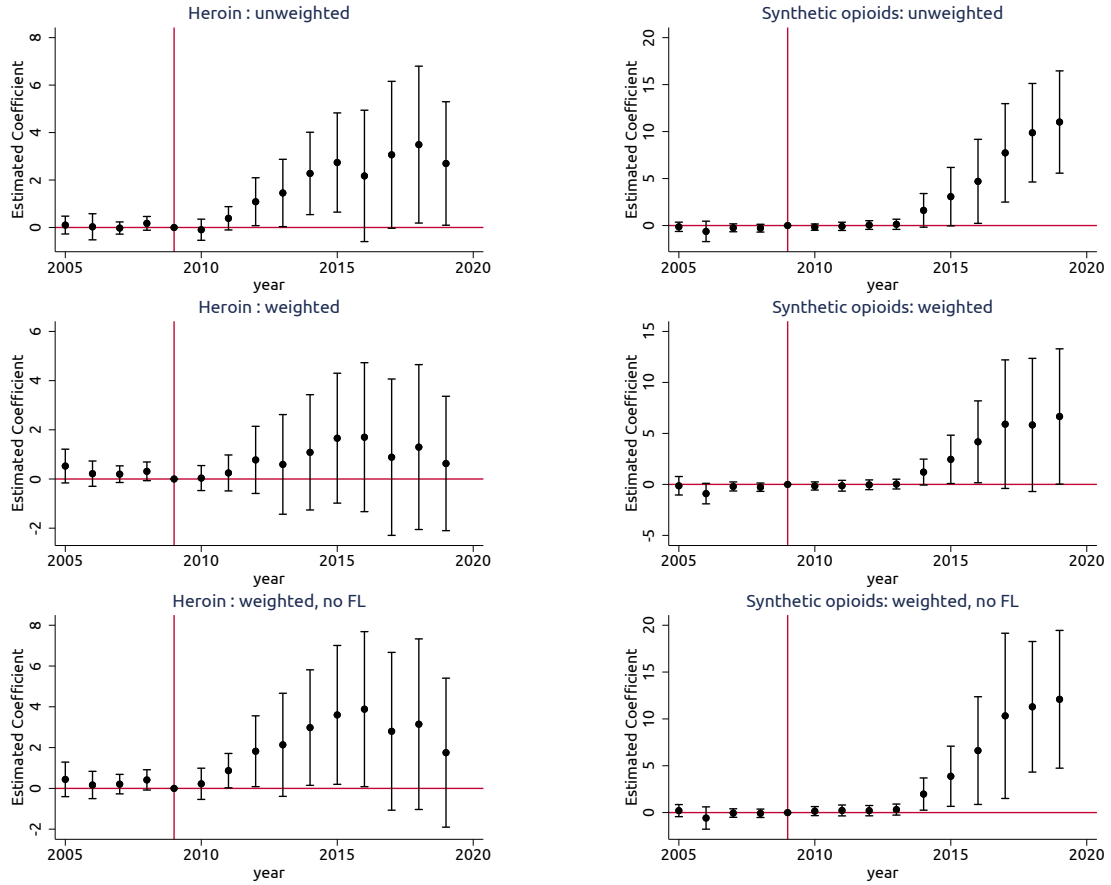
and without all the baseline controls, population weights, and the inclusion of Florida. Generally, across the models, states with one standard deviation higher oxycodone rates pre-2010 experienced on average a relative increase of 2 more deaths from heroin overdoses and 5 more deaths from synthetic opioids per 100 thousand of the prime-age population in the 2011-2019 period relative to the 2005-2010 pre-period. Though we find the results are typically robust, we note the estimates are smaller in the heroin model when including Florida and weighting by population. In our sample, Florida has a high oxycodone rate pre-2010, but some oxycodone prescriptions there were likely diverted to other states as the ‘pill mills’ boomed. In line with the cited literature, we keep Florida and weight by population in our baseline specifications. However, we include results without Florida and without population weights throughout the paper, and find the labor market effects are quite similar.

Figure 7 plots point estimates and 95 percent confidence intervals from equation 2 for heroin and synthetic opioid death rates for regressions without population weights (top row), with population weights (middle row), and with population weights but without Florida (bottom row). In line with the findings in the literature, the pre-trends are flat and heroin deaths rates appreciably step up shortly after 2010. Fentanyl death rates begin rising a few years later, as it took some time for suppliers to introduce fentanyl (Powell and Pacula 2021). In recent years, the coefficient estimates in the heroin mortality models have plateaued or declined. Recently, heroin death have declined somewhat, particularly in states with higher oxycodone exposure pre-2010 (see also figure 3). Thus, we might expect the labor market effects to also plateau or decline in recent years. That said, heroin mortality rates are a noisy proxy for heroin use, as mortality rates reflect risky consumption practices.

3.2 Effects on the labor market

Table 5 presents estimates from equation 3, which quantify the effect of the oxycodone-heroin shift over the 2011-2019 period on EPOP and LFPR for the baseline cohort. Columns 1 and 5 show estimates for the EPOP and LFPR models without other controls, while the remaining columns report estimates including all the baseline controls. With or without controls, the coefficient estimates are quite similar, even as the R-squared is much higher when including controls. The table also reports estimates with and without population weights and with and without Florida. Overall, the coefficient estimates are quite similar in the different specifications. In the model with population weights and Florida, the coefficient estimate on EPOP and LFPR is around -0.30, indicating states with one standard deviation higher oxycodone exposure before 2010 had a 30-basis point relative decline in LFPR and EPOP in the 2011-2019 period relative to the 2005-2010 pre-period. As the estimates on EPOP and LFPR are very close, the effects on the unemployment rate are very small

Figure 7: Effects on heroin and synthetic opioid death rates



The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for overdose deaths per hundred thousand of the prime-age population from heroin and synthetic opioids overdoses. Regressions are unweighted (top row), weighted (middle row), and weighted without Florida (bottom). Baseline controls included. Standard errors clustered by state.

