Drug Potency Shocks and Overdose Mortality: Evidence from Geographically Segmented Heroin Markets*†

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Abstract

We provide the first causal evidence that geographically concentrated shocks to heroin potency drove recent surges in U.S. overdose mortality. Exploiting the fact that white powder heroin markets experienced greater purity variability and fentanyl adulteration beginning in 2012, while black tar markets did not, we compare subsequent mortality across commuting zones. Exposure to these shocks increased overdose death rates by 52% through 2019. These effects arose from heightened fatality risk among existing heroin users, highlighting the dangers of volatility in illicit drug supply and underscoring the central role of supply-side dynamics in shaping the recent trajectory of the overdose epidemic.

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Buyers in illicit drug markets only learn about drug purity and adulteration after consumption (Galenianos et al. 2012; Galenianos and Gavazza 2017), making variability in these product attributes a potential driver of overdose risk. This risk is especially acute for opioids, which have among the narrowest margins between intoxication and fatal overdose of any widely used substance (Gable 2004). Ethnographic research points to shifts in heroin purity and fentanyl adulteration as central to the surge in overdose mortality beginning in the early 2010s (e.g., Ciccarone et al. 2017), but their role in shaping the opioid epidemic has not yet been tested empirically. This paper provides the first causal evidence on this mechanism.

Using Drug Enforcement Administration (DEA) data on undercover heroin purchases, we first document that historical supply chains linked U.S. cities to distinct heroin formulations: in the early 2000s, eastern cities were supplied Colombian white powder heroin, while western cities were supplied with Mexican black tar. After 2011, Mexican drug trafficking organizations assumed control of white powder heroin production, and the resulting product became more variable in purity and increasingly adulterated with potent fentanyl analogues, while black tar heroin remained largely unchanged. Ethnographic studies confirm that fentanyl adulteration was confined to white powder, whose appearance and chemical properties made fentanyl especially difficult for users to detect (Carroll et al. 2017; Mars et al. 2018). We refer to increased variability of heroin purity and fentanyl adulteration collectively as "potency shocks," since both heighten the risk of unexpected exposure to high-potency opioids from the users' perspective.

We examine how exposure to potency shocks affected overdose mortality, considering both their immediate impact and how they shaped the longer-term trajectory of the epidemic. Using all-county death records from 2000 to 2019, we exploit the natural experiment created by these shocks affecting only white powder heroin markets. We compare changes in overdose mortality after 2012 for commuting zones historically supplied with white powder heroin to those supplied with black tar, which serve as an effective control group because their heroin supply chains were unchanged but they faced other widely cited drivers of increased overdoses over this period (e.g., OxyContin's reformulation). Consistent with the potency shocks being exogenous, we observe parallel trends in mortality between the two groups for more than a decade prior to 2012. After the shift in white powder heroin production to Mexico and the introduction of fentanyl, mortality in white powder commuting zones rose far more sharply, first through heroin and heroin–fentanyl combinations and later through fentanyl alone. This suggests that fentanyl's early role as an adulterant facilitated its transition into a standalone product in U.S. drug markets, as surviving heroin users developed tolerance to its far greater potency.

Overall, we estimate that exposure to potency shocks led to a 226% increase in heroin-related deaths and a 990% increase in fentanyl-related deaths between 2012 and 2019, translating into a 52% rise in total overdose mortality. These increases cannot be explained by other factors explored in the literature, such as tighter prescription opioid controls (Alpert et al. 2018; Evans et al. 2019; Kim 2021), greater exposure to imported fentanyl via legal imports (Moore et al. 2023), or changes in enforcement. Instead, evidence suggests the mortality surge was driven by an increase in the rate of death among existing heroin users, whose supply changed. Combining data from the National Survey on Drug Use and Health with vital records, we find that the probability of death among past-year heroin users more than doubled over this period.

We next shed light on two enduring puzzles that existing accounts of the opioid epidemic have struggled to explain. The first is the earlier and disproportionate impact of fentanyl in eastern states (Zoorob 2019; Abouk et al. 2021; Moore et al. 2023). Our findings show these geographic differences stemmed not from differences in OxyContin misuse, but from geographic segmentation in heroin supply chains. The second puzzle is the striking rise in opioid deaths among non-White populations (Ruhm 2022; Mulligan 2024; Powell et al. 2025). Although Blacks historically had lower exposure to prescription opioids, their heroin use rates were similar to Whites, leaving both groups equally vulnerable to potency shocks.

Our main contribution is providing causal evidence that heroin potency shocks were a major driver of the heroin and fentanyl waves of the U.S. opioid epidemic. Prior research emphasizes the unintended consequences of tighter prescription opioid controls—particularly OxyContin's abuse-deterrent reformulation and state-level Prescription Drug Monitoring Programs (PDMPs)—as the primary cause, arguing they induced user substitution to heroin (Alpert et al. 2018; Evans et al. 2019; Kim 2021; Powell and Pacula 2021). We revisit these interventions in light of our findings. When we account for regional exposure to potency shocks within existing empirical specifications, the estimated effects of the OxyContin reformulation and PDMPs shrink considerably, suggesting these policies played a more limited role in rising mortality than previously thought. Our decomposition attributes roughly half of the increase in illicit opioid deaths to potency shocks, about one-third to OxyContin's reformulation, and little to PDMPs.

More broadly, our findings highlight how recent shifts in the composition and structure of illicit drug supply chains represent an urgent national policy concern. We document substantial variation in heroin source countries, formulations, potency, and adulterants across cities and over time, and show that these differences have profound and lasting consequences for overdose mortality rates. Prior research emphasizes that drug quality in illicit markets is non-contractible and only verifiable after consumption, leaving buyers unable to assess product safety or quality ex ante (Galenianos et al. 2012; Galenianos and Gavazza 2017). This informational asymmetry creates moral hazard: sellers have a strong incentive to adulterate supply with more potent or cheaper—yet more dangerous—substances in order to increase addictiveness and profits. While such adverse selection might unravel most markets (Akerlof 1970), the inelastic demand for addictive substances (Becker and Murphy 1988) combined with the presence of thick market externalities (Cutler and Donahoe 2024) can weaken these corrective forces, allowing heightened supply-side risks to persist.

There is a potential role for government intervention to address these informational asymmetries, but programs that monitor illicit drug use and disseminate information about drug quality and attributes remain poorly funded in the United States. In fact, many such programs were de-funded as the opioid epidemic unfolded, such as the Arrestee Drug Abuse Monitoring Program (in 2014) and the Heroin Domestic Monitoring Program used in this study (in 2016). This leaves practitioners, policymakers, and the scientific community in the dark about shifts in illicit drug markets that could be used to inform more responsive public health interventions. Future research should consider whether interventions that address these informational asymmetries—such as provision of drug checking kits or regulated supply for high-risk drug users—can be effective at addressing the epidemic.

1 Data

We begin by describing our data, which informs our evidence on heroin market changes in the next section. Data on heroin markets comes from the DEA's Heroin Domestic Monitoring Program (HDMP), a federal surveillance system that operated until 2016, when it was discontinued under the first Trump administration. The HDMP was the only systematic effort to track heroin's composition and source at the retail level across the U.S. Unlike drug seizure data, which capture wholesale supply and reflect enforcement priorities, HDMP samples came from undercover DEA purchases at the retail level. This distinction is critical: heroin is routinely adulterated or diluted after entering retail markets (Arkes et al. 2008), and it is retail formulations—not wholesale shipments—that drive overdose risk. The HDMP thus provides unique evidence on the drugs that users actually consumed, making it well-suited to study how heroin potency shocks shaped mortality trends.

The program focused on large urban cities with DEA field offices and established heroin markets. We restrict our analysis to the 27 cities consistently sampled before 2011 (Appendix Table C.1). Roughly 700 heroin samples were collected annually across these cities, each tested at the DEA's Special Testing and Research Laboratory for purity, type (black tar, white powder, or brown powder), and common adulterants. A distinctive feature of the HDMP was its "chemical signature analysis," which identified the geographic origin of retail heroin by comparing each exhibit to authentic samples from major source areas: South America (primarily Colombia), Mexico, Southeast Asia, and Southwest Asia (DEA 2016a, p.4). As new sources and adulterants emerged, procedures were updated, typically with a lag. Authentic reference samples were required for comparison (DEA 2016a, p.3), and delays in obtaining these meant some samples were classified as unknown or suspected origin. Additional details about the HDMP are provided in Appendix C.

To measure overdose mortality, we use all-county vital records from the National Center for Health Statistics (NCHS 2021). These data provide information on all recorded deaths in the United States since 1999, including county and year of death, underlying cause of death, and all contributing causes listed on the death certificate. We focus on the pre-COVID pandemic period and follow CDC coding guidance (CDC 2021), using both the underlying and contributing cause-of-death fields (if applicable) to classify deaths.[1] We construct several non-mutually exclusive indicators to capture overdose deaths involving heroin and/or fentanyl. Specifically, we code whether a death involved any heroin, any fentanyl, only heroin (i.e., heroin without fentanyl), only fentanyl (i.e., fentanyl without heroin), both heroin and fentanyl, or either heroin or fentanyl. These and other data sources used in our analyses are described in Appendix C.

[1] Using these data, Appendix Figure A.1 highlights that there have been three waves of the overdose epidemic, each involving a different form of opioids: prescription opioids in the 1990s and early 2000s, heroin beginning in the early 2010s, and synthetic opioids, primarily illegally-produced fentanyl, ever since.

2 Background on U.S. Heroin Supply

2.1 Changes in U.S. Heroin Supply Chains

In the early 1970s, heroin was supplied to the largest and most lucrative heroin markets in the eastern U.S. as a white powder that was sourced from the Golden Triangle of Southeast Asia (Ciccarone 2009). In the 1990s, Colombian DTOs developed a "mimic" white powder heroin to compete for market share (DEA 1994). Owing in part to the infrastructure they developed to distribute cocaine in the 1980s, Colombian DTOs were able to sell their heroin at a lower cost than traffickers coming from Southeast Asia and took over as the dominant suppliers in the east (Rosenblum et al. 2014). Meanwhile, Mexican DTOs established dominance in the heroin markets of the west. There, they produced different types of heroin that resembled black tar and, to a lesser extent, brown powder (DEA 1994).

The result of this is that by the early 2000s, the U.S. heroin market was geographically segmented. Figure 1 highlights this phenomenon by showing that areas further east were supplied white powder heroin and those further west were supplied black tar heroin, where the dividing longitude is approximately -91 degrees; suppliers in the east were almost exclusively Colombian, while those in the west were Mexican. One crude marker cited in DEA reports for this division of supply is the Mississippi River, but there is no evidence this was an explicit boundary. These differences are believed to have endured over time, in part, because users developed sticky tastes for the types of heroin they were sold (Ciccarone 2009).

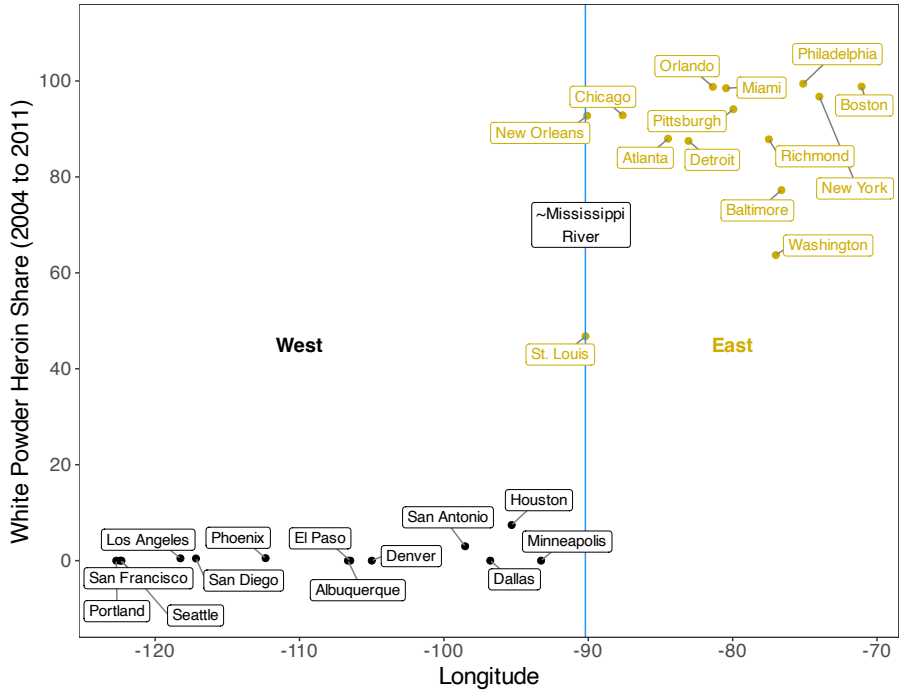


Figure 1: Geographic Segmentation of the U.S. Heroin Market

Notes: The data used to generate this figure comes from the Heroin Domestic Monitoring Program (HDMP) and each dot represents a city that is repeatedly sampled as part of this program. The share of white powder heroin in a given city is calculated by using the total number of exhibits collected by heroin type from 2004 to 2011, excluding exhibits with inconclusive testing (for which we can not confirm type) from the calculation. The centroid of the city is used to plot the longitude.

The means of trafficking heroin into the U.S. by Colombian and Mexican DTOs differed significantly. Colombian groups relied on couriers who smuggled heroin through east coast international airports, whereas Mexican groups trafficked heroin across the U.S.–Mexico border (DEA 2001). Two primary factors pushed Colombian DTOs to exit heroin trafficking. First, the 1997 reinstatement of the U.S.–Colombia extradition treaty led to the arrest, extradition, and imprisonment of major cartel leaders in the early 2000s. Second, heightened airport security following the September 11, 2001 terrorist attacks increased the risks of air smuggling and shifted trafficking routes toward the U.S.–Mexico border, where Mexican DTOs held a geographic comparative advantage (DEA 2003). Ultimately, Colombian DTOs scaled back, focusing on wholesale production and selling heroin to Mexican DTOs, who assumed responsibility for cross-border trafficking and U.S. retail distribution (DEA 2005, p. 68).

These events positioned Mexican DTOs to take over white powder heroin production from Colombian DTOs, a shift that by the mid-2010s would leave Mexican DTOs effectively dominating the entire U.S. heroin market. Estimates of Colombian heroin production declined steadily throughout the 2000s (Ospina et al. 2018). Mexican DTOs initially tried to penetrate eastern markets with black tar heroin, but these products failed to gain traction given user preferences for more potent white powder (DOJ 2007, p.14–15). They also experimented with fentanyl to mimic Colombian potency (DOJ 2007, p.16), fueling a 2005–2007 overdose spike (see Appendix Figure A.2) before the DEA dismantled what was believed to be the sole illicit fentanyl-producing lab at the time (in Toluca, Mexico) (CDC 2008).

DEA data indicate that this transition accelerated beginning 2012-2013. The timing of this shift is summarized in Panel A of Appendix Figure A.3. The 2011 HDMP report noted rising white powder heroin of unknown origin that could have been produced in Mexico, and by 2013, the DEA explicitly flagged the presence of "alleged Mexican white powder heroin" in traditional Colombian markets (DEA 2016b, p.1).[2] That year, 13% of eastern white powder exhibits were suspected to be Mexican; by 2014, the share increased to 74%. Once the DEA obtained an authentic sample of Mexican white powder heroin for comparison, half of samples were classified by the DEA as being Mexican and another 23% were classified as inconclusive but suspected to be Mexican. By 2016, 71% of eastern white powder heroin was Mexican in origin and less than 1% Colombian (down from 60% in 2011). Critically to the identification strategy that we present below, western markets (see Appendix Figure A.3) were unaffected by these shifts and remained dominated by Mexican black tar heroin throughout.

2.2 Drug Potency Changes in White Powder Heroin Markets

Appendix Figure A.3 Panel B compares the cross-city purity distribution of Colombian white powder heroin just before the takeover (2010–2011), suspected Mexican white powder (2013–2014), and confirmed Mexican white powder (2015–2016). Both the alleged and confirmed Mexican products were higher in average purity and exhibited a much heavier tail, meaning a greater chance of very pure heroin. This variability would be expected to heighten overdose risk by

[2]To the best of our knowledge, the 2012 HDMP report was never released. The 2013 report includes purity data for heroin samples classified as Colombian or Mexican in 2012, allowing us to quantify average purity that year, but it does not report the share of samples with unconfirmed origin necessary for inclusion in Appendix Figure A.3.

increasing the likelihood that users consumed more than their tolerance allowed (White and Irvine 1999). Appendix Figure A.4 shows that divergence in purity between white powder and black tar markets as a whole began in 2012.

A likely more consequential change was the adulteration of white powder heroin with fentanyl. Fentanyl is 20 to 50 times more potent than heroin, and a lethal dose is roughly 2 milligrams (DEA 2024). Variations of this amount are common in illicit markets (Larnder et al. 2022). Ethnographic evidence suggests the shift to fentanyl adulteration began in eastern markets around 2012–2013, i.e., in the same cities and at precisely the same time as Mexican-produced heroin displaced Colombian white powder heroin supply (Ciccarone et al. 2017; Mars et al. 2018, 2019). As one man who had been using heroin in Massachusetts for 18 years recalled (Ciccarone et al. 2017, p. 148):

> Q: So the heroin that is out there right now, how long has that been around? Have there been any changes?
>
> *A: It's been changing, like 3-1/2 years ago, maybe 4. 2012–2013, it's like that's when like the bullshit started coming in. Like mother-fucker's realized, if I got 3 grams of dope I can put 3 grams of bullshit on it.*
>
> Q: What do you mean by bullshit?
>
> *A: Like cut. Well, the fake cut or fentanyl or whatever [. . .]*

HDMP data soon validated these reports: the HDMP denoted 7 fentanyl-contaminated heroin samples in 2013 and 21 in 2014 (DEA 2015, p.23). All of them were from cities with white powder markets. In the last year of the HDMP in 2016, 158 heroin samples contained fentanyl (DEA 2016a, p.7). Roughly 99 percent of these were from white powder heroin cities.

