

Sinaloa Cartel Presence and Its Impact on Fentanyl Mortality in the United States

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Abstract

The United States opioid epidemic has killed more than one million Americans since 1999. Its deadliest phase was driven by fentanyl, a synthetic opioid characterized by low production costs and extreme potency. Annual fentanyl deaths rose from roughly 3,000 in 2013 to over 70,000 by 2022, a more than twentyfold increase now accounting for the majority of U.S. overdose deaths. Despite its severity, the drivers behind the fentanyl wave remain understudied. It is documented that most of the illicit fentanyl entering the United States came through the southern border, trafficked by Mexican drug trafficking organizations. Among them, Sinaloa was uniquely positioned to produce and distribute fentanyl at scale, leveraging an established criminal infrastructure built for heroin and methamphetamine. In this paper, I show that the Sinaloa Cartel, which held an early advantage in the production and distribution of fentanyl, played a key role in shaping the impact of the fentanyl crisis. Using DEA intelligence on cartel control, I classify U.S. counties by Sinaloa dominance and compare them to counties dominated by rival cartels in a difference-in-differences design to estimate the effect on overdose mortality. After 2014, fentanyl mortality in Sinaloa-dominated counties increased by 5.5 deaths per 100,000 relative to rival-dominated counties. A substantial share of these additional deaths is explained by polydrug mortality with heroin and cocaine. The health burden is concentrated among non-college-educated populations and, unlike the first two opioid waves, which disproportionately affected non-Hispanic White populations, this wave's mortality burden rises across all racial and ethnic groups, with the largest increase among Hispanic residents.

1. Introduction

The United States opioid epidemic has claimed over one million lives since 1999, making it the deadliest drug crisis in the country's history. The epidemic has unfolded in three waves. The first wave was driven by the aggressive marketing and prescribing of opioid painkillers in the late 1990s. The second wave was driven by a shift toward heroin, as the 2010 reformulation of OxyContin pushed dependent users toward illicit alternatives. The third wave differs fundamentally from its predecessors. Beginning around 2013, it was driven by a major supply shock: the mass introduction of illicitly manufactured fentanyl, a synthetic opioid. Fentanyl was involved in 75.1% of U.S. overdose deaths in 2023 (Centers for Disease Control and Prevention, 2026), yet little is known about the factors determining this wave.¹

Annual fentanyl overdose deaths grew from roughly 3,000 in 2013 to over 70,000 by 2022, a much steeper climb than in the prescription or heroin waves. The third wave stands apart in its lethality. Fentanyl is roughly 50 to 100 times more potent than heroin, abruptly increasing individual exposure risk. It also frequently reached users without their knowledge, mixed into heroin or counterfeit pills, leaving them no way to gauge potency. Its low production cost, high potency, and ease of concealment made fentanyl more profitable to traffic than heroin, so suppliers increasingly turned to it, flooding the market and changing what users were exposed to.²

By 2013, Mexican drug trafficking organizations (DTOs) supplied most of the heroin entering the United States, yet their capacity to produce fentanyl varied sharply. The Sinaloa Cartel held a clear advantage over its rivals: it had long experience producing synthetic drugs and a wholesale distribution network already operating across U.S. markets. When fentanyl arrived at scale in 2014, Sinaloa was positioned to introduce it earlier and more widely than any competing organization.

In this paper, I provide causal evidence that the Sinaloa Cartel played a key role in the third wave of the opioid epidemic: its pre-existing U.S. distribution network determined which places were most affected by fentanyl poisoning.

To measure Sinaloa's footprint before fentanyl, I use the 2015 National Drug Threat Assessment (NDTA), in which U.S. Drug Enforcement Administration (DEA)

¹I use "fentanyl" throughout to refer to the illicitly manufactured drug, as distinct from its legal, prescription form.

²One major difference in the transition to the third opioid wave, compared to the second, is that the shift here was from one illicit drug to another, more potent one, rather than from a prescription drug to an illicit one.

field offices report the share of drug cases linked to each Mexican trafficking organization. I classify a county as Sinaloa-dominated when Sinaloa accounts for the majority of cases in its field office, and counties where a rival organization dominates form the comparison group.³ I then compare overdose mortality across the two groups before and after fentanyl's arrival in 2014, using an event-study difference-in-differences design with county and year fixed effects. Because both groups are established trafficking hubs, the design isolates the identity of the incumbent organization while holding baseline drug-market infrastructure constant.