Table 5: Effects on labor market indicators

	Dependent variables:									
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	EPOP	EPOP	EPOP	EPOP	LFPR	LFPR	LFPR	LFPR	SSDI	PT
	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE	Coef/SE
Oxy Rate pre X										
2011 – 2019	-0.274**	-0.241**	-0.328***	-0.369***	-0.226*	-0.199	-0.287**	-0.346*	0.056	0.203
	(0.12)	(0.11)	(0.11)	(0.14)	(0.13)	(0.13)	(0.13)	(0.18)	(0.05)	(0.13)
Other controls	No	Yes	Yes	Yes	No	Yes	Yes	Yes	Yes	Yes
Weighted	No	No	Yes	Yes	No	No	Yes	Yes	Yes	Yes
FL included	Yes	Yes	Yes	No	Yes	Yes	Yes	No	Yes	Yes
R-squared	0.02	0.86	0.90	0.90	0.15	0.62	0.67	0.65	0.95	0.85
Observations	765	765	765	750	765	765	765	750	707	765

The table shows estimates of β from equation 3. The models in columns 1 and 5 include only the levels and interaction of $Post_t$ and $OxyRate_s^{pre}$. The remaining columns include all baseline controls. Standard errors clustered by state. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

(not shown). Hence, the results suggest the effects on EPOP are entirely from the participation margin.

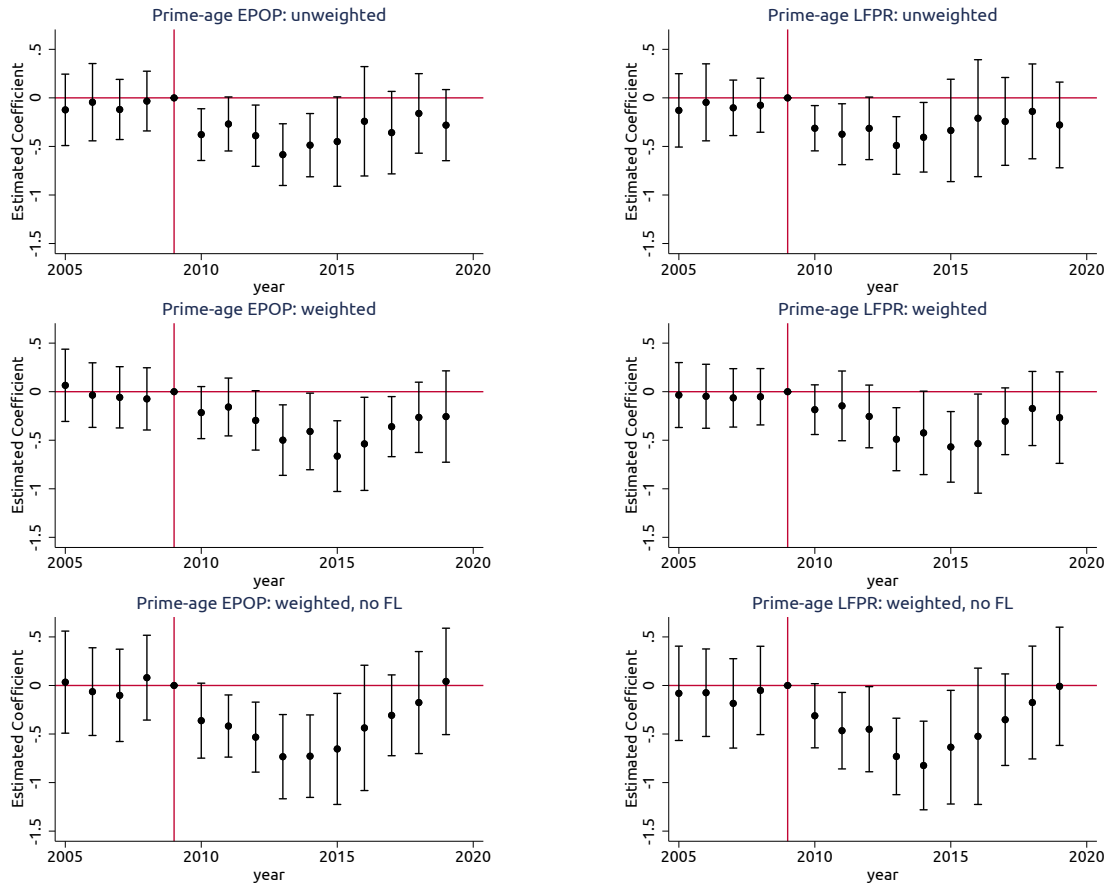
Figure 8 plots point estimates and 95 percent confidence intervals from equation 2 for the baseline EPOP and LFPR models for regressions without population weights (top row), with population weights (middle row), and with population weights but without Florida (bottom row). Prior to 2010, the coefficients are close to zero and not statistically significant. After 2010, however, states with previously larger oxycodone exposure experienced relative declines in EPOP and LFPR. Though the negative effects have persisted through the 2010s, they have grown smaller in recent years, perhaps as differences in heroin use across states have grown smaller. Indeed, the gap in heroin death rates explained by oxycodone exposure has fallen recently (see figure 7).

Overall, the patterns and magnitudes of the coefficient estimates across specifications are quite similar. Hence, from now on we use specifications with population weights and Florida as the baseline. That said, we include additional results in the robustness section for the alternative specifications.

Figure 9 plots point estimates and 95 percent confidence intervals for models of the rate of social security disability insurance (SSDI) beneficiaries and the prime-age part-time employment rate.¹⁹ Consistent with Park and Powell (2021), we find substitution towards heroin has caused some increase in the rate of disability beneficiaries. Within employment, we also find some evidence of an increase in the share of part-time workers. Columns 9 and 10 of Table 5 show a relative increase

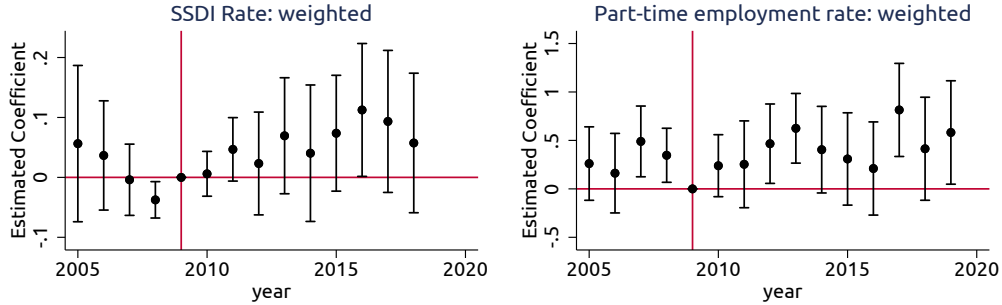
¹⁹Total SSDI beneficiaries data come from the Social Security Administration. The microdata is not available so we could not construct measures for the prime-age cohort in 2010 as we did for CPS variables.