Prior ethnographic research emphasizes asymmetric information as a key reason why fentanyl adulteration was confined to white powder heroin. Heroin and other illicit drugs are experience goods. Buyers are often not able to observe what they are adulterated with until they have consumed them, and there are no institutions to enforce quality in the illicit market. Therefore, sellers face strong incentives to adulterate their products (Galenianos et al. 2012; Galenianos and Gavazza 2017). At the import level, fentanyl is approximately 99% cheaper than heroin due to its compactness, lower trafficking costs, and synthetic production (Pardo and Reuter 2020). As a result, it is an attractive adulterant from the perspective of traffickers. However, early on, many heroin users preferred heroin that did not include it because of the increased risks (Ciccarone et al. 2017; Mars et al. 2018, 2019).

These dynamics were amplified for white powder heroin, with which fentanyl could be mixed invisibly. Black tar heroin, by contrast, is solid and harder to adulterate without detection (see Appendix Figure A.5). As one heroin user explained (Carroll et al. 2017, p.14):

> "Black tar heroin I feel safer doing, It's harder to cut. It's not powder. Powder you can add anything to . . . Black tar heroin, in order to cut it, you have to do all this stuff to it, cook it, and it's a lot harder to cut, so I feel safer doing that. But as far as fentanyl? There's no way to know until you [use] it. Like I said, I avoid the grey dope, but there's nothing you can really do [to avoid it]."

The precise mechanism behind the spread of fentanyl adulteration around 2012 remains uncertain. Mexican DTOs may have played a direct role, consistent with their early experimentation with fentanyl in 2006 and a federal indictment that alleges the Sinaloa cartel conspired to produce fentanyl and mix it with heroin by at least 2014.[3] Alternatively, others argue that Chinese chemical labs initially supplied fentanyl directly to U.S. retailers, with Mexican DTOs stepping in to sustain supply after Chinese exports declined (Dudley et al. 2019). Regardless of the mechanism, fentanyl adulteration first emerged in white powder heroin markets—primarily eastern cities—around 2012-2013. Black tar markets, concentrated in western cities, were largely unaffected. This divergence provides the basis for our empirical strategy in the next section.

3 The Effects of Potency Shocks on Overdose Mortality

3.1 Methodology

We estimate the causal impact of heroin potency shocks using the following event-study design that exploits variation across commuting zones in whether they were historically supplied with white powder or black tar heroin:

$$Y_{ct} = \sum_{\tau \neq 2011} \rho_\tau \times \left(\text{White_Powder_Market}_c \times 1\{t = \tau\}\right) + \lambda_c + \gamma_t + \nu_{ct}, \tag{1}$$

where Y_{ct} are overdose death rates per 100,000 in commuting zone c in year t. A commuting zone is defined as a white powder heroin market ($\text{White_Powder_Market}_c$) if it has an above-median share of heroin purchases that were white powder from 2004 to 2011, but results are robust to alternative thresholds. λ_c and γ_t are commuting zone and year fixed effects, respectively, and regressions are population-weighted. Standard errors are clustered at the commuting zone level. The coefficients of interest are ρ_τ, which capture the dynamic differences in changes in mortality for commuting zones exposed to the potency shocks relative to those that were not, normalized so that the year before the initial shock (2011) is set to zero.

We also estimate an average effects version of Equation (1) with

$$Y_{ct} = \beta(\text{White_Powder_Market}_c \times \text{Post}_t) + \lambda_c + \gamma_t + \varepsilon_{ct}, \tag{2}$$

where Post_t is an indicator variable for the period from 2012 to 2019. In this equation, β captures the average mortality impact among commuting zones that are exposed to the potency shocks relative to ones that are not.

We then extend our analysis nationwide by replacing $\text{White_Powder_Market}_c$ in Equations (1) and (2) with an indicator for whether the centroid of each commuting zone lies east of $-91°$ longitude (denoted Eastern_Market_c), and include all contiguous U.S. commuting zones rather than only those covered by HDMP data. We use longitude as a proxy for exposure because Figure 1 shows a clear geographic segmentation of heroin markets around this cutoff, but results are robust to alternative longitude thresholds.

Identification relies on the assumption that, absent potency shocks beginning in 2012, over-

[3]See U.S. vs. López, available at: https://www.justice.gov/archives/opa/press-release/file/1579746/dl.

dose mortality rates would have followed the same trend in white powder and black tar heroin markets. To support this assumption, we examine pre-trends in opioid mortality in both markets. Appendix Figure A.6 further assesses baseline characteristics that could have influenced post-2012 trends, showing that the two types of markets had nearly identical pre-period overdose mortality rates and sociodemographic characteristics (e.g., population and employment rates). One potential concern is that eastern cities may have been more exposed to the OxyContin reformulation in 2010, but pre-reformulation shipment rates were nearly identical as well.

3.2 Dynamic and Average Impacts

We plot raw trends in death rates among commuting zones by heroin market type (white powder versus black tar) in Appendix Figure A.7. Mortality rates from heroin or any illicit opioid trend similarly from 2000 to 2011 for white powder and black tar heroin cities. Starting in 2012, at the same time as the potency shocks in white powder heroin markets, overdose mortality diverges significantly and increases to a much greater extent in cities with white powder relative to black tar heroin markets. This differential increase continues to grow over time.

We summarize these differential changes using our event study specification. The left panel of Figure 2 provides event studies from estimating Equation (1) separately for overdose deaths involving heroin and fentanyl. We see an immediate and dynamic increase in the heroin death rate in white powder heroin markets relative to black tar heroin markets after 2012 that lasts for approximately five years. Afterwards, it slowly declines but remains significant. As the heroin death rate declines in white powder heroin markets, those same markets experience a dynamic and large increase in fentanyl overdose death rates. Appendix Figure A.8 presents event study results for the nationwide analysis, which closely align with these findings.

The right panel of Figure 2 repeats the event study analysis for overdose deaths involving both heroin and fentanyl, only fentanyl, and only heroin. The patterns show fentanyl progressively replacing heroin in white powder heroin markets. Initially, deaths involving only heroin significantly increased. Combined with Section 2, this suggests that early mortality increases were driven by stronger and more variable heroin. However, after 2014, these heroin-only deaths gave way to a rise in deaths involving heroin-fentanyl combinations and eventually fentanyl alone. This dynamic aligns with ethnographic accounts that white powder heroin enabled fentanyl's entry into U.S. markets—first as an adulterant to white powder heroin, and later as a standalone product once heroin users developed tolerance to fentanyl's much greater potency, information spread, and some heroin users began to seek it out (Mars et al. 2019).

We next estimate Equation (2) and report the average mortality impacts of the potency shocks in Table 1.[4] Column (1) shows that heroin overdose deaths rose by 2.961 per 100,000—a 226% increase relative to the pre-treatment mean. Column (2) indicates an even larger effect for fentanyl, with deaths increasing by 5.939 per 100,000, or 990%. Column (3) confirms that the surge is concentrated in overdoses involving either heroin or fentanyl. By contrast, Column (4) yields a small, statistically imprecise decline in non-opioid overdose deaths. Finally, Column (5) shows that all-cause overdose mortality increased by 5.881 per 100,000, demonstrating that

[4]Appendix Tables B.1 and B.2 use alternative definitions of pre-potency shock white powder heroin markets. Appendix Table B.3 presents the nationwide equivalent of Table 1.

the rise in overall overdose mortality was overwhelmingly driven by illicit opioids.

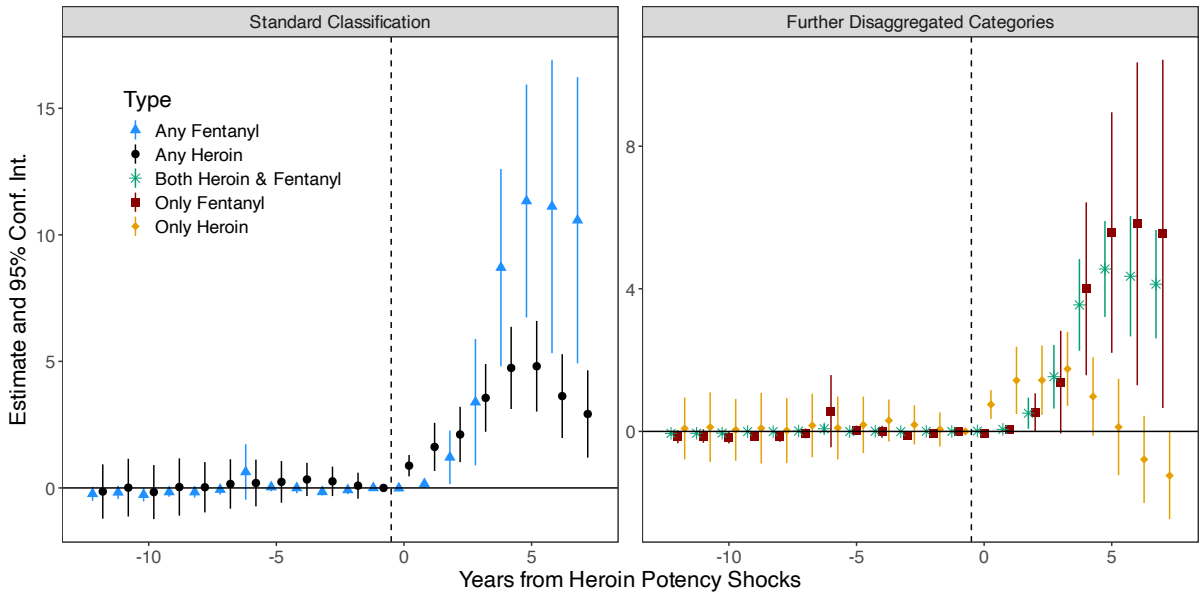


Figure 2: Dynamic Mortality Impacts of Exposure to Potency Shocks

Notes: These figures present the ρ_t coefficients from estimating Equation (1). The White Powder Heroin treatment indicator variable is defined as having an above median share of total pre-potency shock heroin purchases being white powder. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes 27 commuting zones where we have HDMP data on heroin type. The left panel shows deaths involving heroin and deaths involving fentanyl separately, while the right panel breaks these down further into deaths involving only fentanyl (i.e., no heroin present), only heroin, and both heroin and fentanyl.

Table 1: Average Mortality Impacts of Exposure to Potency Shocks

	Any Heroin (1)	Any Fentanyl (2)	Heroin or Fentanyl (3)	Non-Opioids (4)	Any Drug (5)
(White Powder Market)X(Post)	2.961***	5.939***	6.527***	-0.0855	5.881***
	(0.6712)	(1.374)	(1.448)	(0.5971)	(1.660)
Pre-treatment mean	1.31	0.600	1.89	4.85	11.2
R^2	0.776	0.632	0.725	0.790	0.775
Observations	540	540	540	540	540

Notes: This table presents the β coefficients from estimating Equation (2). The White Powder Heroin treatment indicator variable is defined as having an above median share of total pre-potency shock heroin purchases being white powder and Post is an indicator for 2012 and after. The sample is commuting zones that had a city that participated in the Heroin Domestic Monitor Program (HDMP). The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000.

3.3 Robustness Checks

We test alternative explanations by re-estimating Equation (2) with controls for potential drivers of rising overdose death rates in Appendix Figures A.9 and A.10. Controlling for exposure to OxyContin reformulation using pre-reformulation shipments—whether entered continuously or as above/below median interacted with a post-2010 indicator—yields no attenuation. Adding controls for must-access PDMPs (Kim 2021; Balestra et al. 2021), enforcement intensity (officers

per 100,000 from LEOKA), or fentanyl smuggling via trade by interacting per capita imports with a Post-2012 indicator, following Moore et al. (2023), likewise leaves coefficients stable.[5]

Finally, we estimate models with a triple interaction between pre-reformulation OxyContin shipments, white powder heroin markets, and the post-reformulation period, along with all corresponding double interactions (Appendix Tables B.5 and B.6). This specification addresses two concerns: that fentanyl adulteration may have been endogenous to the OxyContin reformulation, and that white powder heroin was a closer substitute for OxyContin than black tar. Across these models we again find no attenuation: mortality increases are driven by exposure to white powder heroin potency shocks, independent of prior OxyContin use. If anything, the negative coefficient on the triple interaction suggests increases in heroin and fentanyl mortality were largest in eastern markets with less pre-existing OxyContin use. Appendix Figure A.11 and Tables B.7 and B.8 show similar results for the nationwide sample.

Beyond regression-based checks, a permutation test in Appendix Figure A.12 confirms that the observed increases in overdose mortality in white powder markets are highly unlikely to arise by chance: our estimated effects lie above the 99th percentile of placebo distributions.

3.4 Primary Mechanism: Rising Mortality Risk for Heroin Users

We next examine whether heroin potency shocks increased overdose risk among existing users. Our first test asks whether white powder heroin markets with higher pre-2012 heroin use experienced larger post-2012 mortality increases, as would be expected if potency shocks disproportionately raised risk among existing users. We proxy baseline use with pre-shock heroin-overdose death rates, measured over 2005–2011 and, for robustness, 2005–2009 (pre-OxyContin reformulation). Consistent with this mechanism, Panel A of Appendix Table B.9 shows that within white powder heroin markets, each additional pre-shock heroin death per 100,000 is related to a 38% larger post-2012 increase in overdose deaths due to heroin potency shocks. The gradient is even steeper for fentanyl-related and total opioid mortality. Results are similar when baseline use is measured with pre-reformulation death rates (Panel B).

We also estimate if annual overdose risk conditional on heroin use changed, combining national data on heroin overdose deaths from NCHS with past-year heroin use from the National Survey on Drug Use and Health (NSDUH) (see Appendix C). Figure 3 presents trends in the ratio of heroin overdoses to the estimated number of heroin users from 2002 to 2019.[6] We overlay them with a regression that includes a pre-2012 slope, a level increase after 2012, and a post-2012 change in slope. Before 2012, the estimated annual probability of overdose death among past-year heroin users was stable at about 0.73%, which is roughly consistent with pre-fentanyl epidemiological studies that placed the risk around 0.52% (Keyes et al. 2022). At the onset of potency shocks in 2012, risk rose sharply: a 0.33 percentage-point level increase followed by 0.20 points per year, implying a 232% total rise in annual mortality risk after 2012.

[5] Because only state-level trade data are available, we assign values to all CZs within a state. Appendix Table B.4 shows state-level results, which are similar.

[6] Because annual state-level NSDUH data are not publicly available, we cannot disaggregate these estimates to compare white powder and black tar heroin markets.

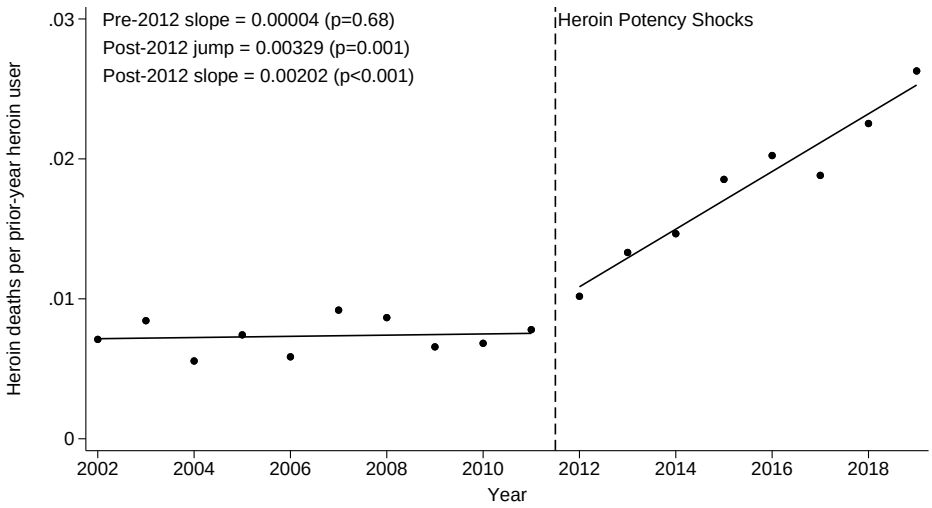


Figure 3: Yearly Trends in Ratio between Heroin Use (NSDUH) and Overdose Mortality (CDC)

Notes: This figure shows the estimated annual probability of heroin overdose death conditional on any prior-year heroin use. Heroin mortality counts are from CDC Vital Statistics; heroin use estimates are based on self-reported past-year use from the National Survey on Drug Use and Health (NSDUH). The plotted series reflects the ratio of overdose deaths to estimated users in each year. Estimates are derived from a segmented linear regression with a breakpoint at 2012. The results indicate a statistically significant level shift of 0.33 percentage points ($p = 0.001$) in 2012, followed by an additional yearly increase of 0.20 percentage points ($p < 0.001$), relative to a flat pre-2012 trend. These findings are consistent with a sharp and sustained rise in the lethality of the heroin supply beginning in 2012.

3.5 The Changing Geography and Demographics of the Opioid Epidemic

Exposure to potency shocks through the geographically segmented heroin market helps explain two longstanding puzzles: why eastern states bore the brunt of illicit opioid mortality increases and why deaths surged after 2012 among groups with little exposure to prescription opioids. Appendix Figure A.13 shows the shifting geography. From 2000 to 2009, increases in prescription opioid mortality tracked state-level OxyContin misuse with no East–West differences, consistent with prior evidence on OxyContin marketing (Alpert et al. 2022; Arteaga and Barone 2025). From 2010 to 2019, however, OxyContin misuse no longer predicted rising heroin and fentanyl mortality, and eastern states experienced mortality increases averaging 15.8 deaths per 100,000 more than western states, regardless of prior misuse. If the reformulation alone had driven the epidemic, mortality would have continued to track OxyContin misuse. Instead, the sharp East–West break aligns with our potency-shock explanation.

Potency shocks also explain why mortality rose sharply among groups less affected by prescription opioids. Appendix Figure A.14 shows that while prescription opioid deaths were much higher for Whites before 2012, heroin mortality levels and trends were similar for Whites and Blacks. Thus, Blacks—despite lower prescription opioid exposure—saw large mortality increases once heroin potency rose in eastern markets. Stratified regressions (Appendix Table B.10) confirm broadly similar effects for Whites, Blacks, and Hispanics, consistent with comparable pre-shock heroin death rates.