After 2014, fentanyl mortality rose sharply in Sinaloa-dominated counties relative to the comparison group, and the gap widened over time, consistent with the dynamic spillovers documented by Cutler and Donahoe (2024). On average, Sinaloa dominance adds 5.5 fentanyl deaths per 100,000 residents aged 15 and older, which accounts for 29.7% of the rise in fentanyl mortality in my sample between 2014 and 2020.

The effect is specific to fentanyl. I find no corresponding increase in non-fentanyl opioid mortality, nor in mortality from cocaine or methamphetamine in the absence of fentanyl. Thus, the shock operates strictly through fentanyl supply channels rather than a generalized expansion of all illicit drug markets.

At the same time, because polydrug use is a defining feature of the opioid epidemic's third wave, mortality involving fentanyl combined with other substances (such as cocaine, methamphetamine, or heroin) increases substantially. Crucially, Sinaloa presence raises fentanyl-only mortality and fentanyl-polydrug mortality by similar magnitudes—2.41 and 3.11 additional deaths per 100,000, respectively—demonstrating that the cartel's impact operated through both channels similarly.

The mortality burden is unevenly distributed along three margins. First, it follows the educational gradient documented for the opioid epidemic (Case & Deaton, 2022; Foote et al., 2025). Among residents without a college degree, Sinaloa presence raises fentanyl mortality by 15.1 deaths per 100,000, roughly ten times the 1.5 estimated for the college-educated. Second, its racial and ethnic profile departs from the first two waves, which fell disproportionately on non-Hispanic Whites (Case & Deaton, 2015). In the fentanyl wave, mortality rises across all racial and ethnic groups, with the largest increase among Hispanic residents. This pattern

³In order to avoid spatial spillovers, I drop rival counties that neighbor a Sinaloa-dominated county and vice versa. Section 3 describes the construction and its validation against an independent, newspaper-based measure of cartel presence.

follows from the third wave's illicit-to-illicit transition: exposure no longer ran through access to a prescription, historically concentrated among non-Hispanic Whites, and Sinaloa's fentanyl-adulterated heroin reached buyers across the existing heroin market regardless of race. Third, the effect is concentrated in the working-age population (ages 15–54), with no detectable effect among residents aged 55 and older.

Beyond mortality, I find no effect on local violent or property crime, in contrast to earlier drug-market transitions (Evans et al., 2016; Grogger & Willis, 2000), consistent with fentanyl spreading through pre-existing distribution networks without territorial conflict. County labor-market aggregates and disability caseloads also show no deterioration. Supplemental Security Income receipt among residents aged 65 and older does rise in treated counties, consistent with economic strain on older households that lose support from prime-age family members.

These results hold up when I control for leading alternative explanations from the literature: prescription drug monitoring programs (PDMPs), local exposure to international trade shocks, and exposure to the earlier prescription-opioid and heroin waves. Separately, permutation tests confirm that the estimated effect is not an artifact of understated standard errors: it exceeds the 99th percentile of a placebo distribution built by randomly reassigning Sinaloa presence across counties. I further show that the estimates are robust to correcting the mortality counts for incomplete drug specification on death certificates, following the regression-based procedure of Drake and Ruhm (2023).

This paper fills a key gap in the literature by providing an additional supply-side explanation for the third wave of the opioid epidemic. Economists have identified clear supply-side origins for the first two waves: Alpert et al. (2022a) show that geographic variation in exposure to OxyContin's introduction shaped the first wave, and Alpert et al. (2018) and Evans et al. (2019) show that its 2010 reformulation displaced dependent users toward heroin, triggering the second. The third wave has lacked a comparable explanation.