Figure 8: EPOP and LFPR effects



The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for the rates of employment to population (left) and labor force participation (right). Regressions are unweighted (top row), weighted (middle row), and weighted without Florida (bottom). Baseline controls included. Standard errors clustered by state.

Figure 9: SSDI and part-time employment rate effects



The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for the rates of social security disability insurance beneficiaries (left) and part-time employment (right). Baseline controls included. Standard errors clustered by state. Observations weighted by population.

in SSDI and the part-time employment rate in states with greater oxycodone exposure though the increases are modest and not statistically significant.

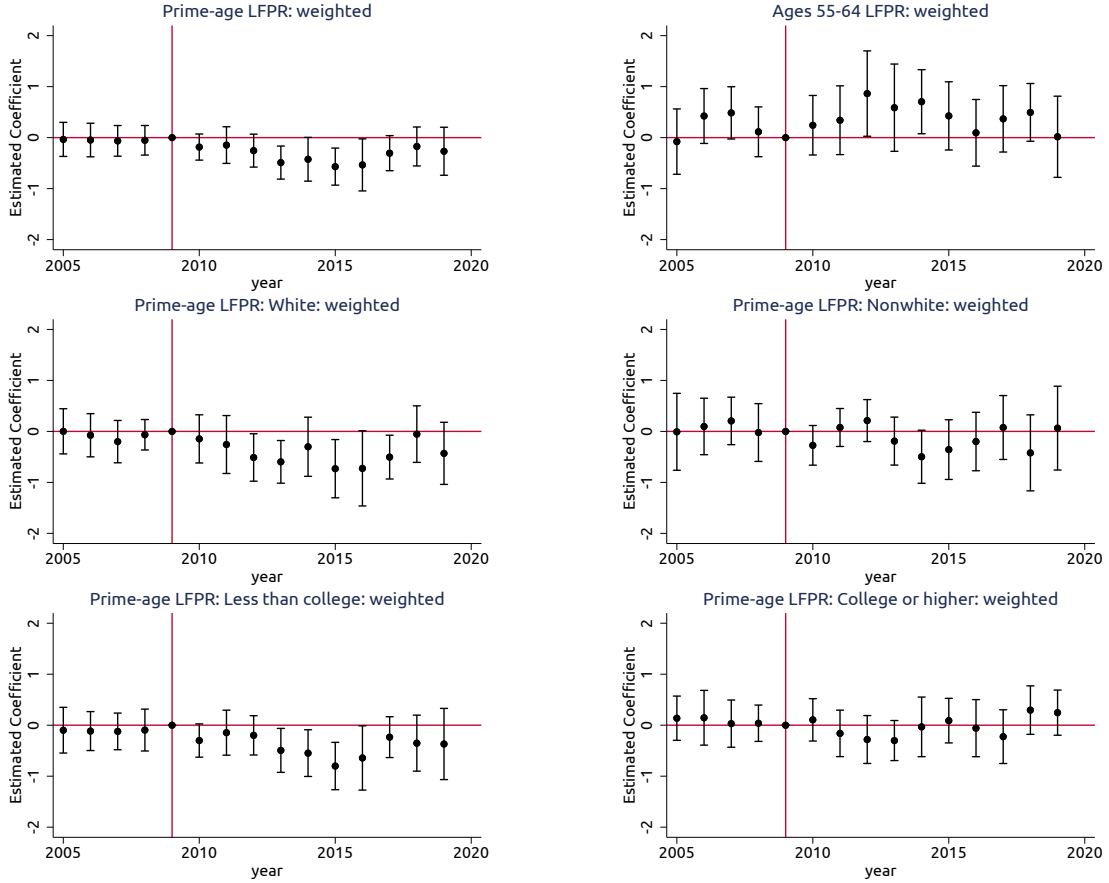
Next, we explore the heterogeneous effects of the oxycodone-heroin epidemic. We estimate separate models of equation 2 for local area aggregates by demographic groups. Figure 10 plots point estimates and 95 percent confidence intervals for LFPR models by age, race and ethnicity, and education. Starting with the age results in the top row, the left panel shows estimates for the baseline prime-age cohort, and the right panel shows the estimates for the cohort aged 55-64 in 2010. For the older cohort, the point estimates are generally not significant and tend to be positive, in contrast with the negative and significant estimates for the younger cohort.

The middle row shows estimates for non-Hispanic whites (left) and nonwhites (right), while the bottom row shows estimates for those without a college degree (left) and those with a college degree or higher (right). For non-Hispanic whites and those without a college degree, the estimates show a negative and significant effect of the increase in heroin use. For nonwhites and college graduates, the estimates are not generally significant and are sometimes positive (for college graduates). Tables 6 and 7 summarize the heterogeneous results for both LFPR and EPOP, respectively, based on estimates from equation 3, showing analysis consistent with figure 10.

Figure 11 plots estimates for LFPR by sex and ethnicity. For white males and females (top row), the estimates are generally negative and significant. In contrast, for nonwhites (bottom row) the effects seem largely flat though they are noisier particularly for nonwhite females.