4 The Overall Determinants of Heroin and Fentanyl Mortality

4.1 Revisiting the OxyContin Reformulation and PDMPs

In response to the first wave of the opioid epidemic, policymakers tightened prescription opioid supply. A prominent literature argues that these interventions—especially the abuse-deterrent reformulation of OxyContin (Alpert et al. 2018; Evans et al. 2019) and must-access Prescription Drug Monitoring Programs (PDMPs) (Kim 2021; Balestra et al. 2021)—backfired by shifting demand first to heroin (Alpert et al. 2018; Evans et al. 2019) and later to fentanyl (Powell and Pacula 2021). These accounts largely overlook contemporaneous potency shocks that arose around the reformulation (2010) and the PDMP roll-outs (post-2012). Re-examining these policy interventions while explicitly accounting for potency shocks is therefore essential, both to examine their independent impacts and to reassess claims about unintended consequences of policy interventions if prior estimates conflate policy effects with exogenous supply-side shifts.

We begin by replicating and extending the state-level design of Alpert et al. (2018) and Powell and Pacula (2021), which relates post-2010 overdose mortality to pre-reformulation OxyContin misuse (2004–2009 NSDUH) interacted with year effects. Our extension augments that specification with an indicator for states east of –91° longitude interacted with year effects to proxy exposure to potency shocks. Regressions are population-weighted with state-clustered standard errors; the full equation and estimation details are in Appendix D.

Appendix Figure A.15 Panel A reproduces the headline finding in specifications without potency-shock controls: heroin deaths rise sharply after the reformulation in states with higher baseline OxyContin misuse beginning in 2011. Once we control for potency shocks, however, the estimates attenuate substantially. Effects remain in 2011–2012 but decline sharply thereafter. On average across post-reformulation years, they fall by 62% and lose statistical significance.

Two concerns could understate reformulation effects. First, OxyContin misuse may correlate with other factors, such as broader prescribing patterns. Panel B therefore adds interactions of general pain-reliever misuse (2004–2009 NSDUH) with year effects (Alpert et al. 2018; Beheshti 2019; Park 2025). Baseline coefficients rise modestly, but attenuation after adding potency-shock controls remains about 58%. Second, NSDUH misuse measures may be subject to measurement error. Following Alpert et al. (2018), Panel C instruments 2008–2009 misuse with 2004–2005 misuse as in (Alpert et al. 2018); the IV estimates again attenuate by roughly 57% when potency shocks are included. Instrumenting for 2004-2009 OxyContin misuse with exact ARCOS OxyContin shipments (2006-2009) yields similar conclusions (66% attenuation).

We also test whether the reformulation itself catalyzed the potency shocks—e.g., by pulling vulnerable users into heroin markets or facilitating fentanyl's spread—via a triple interaction among pre-reformulation OxyContin exposure, geography, and year effects. The negative coefficient (Appendix Tables B.5 and B.6) rejects this pathway, indicating that fentanyl's rise was not endogenous to the reformulation. Complementing this, Appendix Figure A.16 shows that while western states exhibit modest post-reformulation increases where shipments were high, eastern states—exposed to potency shocks beginning in 2012—experienced much larger mortality increases regardless of baseline OxyContin shipments.

Taken together, the reformulation's impact on heroin mortality appears to be about half as

large as previously reported. There remains a detectable effect in 2011–2012 (especially in the West), but longer-run impacts are attenuated and imprecisely estimated. Analogous analyses for fentanyl and any illicit opioids (Appendix Figures A.17 and A.18) yield parallel results: controlling for potency shocks reduces estimated reformulation effects by 40 to 54%.

We conduct a parallel exercise for must-access PDMPs. Details are in the Methods Appendix and results appear in Appendix Figures A.19, A.20, and A.21. After controlling for potency shocks, estimated PDMP effects attenuate sharply—generally to the null—for heroin, fentanyl, and any illicit opioid mortality. Overall, once contemporaneous supply-side potency shocks are accounted for, the evidence suggests a more limited role for these policy interventions in driving the rise in illicit opioid deaths than the prior literature implies.

4.2 Decomposition of Illicit Opioid Mortality Trends

To decompose the determinants of rising illicit opioid mortality, we estimate state-level regressions including exposure to the OxyContin reformulation, PDMPs, and heroin potency shocks (see Appendix D). We then simulate counterfactual trends by sequentially setting each effect to zero. Figure 4 plots observed national death rates against these counterfactuals. Removing the reformulation lowers mortality in 2011–2012, but rates still surge after the 2012 potency shocks; must-access PDMPs have modest effects. By contrast, eliminating potency shocks flattens post-2013 trends, underscoring their central role. By 2019, potency shocks account for 46% of elevated illicit opioid mortality, compared to 36% for the reformulation and 19% for PDMPs, though PDMP estimates are imprecise (Appendix Table B.11).

5 Conclusion

This paper provides the first causal evidence that increased variability in heroin purity and its growing adulteration with fentanyl were central to rising U.S. overdose mortality after 2012. The findings highlight a fundamental economic problem in drug markets: when product quality is only revealed through consumption, abrupt shifts in potency create extreme risks in markets with narrow safety margins. These shocks can also have lasting effects when substances are addictive and information spills over across users (Cutler and Donahoe 2024), embedding new products like fentanyl in the market. More broadly, the results show how volatility in illicit drug supply can generate large and persistent public health crises, pointing to the potential value of systems that rapidly detect and communicate emerging potency risks.

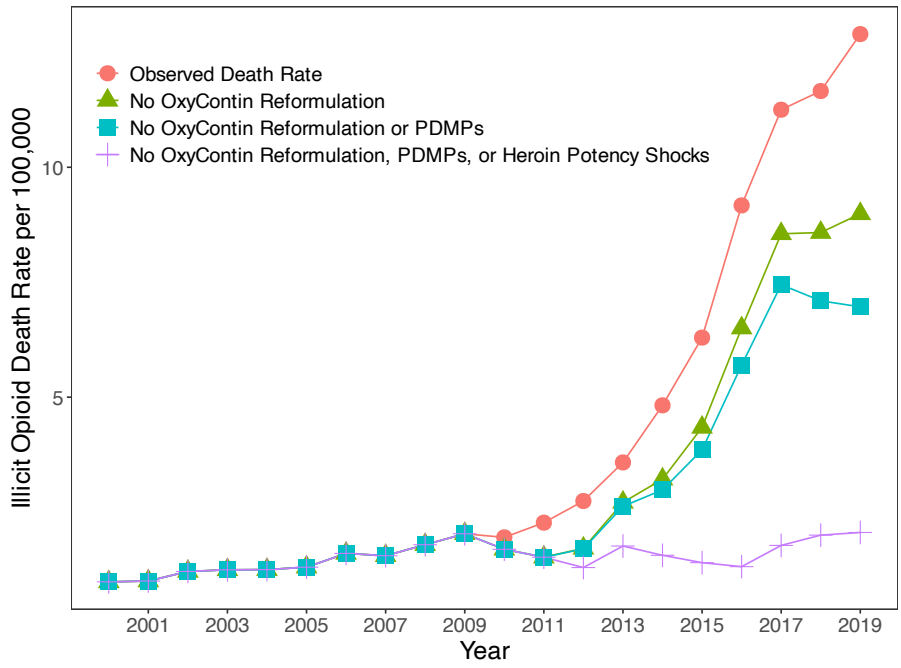


Figure 4: Counterfactual Trends in Illicit Opioid Mortality Rates With vs. Without Exposure to Legal Market Interventions or Heroin Potency Shocks

Notes: This figure presents trends in national illicit opioid (heroin or fentanyl) death rates from 1999 to 2019 and counterfactual trends under three separate scenarios: (1) if OxyContin had not been reformulated (green line with triangle markers), (2) if must-access PDMPs had also not been implemented (blue line with square markers), and (3) if there had also not been an increase in the variability of heroin purity following the transition of heroin production from Colombian to Mexico or supply-side adulteration of heroin with fentanyl (purple line with pluses). We estimate these counterfactuals using estimates of Equation (D.2).

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A Additional Figures

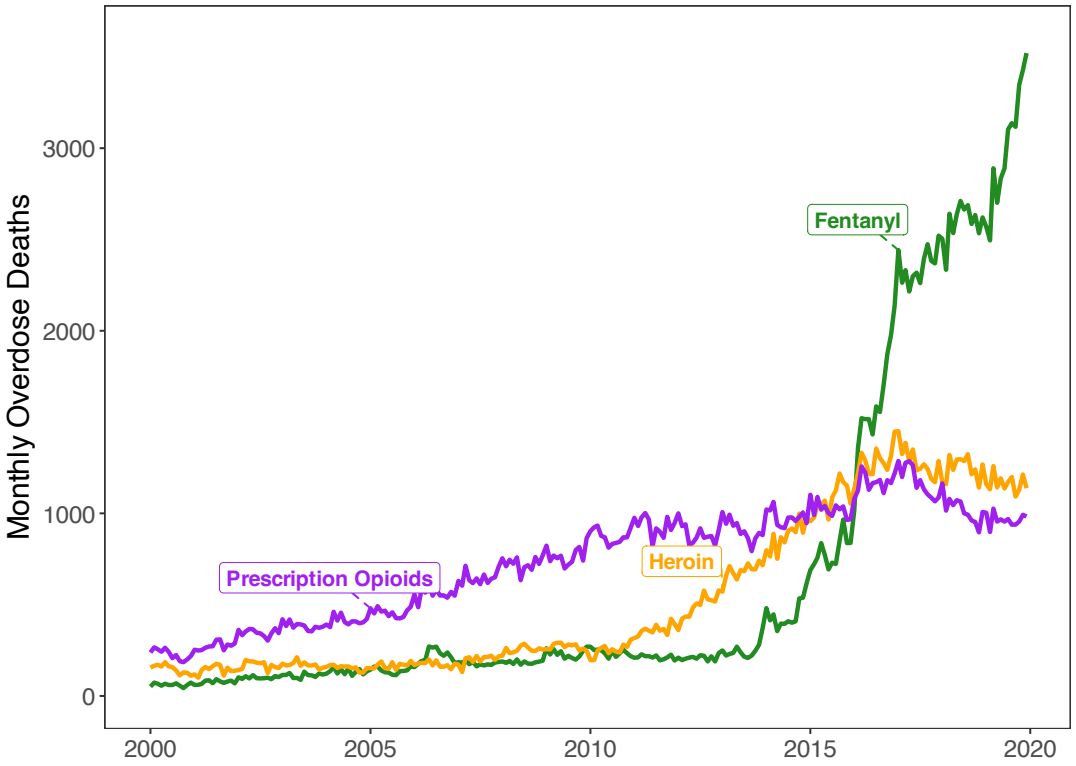


Figure A.1: Monthly U.S. Overdose Deaths by Opioid Type

Notes: Monthly mortality data are from the National Vital Statistics System (NVSS). We use the following ICD-9 codes for overdose death types: T40.2 for prescription opioids, T40.1 for heroin, and T40.4 for fentanyl.

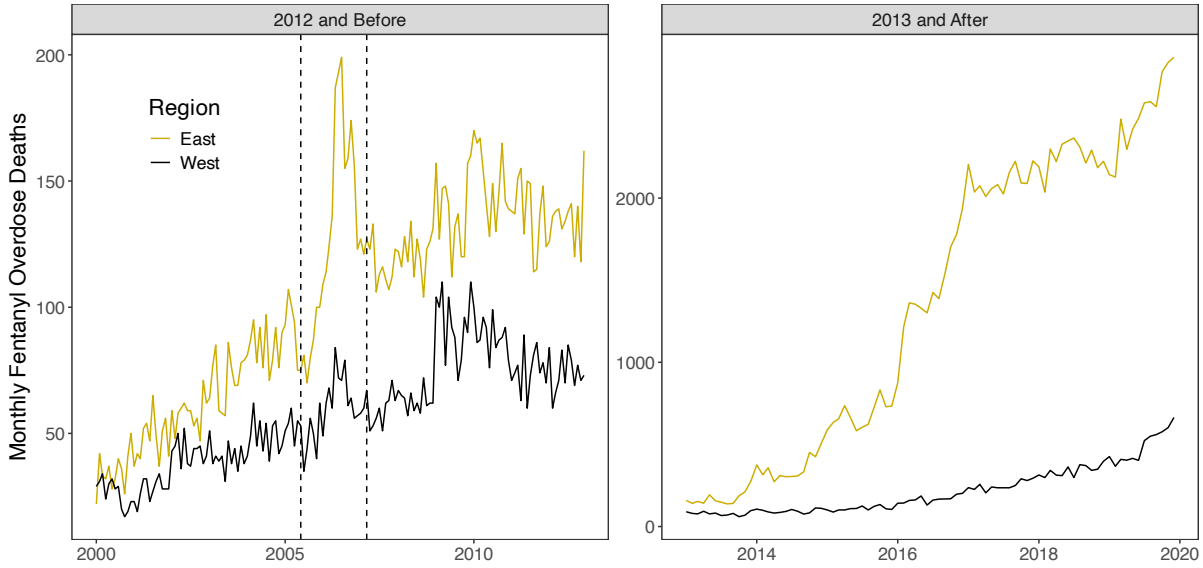
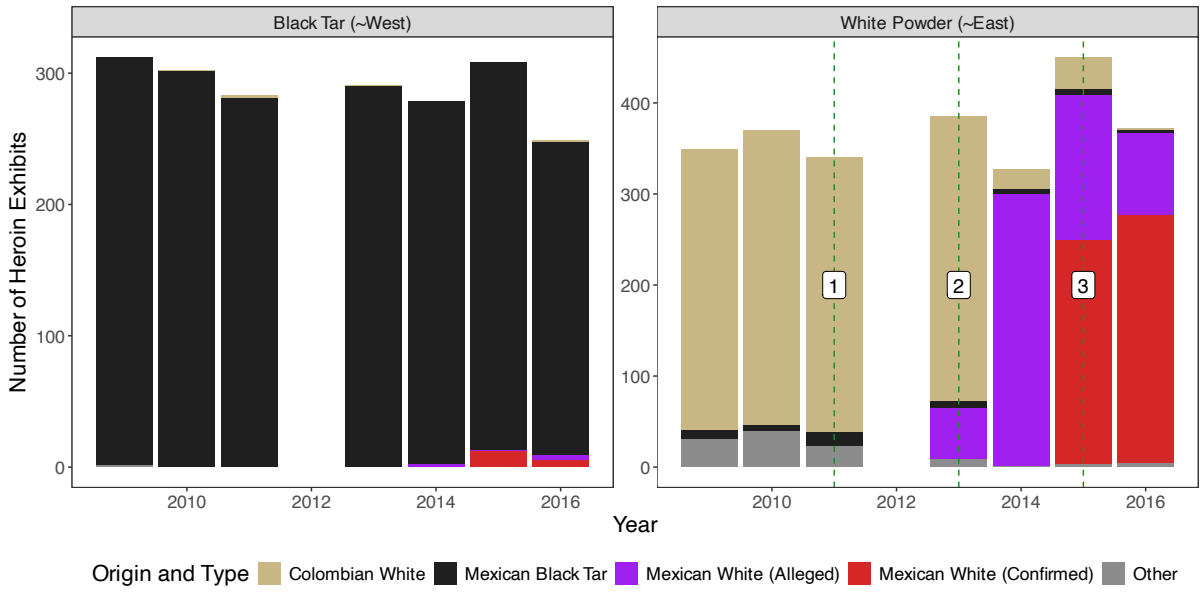


Figure A.2: Monthly Fentanyl Overdose Deaths

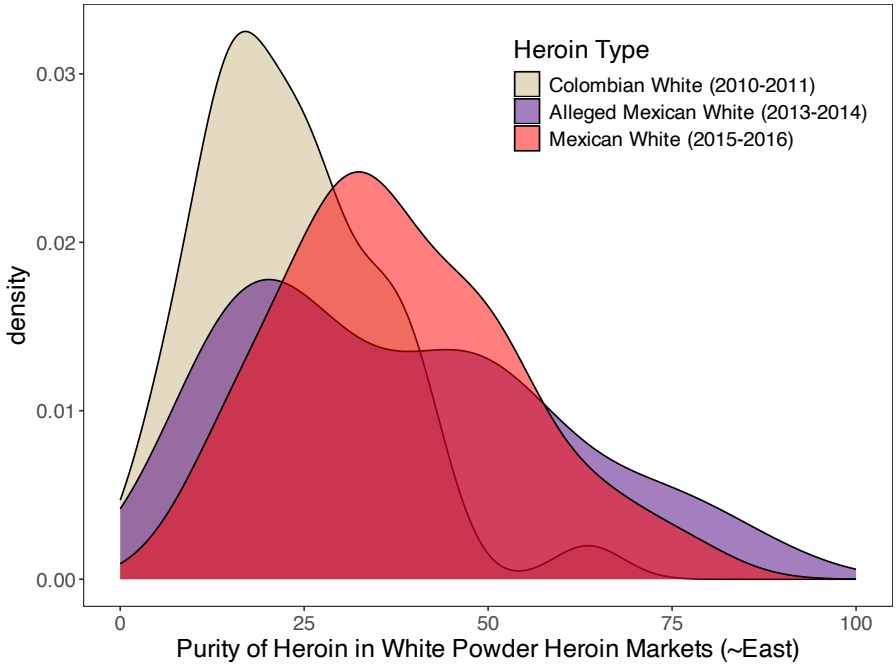
Notes: Data are from the National Vital Statistics System, where we use the ICD-9 code of T40.4 for fentanyl, and the delineation for East and West is the Mississippi River. The left panel highlights the short fentanyl epidemic that occurred when Mexican DTOs made a first attempt at introducing fentanyl in white powder heroin markets (between the two hashed vertical lines). They were a small player in these markets at the time and it led to a spike in fentanyl deaths in eastern cities that ended after the lab that manufactured the fentanyl in Toluca, Mexico was raided in May 2006 (CDC 2008).