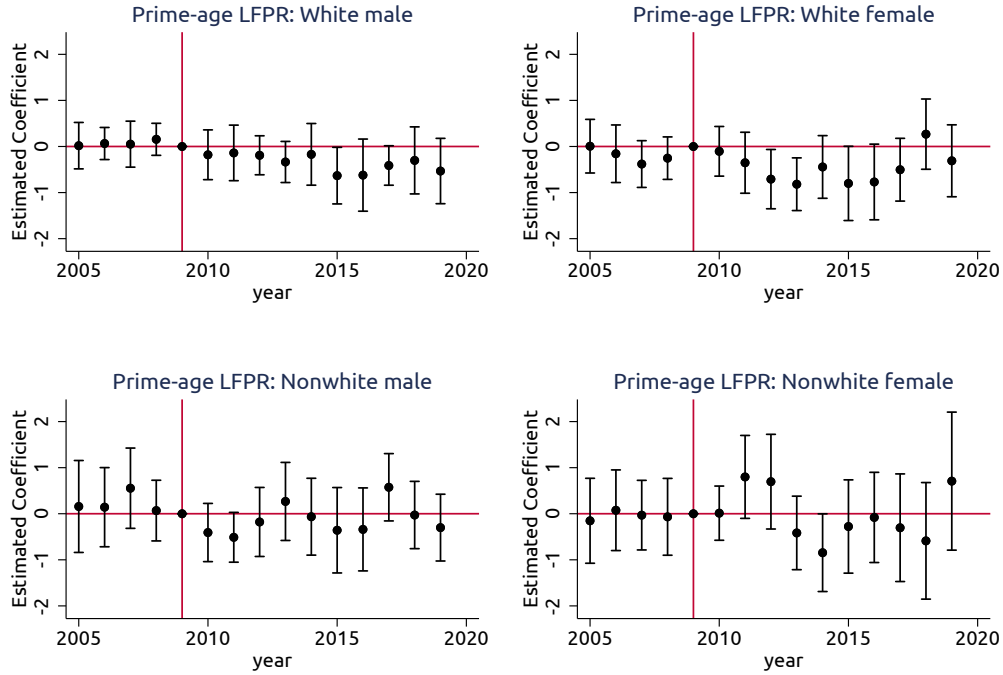
Though the negative effects on the white, younger, and less educated cohorts are stark, we stress we do not interpret these results as showing the oxycodone-heroin epidemic as having a negligible

Figure 10: LFPR effects by age, race and ethnicity, and education



The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for: the prime-age cohort in 2010 (top left); the cohort aged 55-64 in 2010 (top right); prime-age non-Hispanic whites (middle left); nonwhites (middle right); persons without a college degree (bottom left); persons with a college degree or higher (bottom right). Baseline controls included. Standard errors clustered by state. Observations weighted by population.

Figure 11: LFPR effects by sex and race and ethnicity



The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for prime-age in 2010 non-Hispanic white males (top left) and females (top right); and nonwhite males (bottom left) and females (bottom right). Baseline controls included. Standard errors clustered by state. Observations weighted by population.

effect on other groups. The results in table show that for nonwhites and college graduates the estimates are negative, and the standard errors are large enough that we can neither reject the hypothesis that they are zero nor that they are large and negative. Moreover, since 2010, heroin and fentanyl mortality have risen across demographic groups (Drake et al. 2020).

Next, we examine whether these effects could be explained by composition rather than behavioral changes associated with the oxycodone-heroin shock. For example, changes in death rates or net migration across states due to the shift to heroin use would cause changes in composition that could affect the LFPR and EPOP. To explore this possibility, we decompose EPOP and LFPR into age, education, and demographic components. Specifically, we estimate the following equation akin to equation 4 and aggregate the predicted values to the state and year levels.²⁰

$$Y_{ist} = \theta_{age(i)} + \theta_{edu(i)} + \theta_{sex(i)} + \theta_{race(i)} + v_{ist} \quad (5)$$

²⁰For education, the categories used are: some high school or less; high school; some college; college degree; and more than college. For race and ethnicity, the categories used are: non-Hispanic white; Black; Hispanic; and other. The fitted values are aggregated using the person weights provided in the CPS.

Table 6: Effects on LFPR by demographic groups

LFPR:								
	White Coef/SE	WM Coef/SE	WF Coef/SE	No Col Coef/SE	Age 25 – 34 Coef/SE	Nonwhite Coef/SE	Col Coef/SE	Age 55 – 64 Coef/SE
Oxy Rate pre X 2011 – 2019	-0.375** (0.16)	-0.375** (0.17)	-0.358 (0.23)	-0.297* (0.16)	-0.305** (0.15)	-0.133 (0.18)	-0.125 (0.12)	0.237 (0.19)
Other controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.59	0.56	0.48	0.75	0.57	0.50	0.53	0.60
Observations	765	765	765	765	765	765	765	765

The table shows estimates of β from equation 3. Estimates are for the prime-age cohort in 2010 unless otherwise stated. Results shown for: non-Hispanic whites (column 1), white males (column 2), and white females (column 3); persons without a college degree (column 4); the cohort aged 20-32 in 2010 (column 5); nonwhites (column 6); persons with a college degree or higher (column 7); and the cohort aged 55-64 in 2010 (column 8). All baseline controls included. Standard errors clustered by state. Observations weighted by population. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

Table 7: Effects on EPOP by demographic groups

EPOP:								
	White Coef/SE	WM Coef/SE	WF Coef/SE	No Col Coef/SE	Age 25 – 34 Coef/SE	Nonwhite Coef/SE	Col Coef/SE	Age 55 – 64 Coef/SE
Oxy Rate pre X 2011 – 2019	-0.409*** (0.15)	-0.398** (0.17)	-0.404* (0.20)	-0.311** (0.13)	-0.266 (0.16)	-0.221 (0.15)	-0.157 (0.13)	0.201 (0.19)
Other controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.82	0.81	0.68	0.89	0.84	0.84	0.68	0.65
Observations	765	765	765	765	765	765	765	765

The table shows estimates of β from equation 3. Estimates are for the prime-age cohort in 2010 unless otherwise stated. Results shown for: non-Hispanic whites (column 1), white males (column 2), and white females (column 3); persons without a college degree (column 4); the cohort aged 20-32 in 2010 (column 5); nonwhites (column 6); persons with a college degree or higher (column 7); and the cohort aged 55-64 in 2010 (column 8). All baseline controls included. Standard errors clustered by state. Observations weighted by population. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

Table 8: Effects on composition

	EPOP predicted by:			LFPR predicted by:		
	Age Coef/SE	Educ. Coef/SE	Demographics Coef/SE	Age Coef/SE	Educ. Coef/SE	Demographics Coef/SE
Oxy Rate pre X 2011 – 2019	-0.038 (0.03)	0.017 (0.06)	-0.047 (0.05)	-0.031 (0.02)	0.018 (0.05)	-0.034 (0.04)
Other controls	Yes	Yes	Yes	Yes	Yes	Yes
Observations	765	765	765	765	765	765

The table shows estimates of β from equation 3 for EPOP and LFPR predictions based on age, education, and broader set of demographics (see equation 5). All baseline controls included. Standard errors clustered by state. Observations weighted by population. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

We include the predicted values for EPOP and LFPR as dependent variables, for decompositions from age effects only, education effects only, and broad demographic effects including age, education, sex, and race and ethnicity. Table 8 reports the baseline estimates from equation 3. Across the models, the coefficient estimate on oxycodone exposure is close to zero and not significant, indicating states with greater oxycodone exposure did not experience a relative change in their demographics structure after 2010 in a way that majorly impacted EPOP or LFPR. Thus, we interpret the main findings as likely reflecting behavioral changes from greater heroin use rather than compositional effects.

3.3 Aggregate Implications

To gain a sense of the aggregate implications on LFPR, we combine the baseline estimates with a counterfactual assumption in which all states have low oxycodone rates and are unaffected by the oxycodone-heroin shock. As is generally the case, this counterfactual is not observed. We begin by assuming states in the bottom 5 percent of the oxycodone rate distribution were unaffected, and reduce oxycodone rates for the remaining states to the 5th percentile.²¹ Next, we calculate the counterfactual LFPR by year using the baseline estimates from equation 2. This main exercise suggests the oxycodone-heroin shock contributed to an average 40 basis point decline in LFPR from 2010 to 2019.

In an alternative counterfactual, we assume the lowest quartile of the distribution by oxycodone rates was unaffected, and reduce oxycodone rates for the remaining states to the 25th percentile. In this exercise, the oxycodone-heroin shock explains an average 25 basis point decline in LFPR from 2010 to 2019.

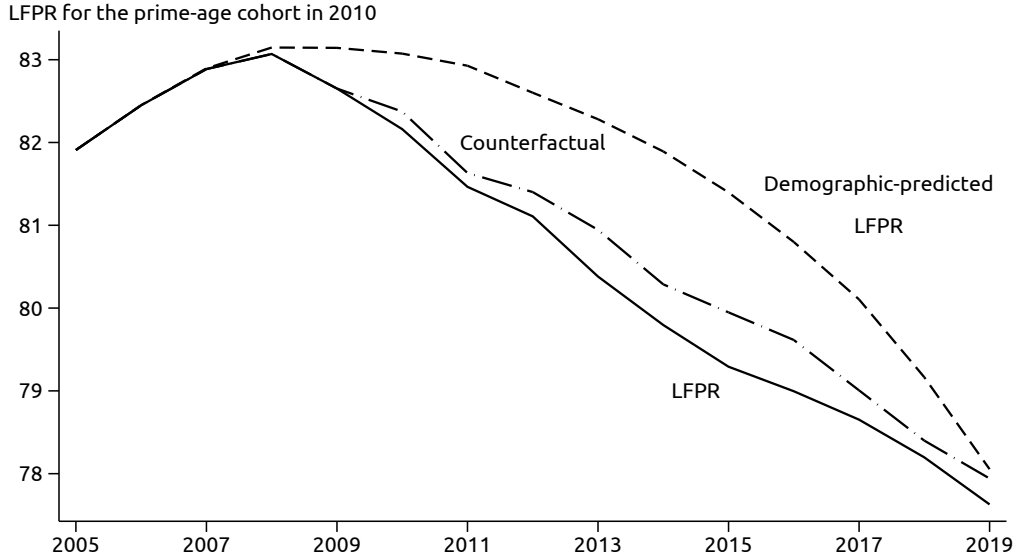
We relate these magnitudes to the gap between the unadjusted LFPR and the LFPR predicted by demographics (age, educational attainment, sex, and race and ethnicity) from equation 5. Figure 12 plots the LFPR, the demographics-predicted LFPR, and the counterfactual LFPR under the main aggregation exercise, with the latter two normalized to start at the same level as the unadjusted series in 2005. On average, LFPR was about 1.5 percentage points below the demographics-predicted LFPR from 2010 to 2019, likely mainly reflecting cyclical effects from the Great Recession (Cajner et al. (2020)). The main counterfactual explains about one-quarter of the gap during this period, whereas the alternative counterfactual explains about one-sixth of the gap.

The accuracy of these aggregation exercises depend on other factors in addition to the counterfactual assumptions. This approach treats states as independent units, but trade and other equilibrium effects could be important.²² Also, there is noise in the baseline estimates. When repeating the aggregation exercise using the 90 percent confidence intervals from equation 2, the oxycodone-heroin shock could explain between 3 and 76 basis point decline in LFPR for the period between 2010 and 2019. Given these caveats, we interpret these exercises as a useful robustness check. That said, there is much uncertainty around the aggregate effect.

²¹Alternatively, we could have assumed that all states had zero oxycodone rates prior to 2010. However, this would require extrapolating out of the sample as all states had positive oxycodone rates. See Alpert et al. (2018) for a related approach and discussion.

²²See Chodorow-Reich (2020) for a discussion on translating regional estimates to aggregate implications.

Figure 12: Counterfactual



The figure plots the LFPR, the LFPR predicted from equation 5, and the LFPR from the main counterfactual where states had low enough oxycodone rates prior to 2010 they were unaffected by the decline in oxycodone supply. The latter two are normalized to equal the level of LFPR in 2005. See the text for details.

3.4 Robustness

In this section we examine the sensitivity of the baseline EPOP and LFPR estimates to alternative specifications. First, we report additional results for specifications without Florida and without population weights. Next, we report estimates for the prime-age cohort each year rather than for the prime-age cohort in 2010. Also, we address the extent to which the findings could be explained by: the cyclicity of drug use; unobserved differences between states in cyclical or demographic characteristics; industry composition; the age-adjustment to LFPR and EPOP; and using metropolitan statistical area (MSA)-level rather than state-level data.