Figure A.3: Heroin Origin, Type, and Purity

Panel A: Trends in Heroin Origin and Type Across Markets



Panel B: Purity Distributions by White Powder Heroin Type



Panel A Notes: Data come from the Heroin Domestic Monitoring Program (HDMP). This figure presents the total exhibits by market, type, and year; 2012 is excluded due to lack of an HDMP report. In the right panel, 1. In 2011, the DEA first reports suspicions of Mexican DTOs mixing Mexican and Colombian heroin or producing white powder heroin that mimics Colombia processing methods, but that Colombian white powder remains the dominant type. 2. In 2013, the DEA first identifies suspected Mexican white powder heroin and inconclusive origin heroin that mimics Colombian heroin in the HDMP report. 3. In 2015, the DEA first implements a test to confirm Mexican white heroin.

Panel B Notes: Data come from the Heroin Domestic Monitoring Program (HDMP). This figure shows the distribution of purity of Colombian white heroin just before the Mexican DTO entry (2010-11), suspected Mexican white heroin during the first two years it is reported (2013-2014), and confirmed Mexican white heroin after testing was available (2015-2016). Appendix Figure A.4 presents yearly trends in average heroin purity and the 90th percentile of purity across black tar and white powder heroin markets.

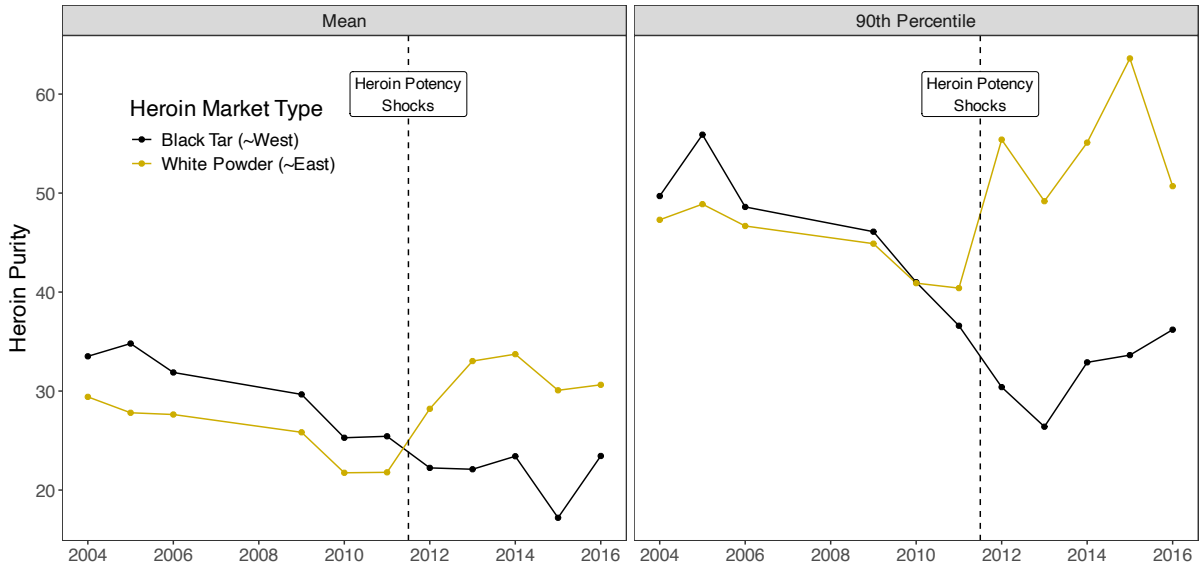
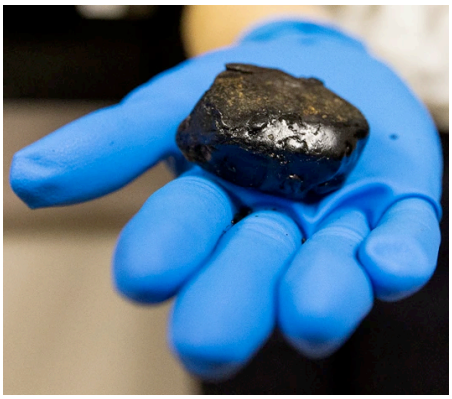


Figure A.4: Trends in Heroin Purity by Market Type

Notes: Data come from the Heroin Domestic Monitoring Program (HDMP) and the figure shows the yearly trends in heroin purity by market. The two measures are the average and the 90th percentile.

Figure A.5: Images of Black Tar Heroin, White Powder Heroin, and Fentanyl

Panel A: Black Tar Heroin



Panel B: White Powder Heroin (left) and Illicitly-Produced Fentanyl (right)



Notes: Images are obtained from here, here, and here, respectively.

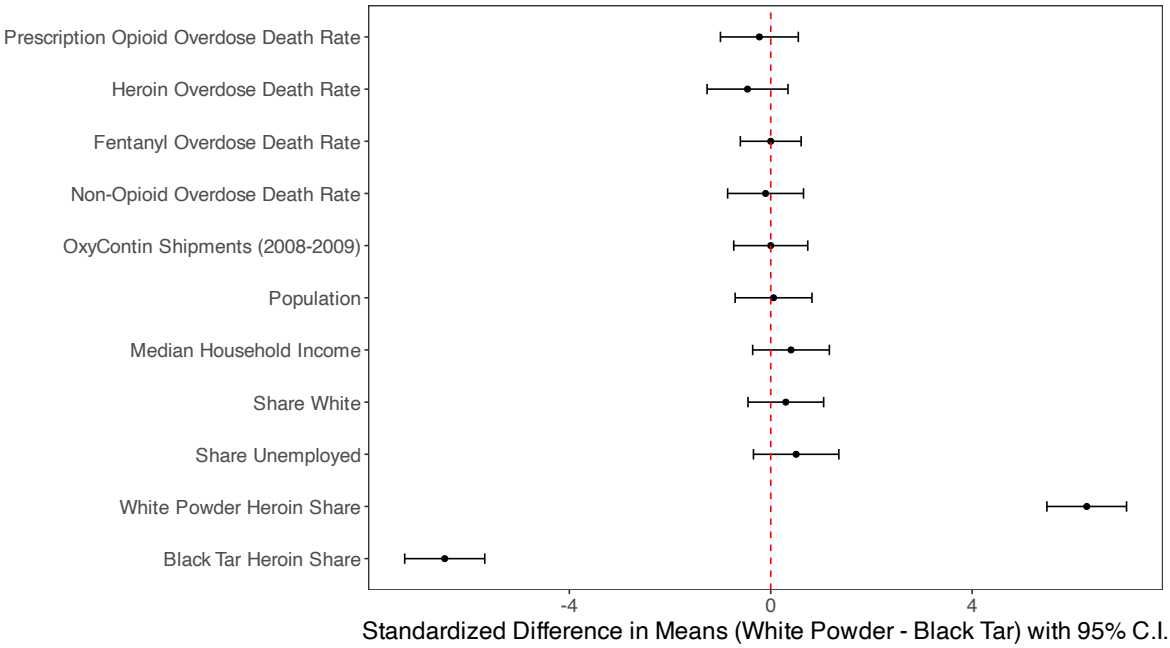
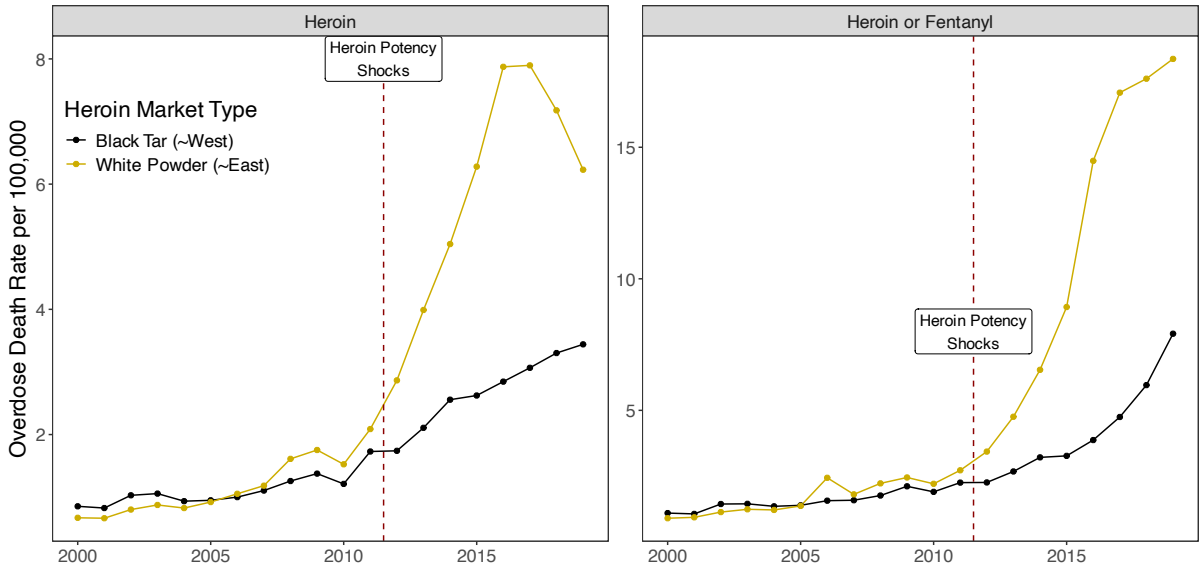


Figure A.6: Characteristics of White Powder and Black Tar Heroin Markets in 2011

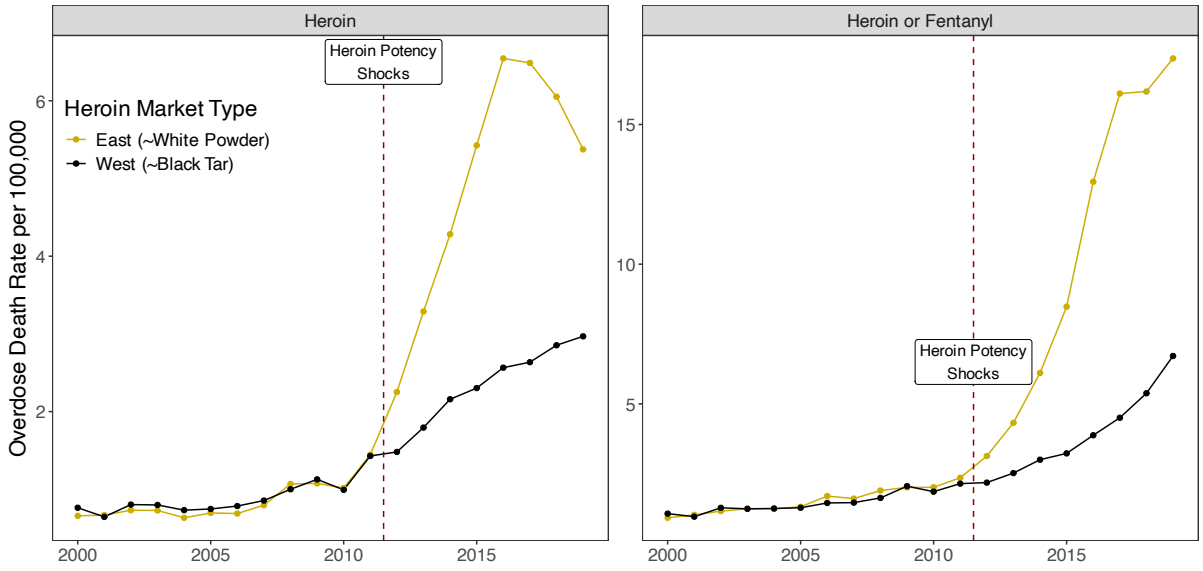
Notes: This figure uses CDC mortality data and ACS census data from 2011, the year before the potency shocks, to compare our treated and control commuting zones for the HDMP sample. The final two rows, White Powder and Black Tar Heroin Shares, use the DEA's HDMP data from 2004 to 2011.

Figure A.7: Yearly Illicit Opioid Overdose Death Rates by Market Type

Panel A: HDMP Sample



Panel B: Nationwide Sample



Notes: This figure presents illicit opioid overdose death rates over time using National Vital Statistics System's restricted mortality data. In Panel A, we limit the sample to commuting zones that had a city that participated in the HDMP. In Panel B, we use data for the contiguous US and define heroin markets by whether they are East or West of the Mississippi River (longitude of -91).

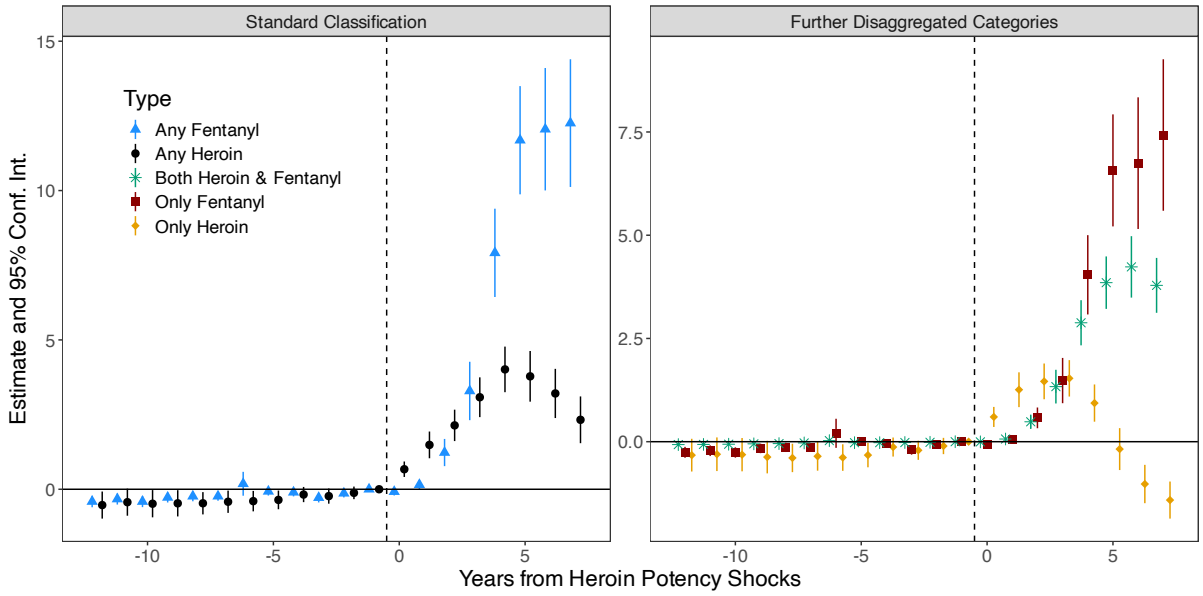


Figure A.8: Dynamic Mortality Impacts of Potency Shocks (Nationwide)

Notes: These figures present the ρ_t coefficients from estimating Equation (1). The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes all commuting zones in the contiguous United States.

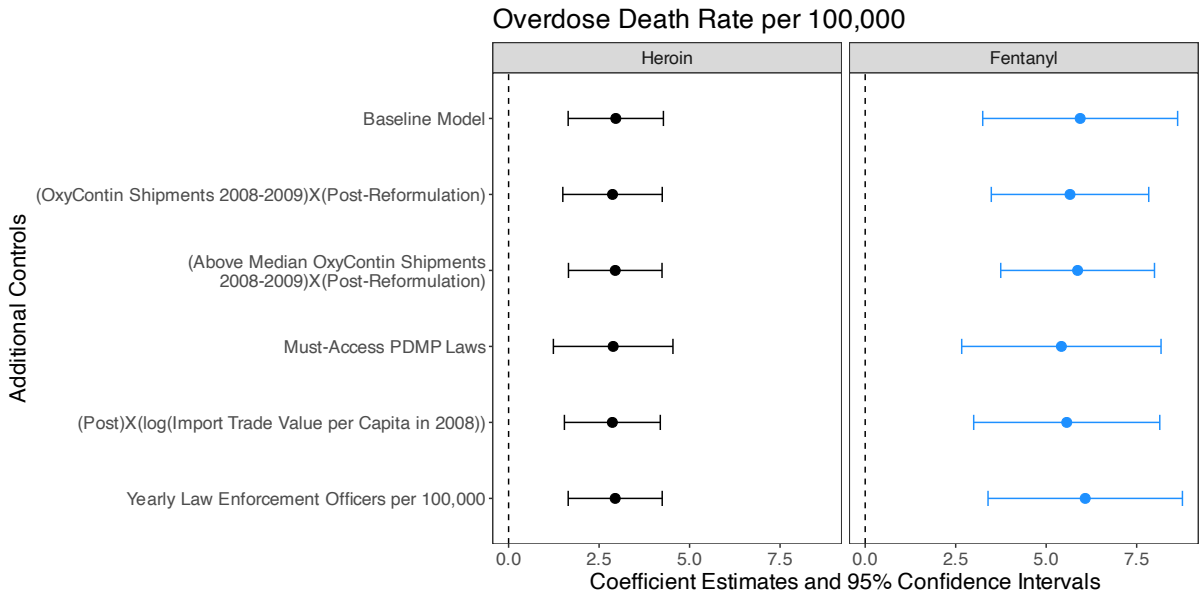


Figure A.9: Average Mortality Impacts of Exposure to Potency Shocks with Additional Controls

Notes: This figure presents the β coefficients from estimating Equation (2) with different controls. Each control is added to the baseline model and estimated independently, except for the triple interaction term, which includes an additional interaction term. The White Powder Heroin treatment indicator variable is defined as having an above median share of total pre-potency shock heroin purchases being white powder, and Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 (left for heroin, right for fentanyl) and the sample includes only commuting zones where we have HDMP data. Appendix Tables B.5 and B.6 are the table versions, and Appendix Figure A.10 provides the associated event studies.