In the main text, we provided results when dropping Florida or estimating regressions without population weights for the main LFPR and EPOP specifications. Here, we provide additional results for different demographic groups. The additional estimates from equation 3 are reported in tables 9 and 10 for EPOP and LFPR, respectively. Also, figures 13 and 14 plot estimates from equation 2 for LFPR for the different demographic groups. Overall, the results without Florida and without population weights are quite similar to the baseline.

In our baseline specification, we track the labor market outcomes for the prime-age cohort in 2010, as this cohort faced a plausibly exogenous and unexpected shock to the supply of oxycodone.

Table 9: Effects on EPOP without Florida or population weights

	No Florida				Unweighted			
	All Coef/SE	White Coef/SE	No Col Coef/SE	Age 55 – 64 Coef/SE	All Coef/SE	White Coef/SE	No Col Coef/SE	Age 55 – 64 Coef/SE
Oxy Rate pre X 2011 – 2019	-0.369*** (0.14)	-0.457** (0.18)	-0.336* (0.18)	0.309 (0.26)	-0.241** (0.11)	-0.310* (0.15)	-0.233 (0.16)	0.171 (0.33)
Other controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.90	0.83	0.89	0.65	0.86	0.78	0.85	0.50
Observations	750	750	750	750	765	765	765	765

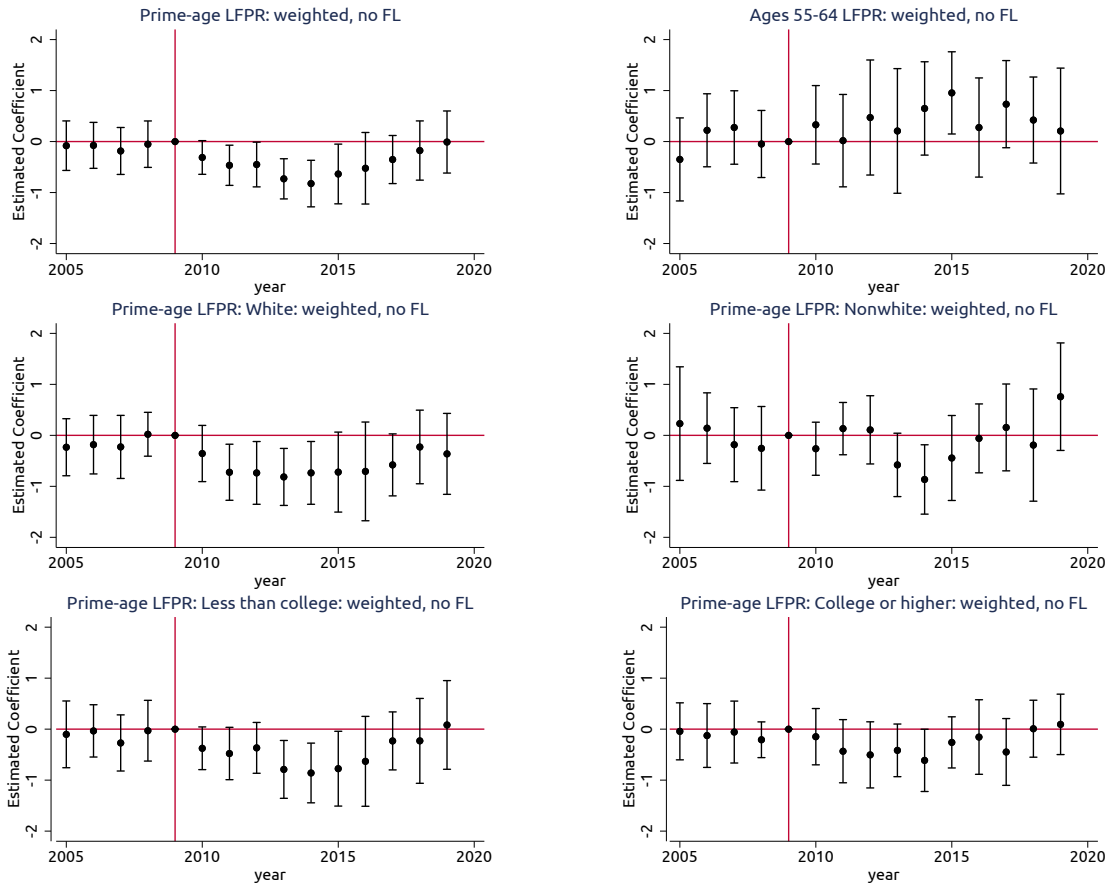
The table shows estimates of β from equation 3 for the baseline prime-age cohort in 2010, non-Hispanic whites, persons without a college degree, and the cohort that was 55-64 in 2010. In columns 1-4, weighted estimates are shown without Florida. In columns 5-8, results are unweighted. Standard errors clustered by state. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

Table 10: Effects on LFPR without Florida or population weights

	No Florida				Unweighted			
	All Coef/SE	White Coef/SE	No Col Coef/SE	Age 55 – 64 Coef/SE	All Coef/SE	White Coef/SE	No Col Coef/SE	Age 55 – 64 Coef/SE
Oxy Rate pre X 2011 – 2019	-0.346* (0.18)	-0.452** (0.20)	-0.340 (0.24)	0.366 (0.27)	-0.199 (0.13)	-0.274* (0.16)	-0.209 (0.19)	0.201 (0.33)
Other controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.65	0.58	0.73	0.61	0.62	0.57	0.69	0.46
Observations	750	750	750	750	765	765	765	765

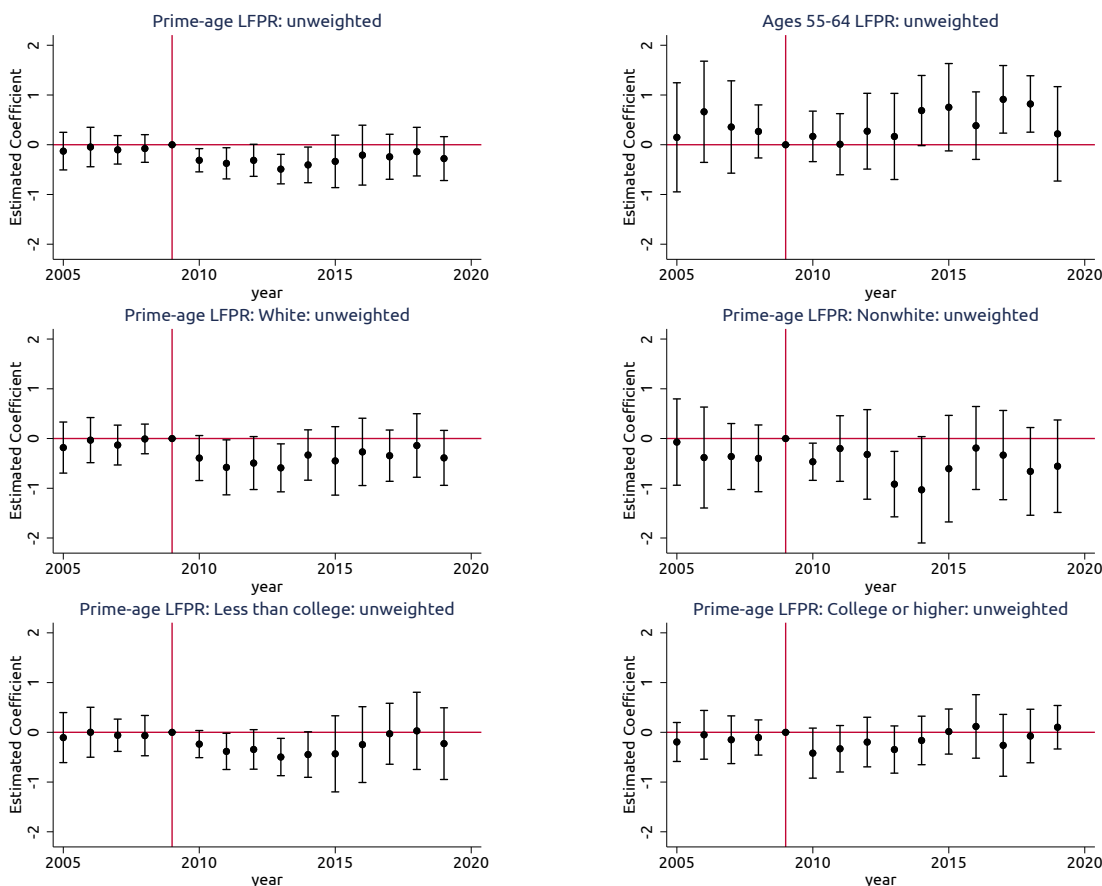
The table shows estimates of β from equation 3 for the baseline prime-age cohort in 2010, non-Hispanic whites, persons without a college degree, and the cohort that was 55-64 in 2010. In columns 1-4, weighted estimates are shown without Florida. In columns 5-8, results are unweighted. Standard errors clustered by state. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

Figure 13: LFPR effects by demographic groups, without FL



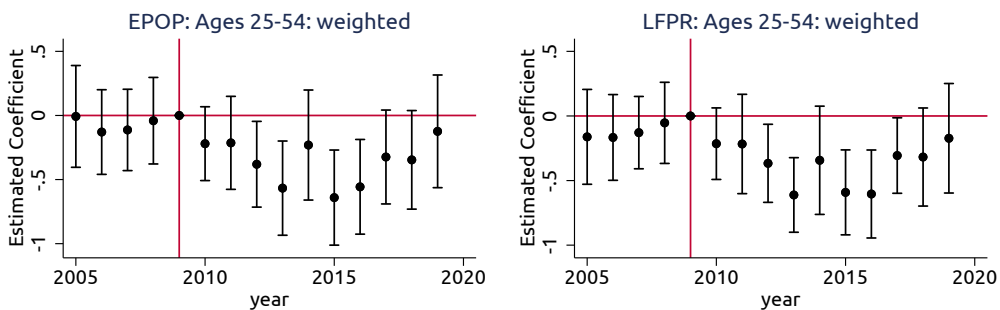
The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for non-Hispanic white males (top left) and females (top right); and nonwhite males (bottom left) and females (bottom right). Baseline controls included. Standard errors clustered by state. Observations weighted by population and Florida excluded.

Figure 14: LFPR effects by demographic groups, unweighted



The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for non-Hispanic white males (top left) and females (top right); and nonwhite males (bottom left) and females (bottom right). Baseline controls included. Standard errors clustered by state.

Figure 15: EPOP and LFPR effects: Ages 25-54



The figure shows point estimates and 95 percent confidence intervals of δ_t from equation 2 (with δ_{2009} normalized to zero) for EPOP and LFPR for persons aged 25-54 each year. Standard errors clustered by state. Observations weighted by population.

Table 11: Effects on EPOP and LFPR by age groups

	EPOP:				LFPR:			
	All Coef/SE	White Coef/SE	No Col Coef/SE	Age 55 – 64 Coef/SE	All Coef/SE	White Coef/SE	No Col Coef/SE	Age 55 – 64 Coef/SE
Oxy Rate pre X								
2011 – 2019	-0.291*** (0.08)	-0.401*** (0.11)	-0.206* (0.11)	0.206 (0.22)	-0.272*** (0.07)	-0.368*** (0.10)	-0.237** (0.10)	0.258 (0.22)
Other controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.91	0.84	0.90	0.46	0.70	0.61	0.76	0.33
Observations	765	765	765	765	765	765	765	765

The table shows estimates of β from equation 3 for the group each year that was prime-age, white, without a college degree, and aged 55-64. Standard errors clustered by state. Observations weighted by population. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

Instead of tracking this cohort, we now report estimates for the prime-age group each year. Figure 15 shows estimates from equation 2 and table 11 shows estimates from equation 3. The results are similar to the baseline.