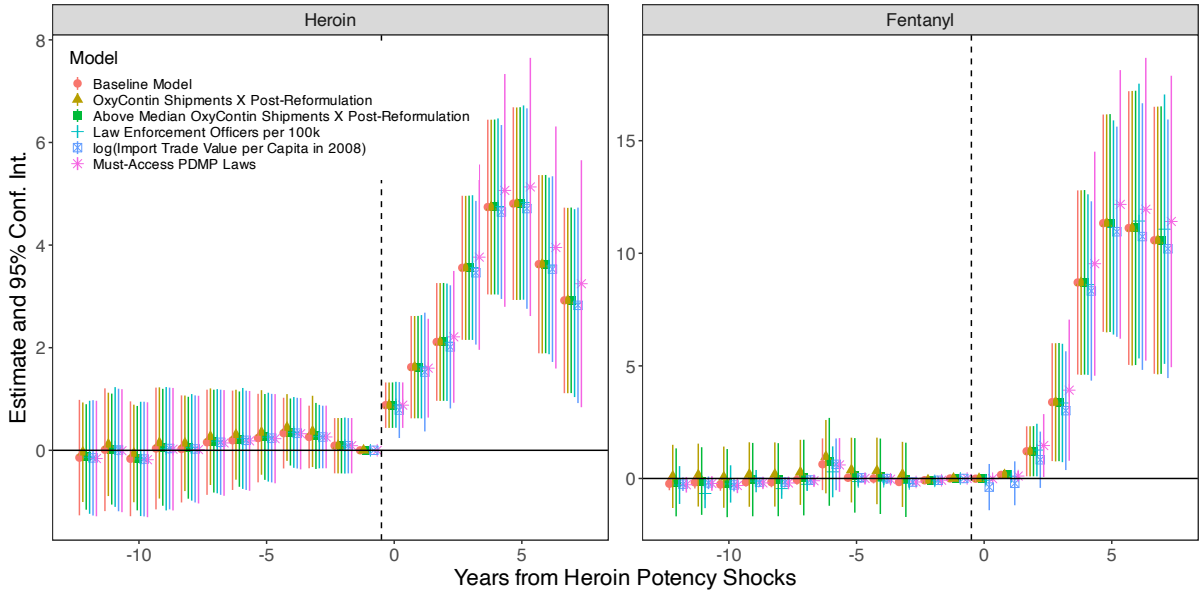


Figure A.10: Dynamic Mortality Impacts of Potency Shocks with Additional Controls

Notes: These figures present the ρ_t coefficients from estimating Equation (1) with additional controls, i.e., the baseline model plus a control variable. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes 27 commuting zones where we have HDMP data on heroin type.

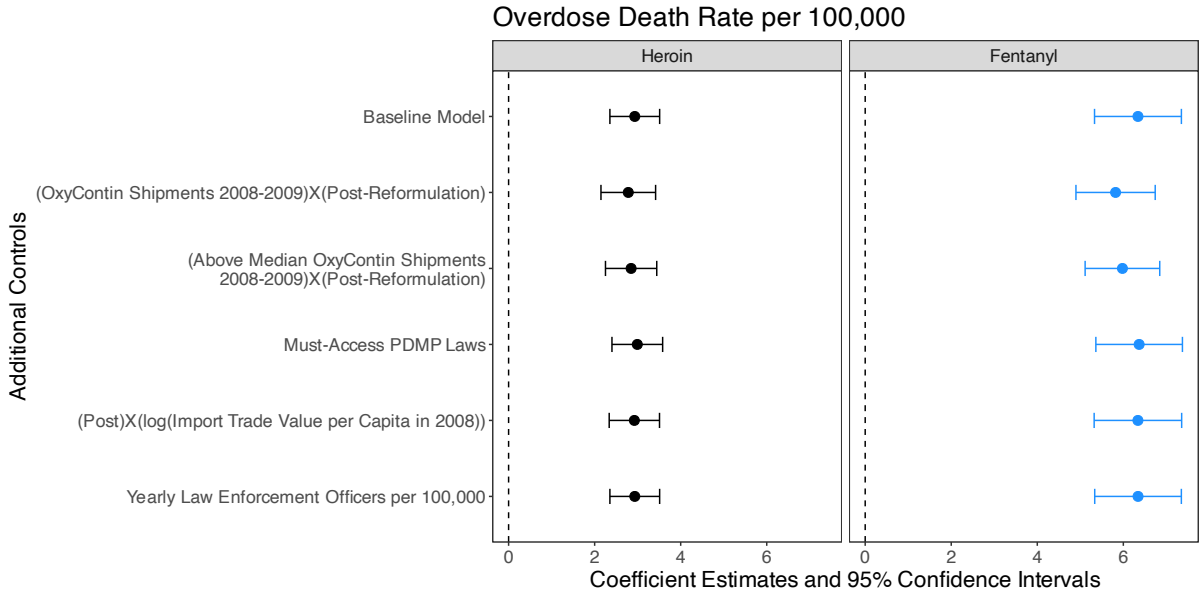


Figure A.11: Average Mortality Impacts of Potency Shocks with Additional Controls (Nationwide)

Notes: This figure presents the β coefficients from estimating Equation (2) with different control variables. The Eastern Market binary treatment variable is equal to one for commuting zones east of -91 degrees longitude and Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes all commuting zones in the contiguous United States.

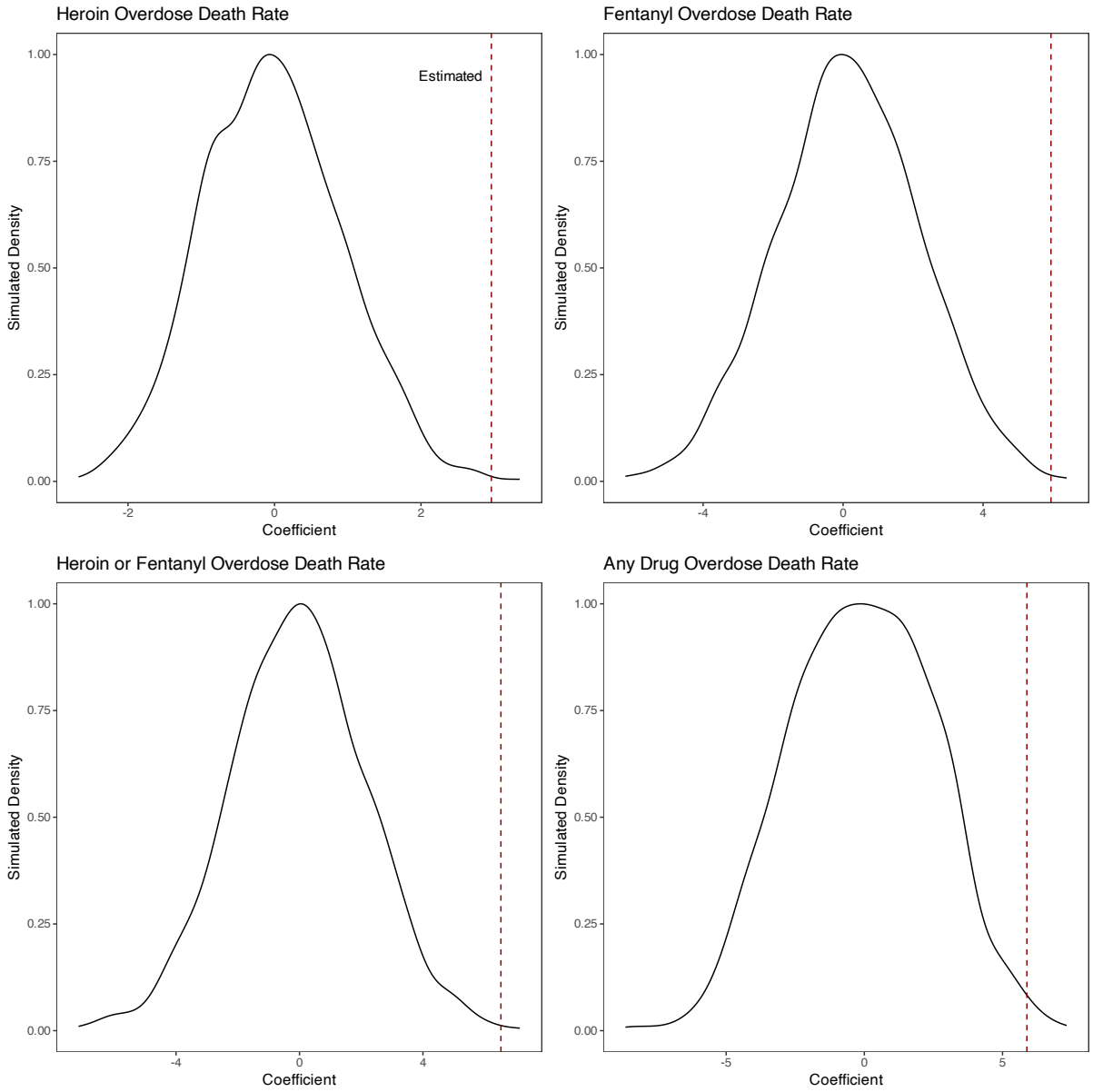
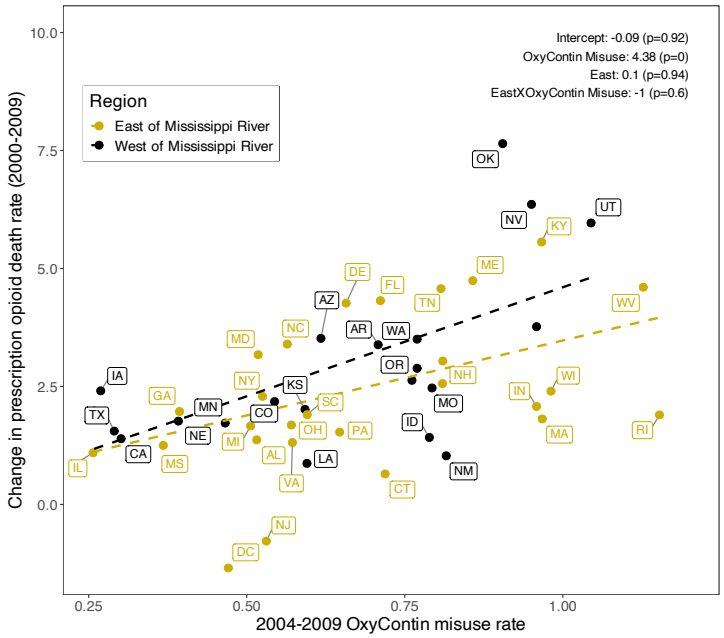


Figure A.12: Permutation Tests for HDMP Market Analysis

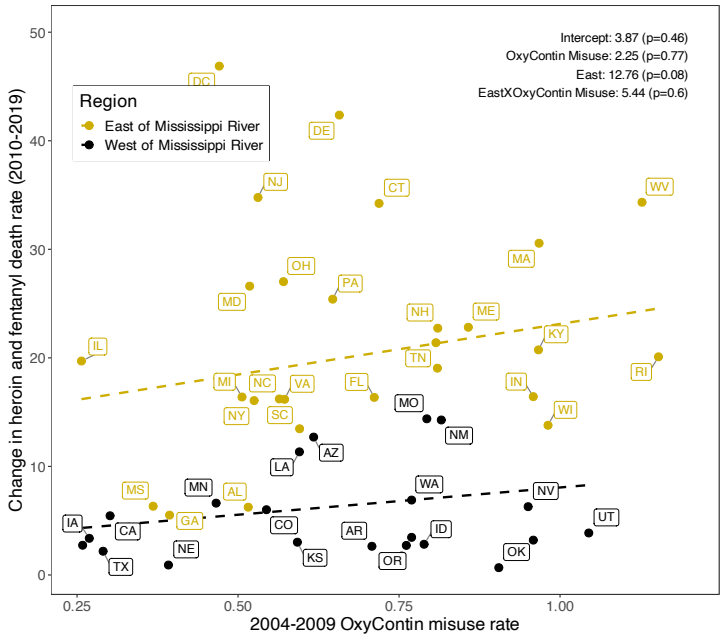
Notes: For these tests, we randomize which commuting zone gets "treated" and then run Equation (2) 1000 times using a new sample each time. The figures present the distribution of coefficients obtained from these regressions. The dotted vertical red line is the estimated coefficient.

Figure A.13: Geography of Epidemic during the Prescription Opioid and Illicit Opioid Waves

Panel A: Change in Prescription Opioid Death Rate (2000-2009)



Panel B: Change in Illicit Opioid Death Rate (2010-2019)



Notes: In both panels, the x-axis uses the state-level NSDUH measure of the OxyContin misuse rate from 2004 to 2009 found in Alpert et al. (2018). The y-axis in Panel A is the change in prescription opioid overdose deaths before the OxyContin reformulation (from 2000 to 2009). In Panel B, it is the change in illicit opioid overdose deaths (heroin and fentanyl) after the OxyContin reformulation (from 2010 to 2019). We also include the results from a simple linear regression in the top right of each figure, where we regress the change in death rates on the misuse measure, whether the state is east of the Mississippi river, and their interaction.

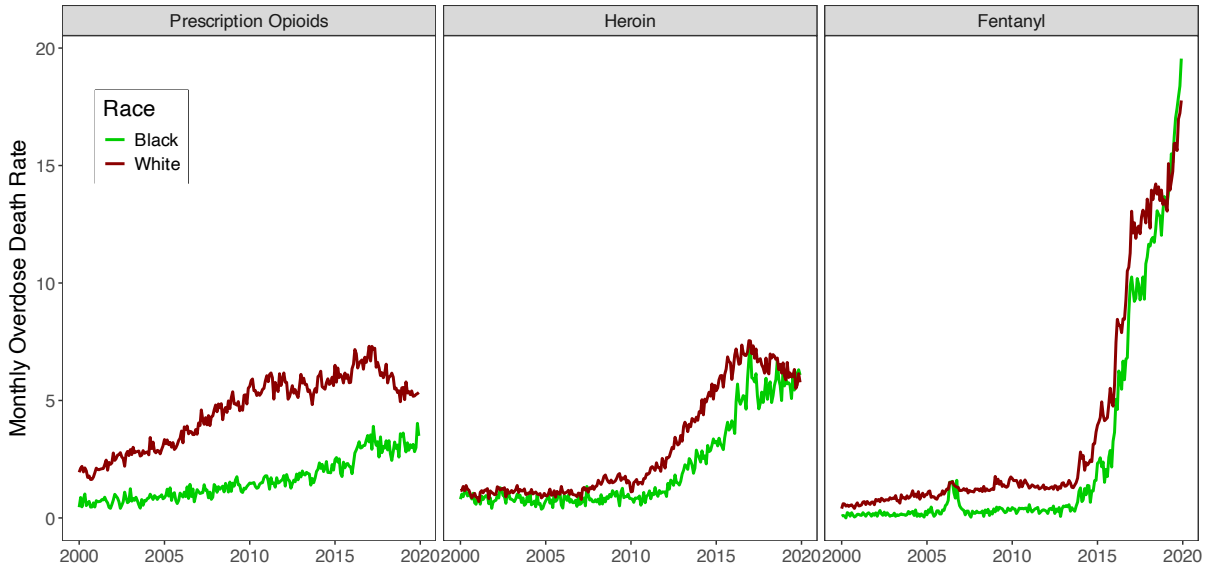
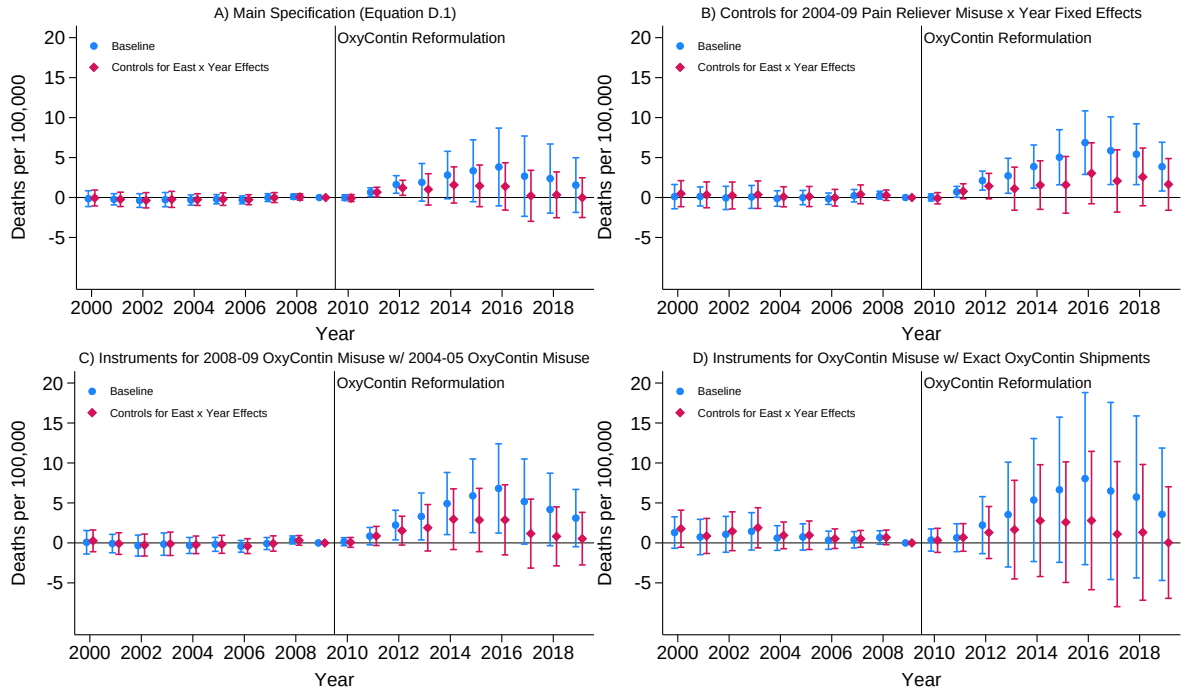


Figure A.14: Monthly Trends in Opioid Overdose Deaths by Race and Opioid Class

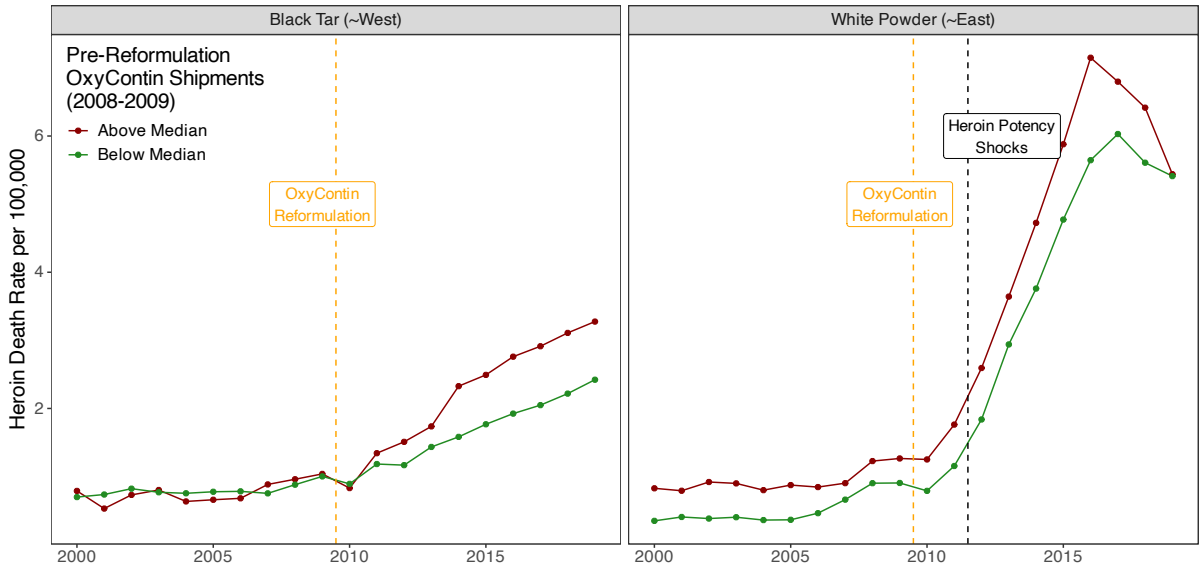
Notes: Monthly mortality data are from the National Vital Statistics System (NVSS). We use the following ICD-9 codes for overdose death types: T40.2 for prescription opioids, T40.1 for heroin, and T40.4 for fentanyl. Rates are calculated separately by race and opioid class.