As drug use could vary with economic conditions (Hollingsworth et al. 2017), it is plausible that geographic variation in oxycodone rates measured over 2006 to 2009 could have been partly caused by differences across states in the severity of the Great Recession. We address this possibility in two ways – by controlling for exposure to other prescription drugs, and by measuring oxycodone exposure in 2000 rather than the 2006-2009 period – and do not find this channel could have an important effect on the estimates. First, we include as additional controls hydrocodone rates measured over 2006 to 2009 with a full set of year effects; these estimates are reported in columns 1-2 of table 12. Hydrocodone is the second largest of the prescription opioids and state-level hydrocodone rates are correlated with the manufacturing share of employment, as shown in figure 5. Second, we repeat the baseline estimates, measuring average oxycodone exposure from 2000 to 2001 instead of from 2006 to 2009. The correlation coefficient between these two measures is 0.85, consistent with Alpert et al. (2019) who find that early differences in oxycodone supply have had persistent implications. Columns 3-4 of table 12 show these estimates. In both cases, the coefficients of interest are very similar to the baseline estimates.

Another possibility is that omitted variable bias may influence the results if there are unobserved factors correlated with oxycodone use and the performance of labor markets. The baseline estimates already flexibly control for differences in the severity of the Great Recession, demographics, and industry composition, as discussed in section 2. Indeed, the LFPR and EPOP pre-trends are flat as shown in figure 8. Also, employment for nonwhites tends to covary more with the cycle than for whites (Cajner et al. (2017)), but our findings are stronger for the latter. Nonetheless, we estimate

Table 12: Robustness checks

	Hydrocodone		Oxy 2000		UR and HPI		Manuf shares	
	EPOP Coef/SE	LFPR Coef/SE	EPOP Coef/SE	LFPR Coef/SE	EPOP Coef/SE	LFPR Coef/SE	EPOP Coef/SE	LFPR Coef/SE
Oxy Rate pre X 2011 – 2019	-0.341*** (0.11)	-0.309** (0.12)			-0.400*** (0.07)	-0.372*** (0.08)	-0.304** (0.12)	-0.301** (0.14)
Oxy Rate 2000 X 2011 – 2019			-0.227* (0.12)	-0.248* (0.14)				
Other controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	
R-squared	0.91	0.69	0.90	0.66	0.88	0.62	0.90	0.67
Observations	765	765	765	765	765	765	765	765

The table shows estimates of β from equation 3 for alternative models. In columns 1-2, hydrocodone rates from 2006 to 2009 are interacted with year effects. In columns 3-4, oxycodone rates are measured from 2000 to 2001. In columns 5-6, three lags of the unemployment rate and house price index are included. In columns 7-8, three lags of the services and manufacturing employment shares are included. Standard errors clustered by state. Observations weighted by population. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

alternative specifications. First, we include three lags of the unemployment rate and house price index as controls, instead of the approach in the baseline where we interact changes from 2003 to 2006 and 2006 to 2009 with year fixed effects. Columns 5-6 of table 12 show these estimates. Second, we include three lags of both the manufacturing share of employment and the service industry share of employment, instead of only controlling for one lag of the service share as in the baseline (columns 7-8). The estimates are close to the baseline results.

In addition, when constructing the EPOP and LFPR aggregates, we strip out a broad set of demographic effects beyond age (see equation 5) and repeat the analysis.²³ Columns 1-2 of table 13 show these estimates. We also report estimates for the unadjusted LFPR and EPOP in columns 3-4 of Table 13. In both cases, the results are similar to the baseline.

Finally, we estimate additional specifications. We report estimates when including division fixed effects interacted with year effects to account for common regional trends (columns 5-6).²⁴ Lastly, we repeat the analysis using MSA-level data rather than state-level data (columns 7-8). We prefer state-level specifications as estimates based on smaller geographies are likely noisier from both greater oxycodone diversion and smaller CPS samples. We are able to aggregate the LFPR and EPOP into about 200 distinct MSAs using the CPS microfiles. In these cases, the coefficient estimates are again similar to the baseline.

²³Specifically, we separately regress LFPR and EPOP on age, education, race and ethnicity, and sex fixed effects and aggregate the residuals to the state-year level.

²⁴The U.S. Census divides the country into 9 divisions. See [here](#) for more info.

Table 13: Additional robustness checks

	Demographics		Unadjusted		Division		MSA	
	EPOP Coef/SE	LFPR Coef/SE	EPOP Coef/SE	LFPR Coef/SE	EPOP Coef/SE	LFPR Coef/SE	EPOP Coef/SE	LFPR Coef/SE
Oxy Rate pre X 2011 – 2019	-0.319** (0.12)	-0.284** (0.14)	-0.366*** (0.11)	-0.318** (0.13)	-0.359** (0.14)	-0.334** (0.15)	-0.326** (0.15)	-0.292* (0.16)
Other controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.90	0.71	0.86	0.90	0.94	0.79	0.53	0.23
Observations	765	765	765	765	765	765	2910	2910

The table shows estimates of β from equation 3 for alternative models. In columns 1-2, EPOP and LFPR are adjusted for age, education, race, and sex (as per equation 5). In columns 3-4, Florida is dropped. In columns 5-6, controls include the interactions of year and U.S. division fixed effects. Columns 7-8 use MSA-level data with outliers dropped (1 percent of each tail by oxycodone rates). Standard errors clustered by state. Observations weighted by population. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.10$.

4 Conclusion

Previous work, including [Alpert et al. \(2018\)](#), [Evans et al. \(2019\)](#), and [Powell and Pacula \(2021\)](#), have shown areas with greater oxycodone exposure prior to 2010 experienced relative increases in heroin and fentanyl death rates after prescription supply tightened in 2010. We contribute to this literature by showing more exposed areas also experienced relative declines in EPOP and LFPR, particularly for white, less educated, and younger cohorts, consistent with the profile of oxycodone nonmedical users prior to 2010. These results indicate that, when accounting for the effects of the opioid crisis, extending beyond prescriptions is important. In particular, while tighter oxycodone supply may have deterred some new addictions, many with existing opioid use disorders turned to heroin and other drugs including fentanyl. In recent years, deaths from heroin and fentanyl overdoses have surpassed deaths from prescription opioid overdoses. We view research on the health and economic effects of medication-assisted and other treatments for opioid use disorder as an important area of further work.

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