Figure A.15: Replication and Extension of OxyContin Reformulation's Impacts on Heroin Mortality



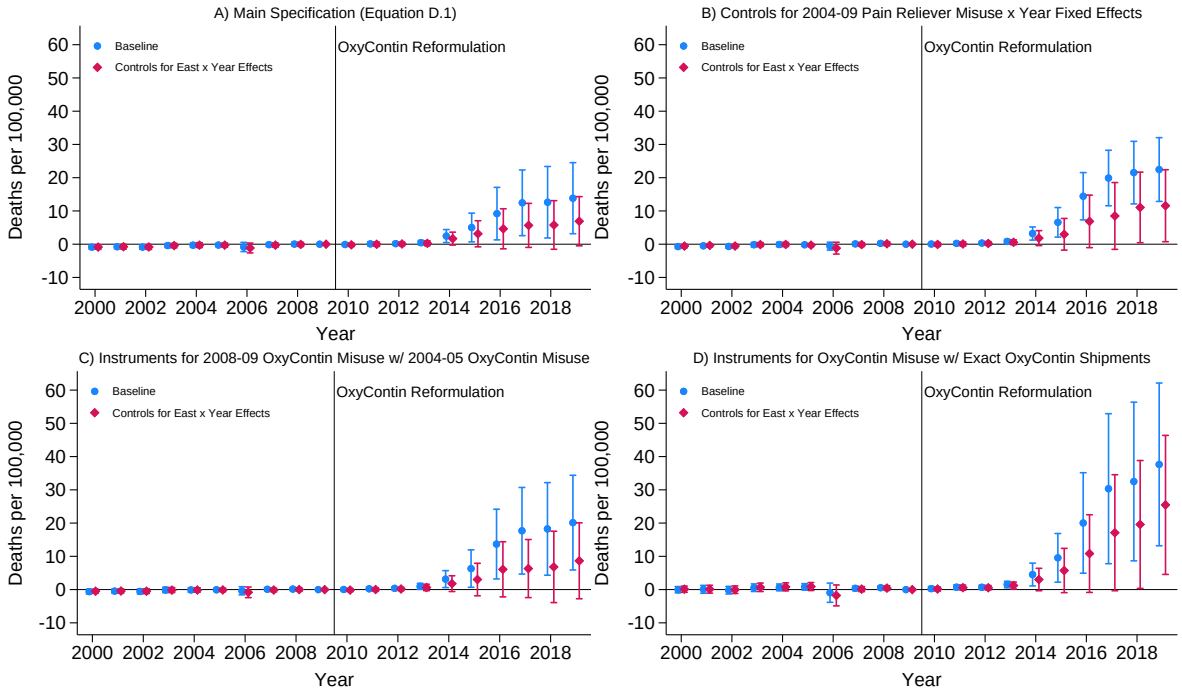
Notes: This figure presents estimates of the impact of one percentage point higher state OxyContin misuse prevalence on heroin overdose mortality rates each year relative to when OxyContin was reformulated to be abuse-deterrent (2010). Each panel presents estimates with and without controlling for an interaction between being an eastern state and year fixed effects. Panel A replicates and extends the baseline specification (Equation 1) in Alpert et al. (2018) (Equation 3 in this manuscript). Panel B adds controls for 2004-2009 pain reliever misuse prevalence interacted with year fixed effects, to adjust for the fact that pre-reformulation OxyContin misuse is non-random (Alpert et al. 2018; Beheshti 2019; Park 2025). Panel C uses 2008-2009 OxyContin misuse prevalence (rather than 2004-2009 OxyContin misuse) and instruments for it using 2004-2005 OxyContin misuse prevalence to address measurement error in OxyContin misuse prevalence (Alpert et al. 2018). Panel D instruments for 2004-2009 OxyContin misuse with 2006-2009 state OxyContin shipment rates (morphine equivalent grams per capita). See Section 4 of the text for more details.

Figure A.16: Heroin Mortality By Market Type and OxyContin Exposure



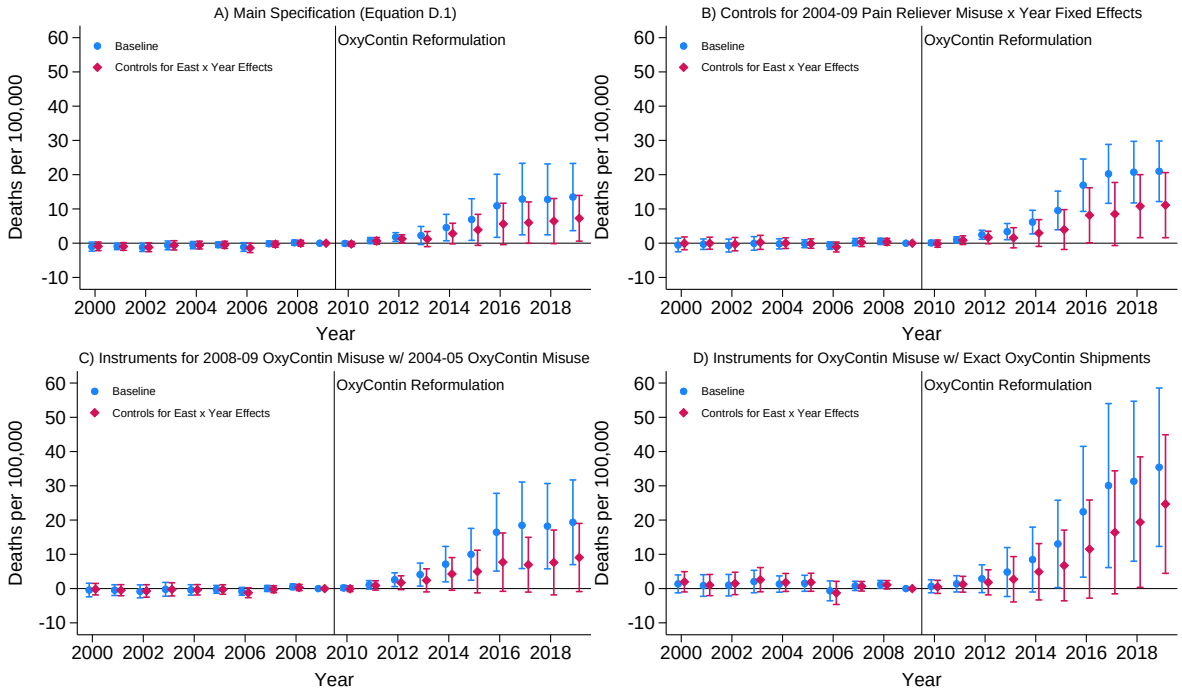
Notes: This figure extends the work of Evans et al. (2019) by examining trends in heroin overdose mortality separately for eastern vs. western states with above vs. below median OxyContin shipments from 2008-2009, before and after the OxyContin reformulation (mid-2010) and the potency shocks (2012) described in Section 2.

Figure A.17: Replication and Extension of the OxyContin Reformulation's Impacts on Fentanyl Mortality



Notes: This figure presents estimates of the impact of one percentage point higher state OxyContin misuse prevalence on synthetic opioid (i.e., fentanyl) overdose mortality rates each year relative to when OxyContin was reformulated to be abuse-deterrent (2010). Each panel presents estimates with and without controlling for an interaction between being an eastern state and year fixed effects. Panel A replicates and extends the baseline specification (Equation 1) in Alpert et al. (2018) (Equation 3 in this manuscript). Panel B adds controls for 2004-2009 pain reliever misuse prevalence interacted with year fixed effects, to adjust for the fact that pre-reformulation OxyContin misuse is non-random (Alpert et al. 2018; Beheshti 2019; Park 2025). Panel C uses 2008-2009 OxyContin misuse prevalence (rather than 2004-2009 OxyContin misuse) and instruments for it using 2004-2005 OxyContin misuse prevalence to address measurement error in OxyContin misuse prevalence (Alpert et al. 2018). Panel D instruments for 2004-2009 OxyContin misuse with 2006-2009 state OxyContin shipment rates (morphine equivalent grams per capita). See Section 4 of the text for more details.

Figure A.18: Replication and Extension of the OxyContin Reformulation's Impacts on Illicit Opioid Mortality



Notes: This figure presents estimates of the impact of one percentage point higher state OxyContin misuse prevalence on illicit opioid (i.e., heroin or fentanyl) overdose mortality rates each year relative to when OxyContin was reformulated to be abuse-deterrent (2010). Each panel presents estimates with and without controlling for an interaction between being an eastern state and year fixed effects. Panel A replicates and extends the baseline specification (Equation 1) in Alpert et al. (2018) (Equation 3 in this manuscript). Panel B adds controls for 2004-2009 pain reliever misuse prevalence interacted with year fixed effects, to adjust for the fact that pre-reformulation OxyContin misuse is non-random (Alpert et al. 2018; Beheshti 2019; Park 2025). Panel C uses 2008-2009 OxyContin misuse prevalence (rather than 2004-2009 OxyContin misuse) and instruments for it using 2004-2005 OxyContin misuse prevalence to address measurement error in OxyContin misuse prevalence (Alpert et al. 2018). Panel D instruments for 2004-2009 OxyContin misuse with 2006-2009 state OxyContin shipment rates (morphine equivalent grams per capita). See Section 4 of the text for more details.

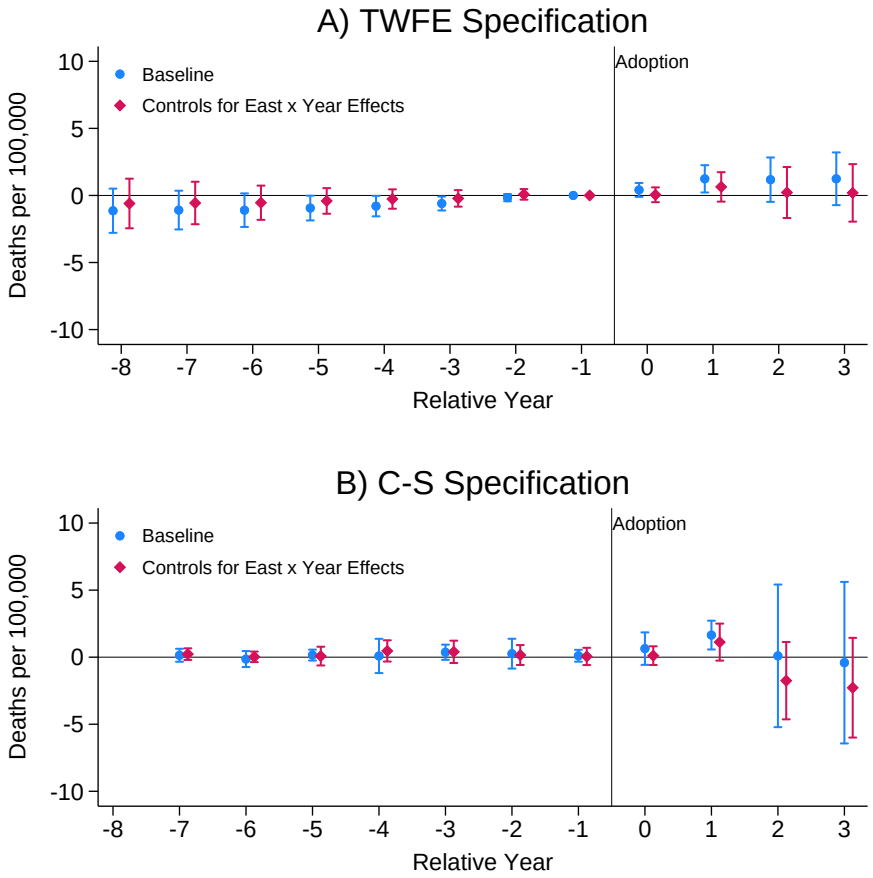


Figure A.19: Extension of the Impacts of Must-Access PDMPs on Heroin Mortality

Notes: The figure presents estimates of the event study coefficients for the impacts of must-access Prescription Drug Monitoring Programs (PDMPs) on heroin overdose mortality rates. Panel A uses a two-way fixed effects model and Panel B uses the estimator described in Callaway and Sant'Anna (2021). In both models, we control for exposure to OxyContin reformulation by interacting pre-reformulation OxyContin shipments with year fixed effects and present results with and without controlling for east interacted with year fixed effects to account for exposure to potency shocks.

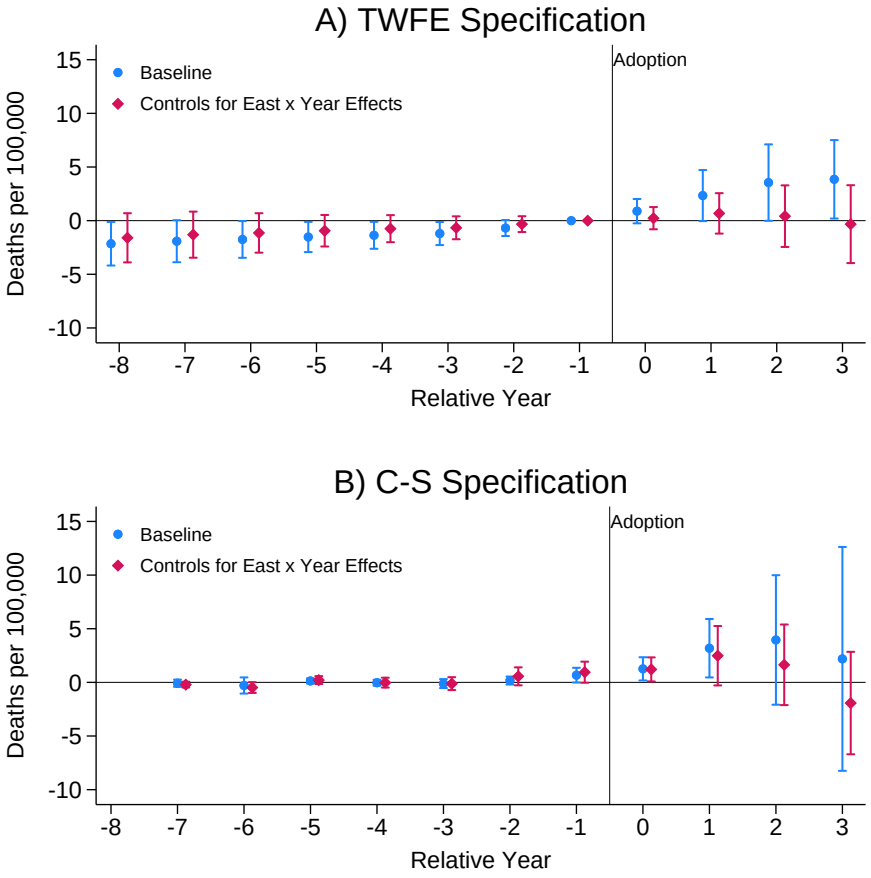


Figure A.20: Extension of the Impacts of Must-Access PDMPs on Fentanyl Mortality

Notes: The figure presents estimates of the event study coefficients for the impacts of must-access Prescription Drug Monitoring Programs (PDMPs) on synthetic opioid (i.e., fentanyl) overdose mortality rates. Panel A uses a two-way fixed effects model and Panel B uses the estimator described in Callaway and Sant'Anna (2021). In both models, we control for exposure to OxyContin reformulation by interacting pre-reformulation OxyContin shipments with year fixed effects and present results with and without controlling for east interacted with year fixed effects to account for exposure to potency shocks.

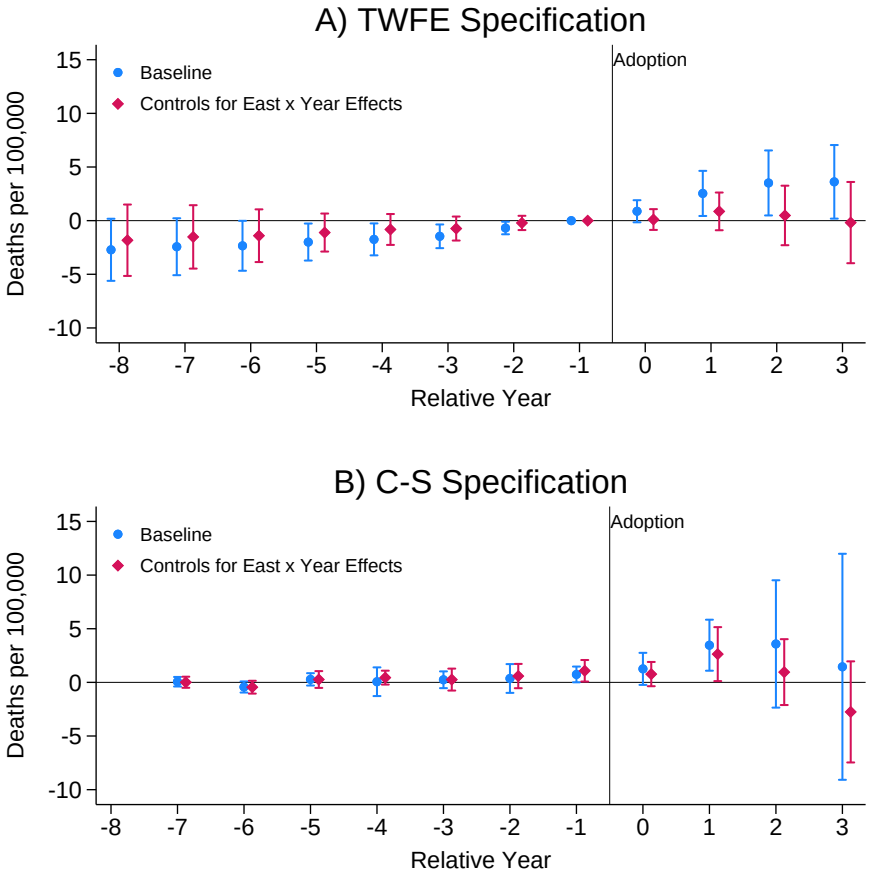


Figure A.21: Extension of the Impacts of Must-Access PDMPs on Illicit Opioid Mortality

Notes: The figure presents estimates of the event study coefficients for the impacts of must-access Prescription Drug Monitoring Programs (PDMPs) on illicit opioid (i.e., heroin or fentanyl) overdose mortality rates. Panel A uses a two-way fixed effects model and Panel B uses the estimator described in Callaway and Sant'Anna (2021). In both models, we control for exposure to OxyContin reformulation by interacting pre-reformulation OxyContin shipments with year fixed effects and present results with and without controlling for east interacted with year fixed effects to account for exposure to potency shocks.

B Additional Tables

Table B.1: Average Mortality Impacts of Potency Shocks (Continuous Treatment)

	Any Heroin (1)	Any Fentanyl (2)	Heroin or Fentanyl (3)	Non-Opioids (4)	Any Drug (5)
(Pre-Takeover White Powder Heroin Share)X(Post)	0.0380***	0.0842***	0.0905***	0.0019	0.0844***
	(0.0097)	(0.0200)	(0.0219)	(0.0094)	(0.0253)
Pre-treatment mean	1.31	0.600	1.89	4.85	11.2
R^2	0.766	0.637	0.727	0.790	0.779
Observations	540	540	540	540	540

Notes: This table presents the β coefficients from estimating Equation (2) using a continuous treatment measure of white powder heroin in the pre-potency shock period. Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes only commuting zones where we have HDMP data.

Table B.2: Average Mortality Impacts of Potency Shocks (Binned Treatment)

	Any Heroin (1)	Any Fentanyl (2)	Heroin or Fentanyl (3)	Non-Opioids (4)	Any Drug (5)
(Post) × WhitePowderQuartile = 2	-0.5110	0.7295	-0.0068	0.5438*	1.054
	(0.6930)	(0.9717)	(1.296)	(0.2918)	(1.276)
(Post) × WhitePowderQuartile = 3	3.094***	5.794***	6.379***	-0.4272	5.912***
	(0.9336)	(1.743)	(1.758)	(0.4096)	(1.749)
(Post) × WhitePowderQuartile = 4	2.227***	6.944***	6.659***	0.8866	7.103***
	(0.7706)	(1.825)	(1.894)	(1.074)	(2.471)
Pre-treatment mean	1.31	0.600	1.89	4.85	11.2
R^2	0.780	0.634	0.725	0.796	0.777
Observations	540	540	540	540	540

Notes: This table presents the β coefficients from estimating Equation (2) using a binned treatment measure of white powder heroin in the pre-potency shock period, where the reference category is the first quartile. Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes only commuting zones where we have HDMP data.

Table B.3: Average Mortality Impacts of Potency Shocks (Nationwide)

	Any Heroin (1)	Any Fentanyl (2)	Heroin or Fentanyl (3)	Non-Opioids (4)	Any Drug (5)
(Eastern Market)X(Post)	2.933***	6.340***	7.133***	-0.6466**	6.846***
	(0.2949)	(0.5151)	(0.5765)	(0.2569)	(0.6891)
Pre-treatment mean	0.388	0.942	1.32	5.22	10.7
R^2	0.698	0.615	0.697	0.671	0.742
Observations	13,740	13,740	13,740	13,740	13,740

Notes: This table presents the β coefficients from estimating Equation (2). The East binary treatment variable is equal to one for commuting zones east of -91 degrees longitude, and Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. The sample includes all commuting zones in the contiguous United States. Outcomes are overdose deaths per 100,000.

Table B.4: Average Mortality Impacts of Potency Shocks (State-level, Additional Controls)

	Any Heroin				log(heroin)		Any Fentanyl					log(fentanyl)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
(Eastern Market)X(Post)	2.714***	2.615***	3.006***	2.714***	2.770***	0.9887***	6.040***	5.693***	5.421***	6.054***	6.054***	1.132***
	(0.5155)	(0.5217)	(0.5428)	(0.5169)	(0.4938)	(0.2488)	(0.8465)	(0.8239)	(0.9003)	(0.8330)	(0.8226)	(0.1548)
(OxyContin Misuse 2004-2009)X(Post-Reformulation)		0.6241	0.9998					2.194**	1.932**			
		(0.6668)	(0.6029)					(1.041)	(0.8470)			
(Eastern Market)X(OxyContin Misuse 2004-2009)X(Post-Reformulation)			-0.5955						0.4155			
			(0.4058)						(0.6245)			
Yearly Law Enforcement Officers per 100,000				0.0002						0.0096		
				(0.0038)						(0.0130)		
Must-Access PDMP Laws					0.4027						0.1024	
					(0.3960)						(0.6472)	
(Post)X(Log(Import Trade Value per Capita in 2008))						-0.1977						0.1517
						(0.2044)						(0.1290)
Pre-treatment mean	0.795	0.795	0.795	0.795	0.795	0.795	0.801	0.801	0.801	0.801	0.801	0.801
R^2	0.779	0.781	0.782	0.779	0.781	0.798	0.706	0.711	0.711	0.707	0.706	0.868
Observations	980	980	980	980	980	902	980	980	980	980	980	969

Notes: This table presents the β coefficients in the first row from estimating Equation (2) with different control variables. The Eastern Market binary variable is equal to one for states with centroids east of -91 degrees longitude and Post is an indicator for 2012 and after. All models include state and year fixed effects. Standard errors are clustered at the state-level, and regressions are weighted by state population. The outcome is overdose deaths per 100,000, except for Columns 6 and 12 (logarithms of rates), where Columns 1-6 are for heroin and 7-12 are for fentanyl. The sample includes all contiguous U.S. states.

Table B.5: Average Heroin Mortality Impacts of Potency Shocks (HDMP, Additional Controls)

	Any Heroin							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
(White Powder Market)X(Post)	2.961***	2.946***	3.355***	2.871***	3.878***	2.889***	2.945***	2.866***
	(0.6712)	(0.6593)	(0.6487)	(0.7007)	(0.8402)	(0.8427)	(0.6625)	(0.6758)
(Above Median OxyContin 2008-09)X(Post-Reformulation)		0.7302	1.372*					
		(0.6364)	(0.7739)					
(White Powder Market)X(Post-Reformulation)X(Above Median OxyContin 2008-09)			-1.274					
			(1.078)					
(OxyContin 2008-09)X(Post-Reformulation)				1.238	1.808***			
				(0.9019)	(0.6216)			
(White Powder Market)X(Post-Reformulation)X(OxyContin 2008-09)					-1.406**			
					(0.6210)			
Post-Must Access PDMP Reform						-0.2154		
						(0.8126)		
Yearly Law Enforcement Officers per 100,000							-0.0017	
							(0.0035)	
(Post)X(log(Import Trade Value per Capita in 2008))								-0.5540
								(0.7139)
Pre-treatment mean	1.31	1.31	1.31	1.31	1.31	1.31	1.31	1.31
R^2	0.776	0.780	0.785	0.788	0.796	0.776	0.776	0.778
Observations	540	540	540	540	540	540	540	540

Notes: This table presents the β coefficients in the first row from estimating Equation (2) with different control variables. The White Powder Heroin treatment indicator variable is defined as having an above median share of total pre-potency shock heroin purchases being white powder, and Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes only commuting zones where we have HDMP data.

Table B.6: Average Fentanyl Mortality Impacts of Potency Shocks (HDMP, Additional Controls)

	Any Fentanyl							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
(White Powder Market)X(Post)	5.939***	5.868***	4.937***	5.659***	5.390***	5.420***	6.079***	5.568***
	(1.374)	(1.084)	(0.7323)	(1.110)	(1.192)	(1.405)	(1.370)	(1.311)
(Above Median OxyContin 2008-09)X(Post-Reformulation)		3.479***	2.014**					
		(1.034)	(0.9580)					
(White Powder Market)X(Post-Reformulation)X(Above Median OxyContin 2008-09)			2.907*					
			(1.619)					
(OxyContin 2008-09)X(Post-Reformulation)				3.859***	3.707***			
				(0.7734)	(0.5161)			
(White Powder Market)X(Post-Reformulation)X(OxyContin 2008-09)					0.3744			
					(1.032)			
Post-Must Access PDMP Reform						-1.544		
						(2.665)		
Yearly Law Enforcement Officers per 100,000							0.0145	
							(0.0091)	
(Post)X(log(Import Trade Value per Capita in 2008))								-2.169
								(1.360)
Pre-treatment mean	0.600	0.600	0.600	0.600	0.600	0.600	0.600	0.600
R^2	0.632	0.657	0.662	0.661	0.661	0.635	0.636	0.640
Observations	540	540	540	540	540	540	540	540

Notes: This table presents the β coefficients in the first row from estimating Equation (2) with different control variables. The White Powder Heroin treatment indicator variable is defined as having an above median share of total pre-potency shock heroin purchases being white powder, and Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes only commuting zones where we have HDMP data.

Table B.7: Average Heroin Mortality Impacts of Potency Shocks (Nationwide, Additional Controls)

	Any Heroin							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
(Eastern Market)X(Post)	2.933***	2.848***	2.873***	2.782***	2.781***	2.991***	2.933***	2.923***
	(0.2949)	(0.3040)	(0.3455)	(0.3240)	(0.3687)	(0.3005)	(0.2949)	(0.2983)
(Above Median OxyContin OxyContin 2008-09)X(Post-Reformulation)		0.4853	0.5208**					
		(0.3092)	(0.2323)					
(Eastern Market)X(Post-Reformulation)X(Above Median OxyContin OxyContin 2008-09)			-0.0573					
			(0.3903)					
(OxyContin OxyContin 2008-09)X(Post-Reformulation)				0.6402*	0.6396**			
				(0.3628)	(0.2901)			
(Eastern Market)X(Post-Reformulation)X(OxyContin OxyContin 2008-09)					0.0009			
					(0.2223)			
Post-Must Access PDMP Reform						0.4956**		
						(0.2216)		
Yearly Law Enforcement Officers per 100,000							-4.27×10^{-5}	
							(0.0008)	
(Post)X(log(Import Trade Value per Capita in 2008))								0.2234
								(0.2855)
Pre-treatment mean	0.388	0.388	0.388	0.388	0.388	0.388	0.388	0.388
R^2	0.698	0.700	0.700	0.701	0.701	0.700	0.698	0.698
Observations	13,740	13,740	13,740	13,740	13,740	13,740	13,740	13,740

Notes: This table presents the β coefficients in the first row from estimating Equation (2) with different control variables. The Eastern Market binary variable is equal to one for commuting zones east of -91 degrees longitude and Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are heroin overdose deaths per 100,000 and the sample includes all commuting zones in the contiguous United States.

Table B.8: Average Fentanyl Mortality Impacts of Potency Shocks (Nationwide, Additional Controls)

	Any Fentanyl							
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
(Eastern Market)X(Post)	6.340***	5.979***	5.150***	5.820***	5.328***	6.368***	6.342***	6.339***
	(0.5151)	(0.4424)	(0.3522)	(0.4700)	(0.4893)	(0.5133)	(0.5137)	(0.5188)
(Above Median OxyContin OxyContin 2008-09)X(Post-Reformulation)		2.060***	0.8770***					
		(0.3676)	(0.2294)					
(Eastern Market)X(Post-Reformulation)X(Above Median OxyContin OxyContin 2008-09)			1.909***					
			(0.3892)					
oxycontin_0809 × postreformulation				38.16***	29.25***			
				(5.579)	(4.524)			
long91 × oxycontin_0809 × postreformulation					11.34***			
					(3.481)			
Post-Must Access PDMP Reform						0.2322		
						(0.4659)		
Yearly Law Enforcement Officers per 100,000							0.0013	
							(0.0026)	
(Post)X(log(Import Trade Value per Capita in 2008))								0.0262
								(0.4112)
Pre-treatment mean	0.942	0.942	0.942	0.942	0.942	0.942	0.942	0.942
R^2	0.615	0.623	0.626	0.625	0.625	0.615	0.615	0.615
Observations	13,740	13,740	13,740	13,740	13,740	13,740	13,740	13,740

Notes: This table presents the β coefficients in the first row from estimating Equation (2) with different control variables. The Eastern Market binary variable is equal to one for commuting zones east of -91 degrees longitude and Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are heroin overdose deaths per 100,000 and the sample includes all commuting zones in the contiguous United States.

Table B.9: Average Mortality Impacts of potency shocks by Pre-Existing Heroin Use

Panel A: Pre-Potency Shocks (2005 to 2011)

	Any Heroin (1)	Any Fentanyl (2)	Heroin or Fentanyl (3)	Non-Opioids (4)	Any Drug (5)
(Eastern Market)X(Post)	1.760***	3.470***	4.157***	-1.000***	3.396***
	(0.3550)	(0.5851)	(0.6680)	(0.3364)	(0.7962)
(2005-2011 Heroin Death Rate)X(Post)	0.0551**	0.0140	0.0682**	-0.0481**	-0.0129
	(0.0242)	(0.0158)	(0.0344)	(0.0215)	(0.0381)
(2005-2011 Heroin Death Rate)X(Eastern Market)X(Post)	0.1573***	0.3985***	0.4073***	0.0546	0.4824***
	(0.0496)	(0.0822)	(0.0998)	(0.0349)	(0.0955)
Pre-treatment mean	0.388	0.942	1.32	5.22	10.7
R^2	0.740	0.655	0.740	0.672	0.767
Observations	13,740	13,740	13,740	13,740	13,740

Panel B: Pre-OxyContin Reformulation (2005 to 2009)

	Any Heroin (1)	Any Fentanyl (2)	Heroin or Fentanyl (3)	Non-Opioids (4)	Any Drug (5)
(Eastern Market)X(Post)	1.892***	3.637***	4.416***	-0.9578***	3.804***
	(0.3352)	(0.5680)	(0.6326)	(0.3409)	(0.7618)
(2005-2009 Heroin Death Rate)X(Post)	0.0660**	0.0117	0.0777*	-0.0660**	-0.0304
	(0.0320)	(0.0213)	(0.0452)	(0.0307)	(0.0516)
(2005-2009 Heroin Death Rate)X(Eastern Market)X(Post)	0.2277***	0.6006***	0.6001***	0.0731	0.6785***
	(0.0634)	(0.1134)	(0.1321)	(0.0562)	(0.1276)
Pre-treatment mean	0.388	0.942	1.32	5.22	10.7
R^2	0.730	0.650	0.732	0.672	0.761
Observations	13,740	13,740	13,740	13,740	13,740

Notes: This table presents the coefficients from estimating Equation (2) with additional interaction terms. The Eastern Market binary treatment variable is equal to one for commuting zones east of -91 degrees longitude and Post is an indicator for 2012 and after. In Panel A, we use Heroin OD$c, 2005-2011$, the heroin overdose death rate from 2005 to 2011, while in Panel B, we use Heroin OD$c, 2005-2009$, the heroin overdose death rate from 2005 to 2009. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes all commuting zones in the contiguous United States.

Table B.10: Average Mortality Impacts of Potency Shocks by Race/Ethnicity

Panel A: HDMP Analysis

	Heroin or Fentanyl	White	Black	Hispanic	Native	Other
	(1)	(2)	(3)	(4)	(5)	(6)
(White Powder Market)X(Post)	6.527***	8.113***	5.132	5.705***	-3.515	0.4581***
	(1.448)	(1.579)	(3.545)	(1.628)	(5.446)	(0.1616)
Pre-treatment mean	1.89	2.61	1.67	0.953	1.71	0.115
R^2	0.725	0.799	0.660	0.623	0.510	0.616
Observations	540	540	540	540	540	540

Panel B: Nationwide Analysis

	Heroin or Fentanyl	White	Black	Hispanic	Native	Other
	(1)	(2)	(3)	(4)	(5)	(6)
(Eastern Market)X(Post)	7.132***	7.960***	6.327***	5.793***	7.030***	0.4927***
	(0.5777)	(0.6907)	(1.166)	(0.8912)	(2.311)	(0.0954)
Pre-treatment mean	1.35	1.61	0.878	0.575	1.78	0.046
R^2	0.697	0.715	0.594	0.572	0.265	0.281
Observations	12,009	12,009	12,009	12,009	12,009	12,006

Notes: This table presents the β coefficients from estimating Equation (2) for overdose deaths for each race category. In Panel A, the White Powder Heroin treatment indicator variable is defined as having an above median share of total pre-potency shock heroin purchases being white powder and in Panel B, the Eastern Market treatment variable is equal to one for commuting zones east of -91 degrees longitude. The sample in Panel A is only commuting zones that had a city that participated in the Heroin Domestic Monitor Program (HDMP) and in Panel B, it is all commuting zones in the contiguous United States. Post is an indicator for 2012 and after. The models include commuting zone and year fixed effects, standard errors are clustered at the commuting zone, and the regressions are population weighted. Outcomes are overdose deaths per 100,000 and the sample includes only commuting zones where have population counts for all races.

Table B.11: Decomposition of Changes in Average Annual Opioid Death Rates (Nationwide), 2009 to 2019

	Heroin	Fentanyl	Illicit Opioids
	(1)	(2)	(3)
Heroin Potency Shocks	1.09	5.68	5.00
	[0.23, 1.94]	[3.42, 7.93]	[2.91, 7.08]
OxyContin reformulation	-0.02	3.27	3.91
	[-1.74, 1.70]	[-1.01, 7.54]	[-0.05, 7.87]
Must-access PDMPs	1.07	1.79	2.02
	[0.13, 2.02]	[-0.79, 4.36]	[-0.50, 4.55]
Average Increase (rel. 2009)	3.27	10.32	10.87

Notes: Decomposition of changes in heroin, synthetic opioid (fentanyl), and illicit opioid (heroin or fentanyl) from 2009 to 2019 based on interacting 2019 coefficients from Equation (D.2) with weights proportional to the impact of each intervention/shock in the average state. 95% confidence intervals are presented in brackets and use standard errors that were constructed via the Delta method. The bottom row presents the average annual change in each cause of death from 2009 to 2019, for comparison.

C Data Appendix

In this Appendix, we provide additional details on our data sources.

C.1 Heroin Domestic Monitoring Program

To analyze retail heroin supply in U.S. cities, we draw on the DEA's Heroin Domestic Monitoring Program (HDMP), which operated until 2016. The HDMP was designed as a surveillance program rather than an enforcement tool: DEA agents conducted undercover purchases of heroin at the retail level, which were then sent to DEA laboratories for chemical analysis. Unlike seizure data, which reflect wholesale or upstream flows, HDMP samples capture the heroin actually consumed by users. This distinction is crucial, as heroin is typically diluted or adulterated as it moves down the supply chain; seizure data alone cannot reliably measure the formulations, adulterants, or potency available to end-users. Roughly 900 samples were collected each year, with most cities contributing 15 to 40 depending on local conditions and field office capacity. These samples were not randomly drawn but instead reflected operational intelligence and agent access, making the data best understood as informed surveillance rather than a statistically representative sample. Nevertheless, HDMP remains the most systematic and granular source on retail heroin supply available at a national scale.

We restrict attention to a consistent subset of 27 cities covered by the program between 2004 to 2016, excluding smaller locations and earlier "geo-probe" efforts that were limited, irregular, and often lacked systematic detail on heroin type or purity. Earlier HDMP reports from the early 1990s are also sparse, omit several major cities included in later years, and are inconsistent in coverage, so we focus on 2004 to 2016 to ensure comparability. Some later reports back-filled data for prior years when prior reports were not released (e.g., the 2006 report includes 2004–2005), which allows us to construct a balanced panel for 2004–2006 and 2009–2016. The gap in 2007–2008 reflects years in which no HDMP reports were released. For each sample, DEA labs measured heroin purity, identified formulation (black tar, white powder, or brown powder), tested for adulterants, and used chemical profiling to assign a source country. As new substances emerged, such as fentanyl analogs, laboratory protocols were updated accordingly. A key limitation of these data is that they are only available at an aggregate city-year level, which likely obscures important within-city variation in heroin purity. Our estimates of variability of heroin purity in Figure A.3 Panel B thus understates true variability of heroin purity, as we are already averaging over the variability of purity within cities by using aggregate city-level data. Despite these limitations, the HDMP provides unique and otherwise unavailable evidence on the heroin supply actually consumed by users over the critical period of the onset of the heroin and fentanyl waves of the U.S. opioid epidemic in the early 2010s.

In Appendix Table C.1, we list the 27 cities included in our analysis, reporting the average number of heroin samples and heroin characteristics for each city. To our knowledge, the HDMP program was discontinued after 2016 due to shifting DEA priorities at the start of the first Trump administration and a broader strategic pivot toward synthetic opioids. According to a private conversation with a former DEA official, the termination also reflected political concerns about the optics of transferring federal funds to retail heroin sellers through undercover purchases.

Table C.1: Summary of City-Level Data from HDMP, 2004 to 2016

HDMP City	Average Purchases	Average Purity (%)	% White Powder	% Mexico (pre-2012)	% Colombia (pre-2012)
Albuquerque	16.9	17.4	0.0	100.0	0.0
Atlanta	28.5	36.7	53.8	5.0	95.0
Baltimore	33.1	18.4	46.4	4.0	96.0
Boston	31.9	17.4	68.9	1.2	98.8
Chicago	32.7	15.4	59.1	4.9	95.1
Dallas	36.2	16.6	4.8	100.0	0.0
Denver	36.2	31.0	0.0	100.0	0.0
Detroit	35.0	35.0	50.9	0.8	99.2
El Paso	14.2	42.6	0.0	100.0	0.0
Houston	27.5	22.6	25.0	92.6	7.4
Los Angeles	36.2	29.9	10.6	99.5	0.5
Miami	23.6	19.5	60.6	1.5	98.5
Minneapolis	3.5	52.8	0.0	100.0	0.0
New Orleans	30.1	24.0	53.6	4.3	95.7
New York	96.3	39.9	53.0	0.4	99.6
Orlando	22.6	29.0	72.0	1.2	98.8
Philadelphia	34.9	55.2	63.6	0.6	99.4
Phoenix	34.5	37.5	11.2	99.5	0.5
Pittsburgh	12.7	41.5	55.6	3.0	97.0
Portland	15.2	28.8	0.0	100.0	0.0
Richmond	16.2	23.9	53.9	6.1	93.9
San Antonio	18.3	9.9	4.8	97.0	3.0
San Diego	34.7	42.0	9.1	99.5	0.5
San Francisco	33.2	9.8	0.0	100.0	0.0
Seattle	30.3	16.0	0.0	100.0	0.0
St. Louis	28.2	31.5	48.3	51.0	49.0
Washington	27.0	21.7	48.5	5.9	94.1

C.2 Vital Records

Data on overdose mortality comes from restricted all-county cause-of-death data from the National Center for Health Statistics (NCHS) National Vital Statistics System (NVSS). We follow coding conventions used in CDC (2021) identify overdose deaths by cause, including prescription opioids, heroin, synthetic opioids, any opioids, deaths where a specific drug was not mentioned, and any drug. In particular, drug overdose deaths were identified based on the International Classification of Diseases (ICD), 10th edition underlying cause-of-death codes X40–X44, X60–X64, X85, and Y10–Y14. Cause-specific opioid overdose deaths were identified using multiple-cause-of-death codes T40.1 (heroin), T40.2 (prescription opioids), and T40.4 (synthetic opioids other than methadone, i.e., mostly fentanyl). Each of these codes as well as T40.3 (methadone) and T40.6 (unspecified narcotics) were used to define total opioid overdose deaths. We construct overdose death rates by dividing counts of overdose deaths by commuting zone or state population, drawn from the U.S. Census Bureau's County Intercensal Dataset and County Population Totals.

C.3 National Survey on Drug Use and Health

We use data from the National Survey on Drug Use and Health (NSDUH) to examine trends in prior-year heroin use over time, which we compare to counts of heroin overdose deaths in Section 3.4. The NSDUH provides the only consistent national data on heroin use over time. Annual data on heroin use are not publicly available at the state, county, or city levels. Thus,

we can only use it to provide national estimates and cannot present estimates solely for white powder heroin markets (i.e., eastern cities or states). One additional concern with the NSDUH is that it undercounts heroin use due to under-reporting and omits high-risk populations (e.g., individuals experiencing homelessness) (Reuter et al. 2021). However, assuming this undercounting is time-invariant—an assumption that we support by examining pre-period trends in Figure 3—the data still allow us to assess how the risk of overdose among heroin users evolved following the potency shocks that began in 2012.

Using these data, trends in prior-year heroin use (alongside heroin overdose deaths) are presented in Appendix Figure C.1. As shown, heroin use grew at roughly a constant rate from 2002 until 2017, before decreasing in 2018 and 2019. Thus, the NSDUH picked up rising heroin use over time. The sharp trend break in the probability of death conditional on use reported in Figure 3 comes from death rates growing much more quickly than use beginning in 2012.

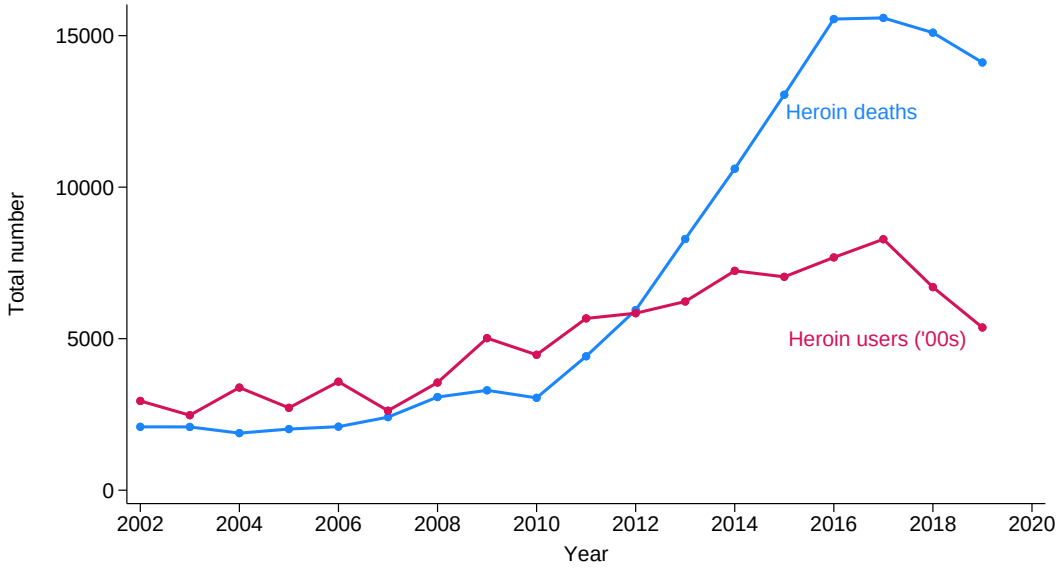


Figure C.1: Yearly Trends in Heroin Use (NSDUH) and Overdose Mortality (CDC)

Notes: The National Survey on Drug Use and Health (NSDUH) data capture self-reported heroin use, while mortality trends reflect overdose death records from the CDC.

We also use the NSDUH to measure state OxyContin misuse prevalence, as in Alpert et al. (2015), in Sections 4 and 4.2. Data are reported as a 5-year average from 2004-2009 and, in the instrumental variables analysis to account for measurement error, in 2-year averages from 2004-2005 and 2008-2009. These data were obtained directly from the replication package published alongside Alpert et al. (2015).

C.4 Additional Data Sources

To measure OxyContin distribution below the state level, we use the DEA's Automation of Reports and Consolidated Orders System (ARCOS). ARCOS records every controlled substance shipment in the United States from 2006 to 2019. Each transaction includes the reporter and buyer's DEA number, address, name, and business type, along with details such as the shipment

date, opioid drug name, quantity, strength, and Morphine Milligram Equivalent (MME) conversion factor. These data allow us to construct measures of OxyContin shipments at any temporal or geographic level. Our primary measure is average morphine equivalent grams (MEGs) per capita OxyContin shipments at the commuting zone level during 2006–2009, just before OxyContin was reformulated to be abuse-deterrent. Data on state-level must-access Prescription Drug Monitoring Program (PDMP) implementation dates came from Horwitz et al. (2021).

We use data from the Federal Bureau of Investigation's LEOKA program to measure local law enforcement intensity. Specifically, we construct annual counts of sworn law enforcement officers per 100,000 residents at the commuting zone (CZ) level. This provides our best nationwide proxy for enforcement capacity and allows us to assess whether changes in policing might confound the estimated effects of heroin potency shocks. Because LEOKA reports at finer spatial scales, we aggregate the data to CZs and align them with our main analytic sample. These measures are incorporated as time-varying controls in robustness specifications (see Appendix Figures and Tables referenced in the main text).

To account for the possibility that fentanyl spread was linked to illicit smuggling through legal trade channels, we use state-level import data from the U.S. Census Bureau's Trade Online portal, following the approach of Moore et al. (2023). We focus on 2008 import volumes per capita, using the "state of destination" variable to assign flows geographically. We also exclude oil and gas imports. Because the data are available only at the state level, we assign state import volumes to all CZs within a state. For multi-state CZs, we compute population-weighted averages. This procedure introduces potential measurement error, which could attenuate estimates, but provides the best feasible proxy for fentanyl smuggling exposure via state imports. As an additional check, we re-estimate key robustness models at the state level, which yields highly consistent results with our CZ-level analysis.

D Methods Appendix

D.1 Revisiting the Effects of OxyContin Reformulation

To examine the effects of the OxyContin reformulation on heroin and fentanyl overdose mortality, we replicate and extend the work of Alpert et al. (2018) and Powell and Pacula (2021). Aggregating to the state level, we estimate

$$Y_{st} = \alpha_s + \gamma_t + \delta_t \, \text{OxyMisuse}_s^{\text{Pre}} + \beta_t \, \text{WhitePowderMarket}_s + \epsilon_{st}, \tag{D.1}$$

where the first four terms are the same as Alpert et al. (2018). Y_{st} are yearly overdose death rates by state, and we examine deaths due to heroin, fentanyl, and any illicit opioid (heroin or fentanyl). α_s and γ_t are state and year fixed effects. $\delta_t \text{OxyMisuse}_s^{Pre}$ interacts year fixed effects with each state's fixed pre-reformulation rate of OxyContin misuse, drawn from the 2004-2009 NSDUH. We normalize the 2009 coefficient (just before reformulation) to zero. Thus, the δ_t coefficients identify changes in illicit opioid mortality rates after the reformulation for each one percentage point higher pre-reformulation OxyContin misuse prevalence. We then add $\beta_t \text{White_Powder_Market}_s$ to control for exposure to potency shocks: it is an indicator for being east of -91 degrees longitude, measured based on each state's centroid, interacted with year fixed effects. We weight regressions by state population and cluster standard errors at the state-level.

D.2 Revisiting the Effects of Must-Access PDMPs

Our analysis of the effects of must-access Prescription Drug Monitoring Programs (PDMPs) largely follows Kim (2021), with a few key differences. First, we use annual rather than quarterly data and incorporate more recent years of data on overdose deaths. We also do not adjust for under-reporting of opioid-related deaths using the Ruhm (2018) method to maintain consistency with the reporting on overdose deaths that we use throughout the paper. We also do not include state-level time-varying controls beyond exposure to the OxyContin reformulation and heroin potency shocks. Otherwise, key aspects of Kim (2021)'s approach are retained.

We estimate a two-way fixed effects model with indicators for each year relative to PDMP adoption, both with and without controlling for the interaction between east and year fixed effects. Exposure to OxyContin reformulation is controlled for by interacting 2006–2009 OxyContin shipment rates with year fixed effects, as in Kim (2021). Due to staggered adoption timing, some states lack observations in distant relative future periods. Following Kim (2021) and leveraging our extended data, we construct a balanced sample of 17 treated states with a -8 to +3 year window relative to adoption and 15 control states without PDMP adoption by 2019. Indicators outside this window are trimmed. We also test robustness using the Callaway and Sant'Anna (2021) estimator, which is robust to heterogeneous treatment effects under staggered adoption, and is important to account for due to must-access PDMPs being rolled out at different points in time across states. Regressions are weighted by state population, with standard errors clustered at the state level.

D.3 Decomposition of Illicit Opioid Mortality Trends

To decompose the overall determinants of rising illicit opioid mortality rates over time, we estimate

$$Y_{st} = \alpha_s + \gamma_t + \delta_t \text{OxyMisuse}_s^{Pre} + \beta_t \text{White_Powder_Market}_s + \phi_r \text{PDMP}_s + \epsilon_{st}, \tag{D.2}$$

which includes interactions between each policy/shock and relative year dummies during the post-period, while omitting interactions with pre-period dummies. Pre-trends are parallel and null for each outcome, as shown in prior sections, so results are robust to this choice. Omitting pre-period indicators mainly helps clarify dynamics for early shocks that preceded others—e.g., the OxyContin reformulation occurred before must-access PDMPs or potency shocks and thus should explain all divergence in mortality trends prior to those later shocks. We use this specification to examine dynamic effects and assess how the effects of exposure to each policy/shock evolves over time. The analysis uses data from all states, weights observations by state population, and clusters standard errors at the state level.

An important methodological point is that this decomposition includes any dynamic spillovers (e.g., individuals initially exposed to fentanyl-adulterated heroin later independently demanding fentanyl as a standalone product) or spatial spillovers (e.g., fentanyl use spreading through social networks) that resulted from the introduction of fentanyl into white powder heroin markets (Cutler and Donahoe 2024). The decomposition also does not consider the effects of the earlier, long-standing prescription opioid epidemic, which expanded the population vulnerable to heroin potency shocks by increasing opioid addiction (Eichmeyer and Zhang 2021). Consistently rising heroin use can be observed, for example, from 2002 to 2012 in Appendix Figure C.1. If fewer people had initiated heroin use over this time after becoming addicted to prescription opioids and transitioning, there would have been fewer people susceptible to the introduction of fentanyl into white powder heroin supply. Our focus in this exercise is on what caused the changes in illicit opioid mortality rates over the period from 2010 to 2019, rather than what illicit opioid mortality would have been from 2010 to 2019 if deeper historical drivers of the opioid epidemic (e.g., Purdue Pharmaceutical's introduction of OxyContin) had never occurred. For analyses of these historical drivers of the opioid epidemic and their role in U.S. drug overdose mortality trends, readers should refer to Cutler and Glaeser (2021), Alpert et al. (2022), Arteaga and Barone (2025), and Evans and Lieber (2025).