A growing literature is working to fill that gap. Moore et al. (2023) use state-level variation in legal trade flows and find that counties with greater import exposure saw larger increases in fentanyl mortality, evidence that fentanyl entered the country through ordinary commercial channels. Donahoe and Soliman (2025) use the 2012 takeover of white-powder heroin production by Mexican DTOs, displacing Colombian suppliers, and find that markets exposed to this takeover saw sharp increases in heroin and fentanyl mortality as heroin quality degraded. Mulligan

(2024) treats the fall in illicit-opioid prices as exogenous; Sinaloa's network provides geographic variation in that supply shock. I complement these papers by isolating the effect of a particular criminal actor, rather than of pre-existing epidemiological conditions, and by providing a more granular, county-level analysis.

The paper also speaks to two further literatures. First, it connects to work on the consequences of the opioid crisis. One strand documents the mortality burden of the epidemic and its sharp spatial concentration (Moore et al., 2023; Zoorob, 2019), and the widening mortality gap between college- and non-college-educated adults (Case & Deaton, 2022; Foote et al., 2025). A second strand traces the crisis's effects beyond mortality, to local labor markets (Alpert et al., 2022b; Cho et al., 2021), family structure (Buckles et al., 2023), and political behavior (Arteaga & Barone, 2026), the latter two using geographic variation in OxyContin marketing intensity as an instrument. Pierce and Schott (2020) and Moore et al. (2023) instead link localized economic shocks (exposure to Chinese import competition and to legal trade flows, respectively) to overdose mortality. While this work draws on variation in prescription-opioid marketing or in trade exposure, I use variation in illicit fentanyl supply generated by a single trafficking organization, which lets me trace the consequences of the third wave specifically.

Second, the paper connects to a growing economics literature on how DTOs are organized and how they respond to shocks. One strand models the economics of drug production and interdiction (Mejia & Restrepo, 2016); another shows how a political shock (the breakdown of one-party protection networks) triggered cartel violence (Trejo & Ley, 2018); and a third shows how a policy shock (crackdowns on drug trafficking) pushed cartels to diversify into new illicit sectors (Battiston et al., 2024). López Cruz and Torrens (2026) show that the fentanyl shock reorganized trafficking routes inside Mexico, as Pacific-based organizations redirected corridors toward fentanyl-precursor ports and highways, igniting a second wave of homicides. These four papers study the impact of international drug trafficking on socioeconomic and mortality outcomes. My contribution is to leverage a pre-existing advantage of the Sinaloa Cartel to examine how wholesale supply dynamics shaped the U.S. fentanyl epidemic.

The remainder of the paper proceeds as follows. Section 2 describes the institutional background: the structure of the U.S. opioid crisis and the Sinaloa Cartel's pre-existing capacity to produce and distribute synthetic drugs. Section 3 details how I measure Sinaloa presence and lays out the empirical strategy. Section 4 describes the mortality, seizure, and auxiliary data. Section 5 presents the main

estimates and the heterogeneity results. Section 6 reports robustness checks and addresses threats to identification. Section 7 examines downstream effects beyond mortality. Section 8 concludes.

2. Background

2.1. The Opioid Epidemic in the United States

The opioid epidemic is the deadliest drug crisis in U.S. history. Since 1999, more than one million Americans have died from drug overdoses. For most of the past decade, overdose has been the leading cause of accidental death in the United States, ahead of motor-vehicle crashes. Among adults aged 18–44, it is now the leading cause of death of any kind (Centers for Disease Control and Prevention, 2025; National Center for Health Statistics, 2025). The age-adjusted overdose death rate more than doubled between 2014 and 2022, from 14.7 to 32.6 per 100,000 (National Center for Health Statistics, 2025). The burden extends well beyond mortality. The Council of Economic Advisers valued the annual cost of the crisis at several hundred billion dollars (Council of Economic Advisers, 2017), and a large literature ties opioid mortality to labor-market decline (Case & Deaton, 2015), family disruption (Buckles et al., 2023), and political realignment (Arteaga & Barone, 2026).

The epidemic has unfolded in three overlapping waves, each defined by a distinct dominant opioid and each shaped in large part by supply shocks (Figure 1). The first began in the late 1990s with the introduction and aggressive marketing of prescription opioids (Alpert et al., 2022a). The second was triggered around 2010, when the reformulation of OxyContin displaced dependent users toward heroin (Alpert et al., 2018; Evans et al., 2019). The third, and by far the most lethal, began around 2013 with the arrival of illicit fentanyl. Synthetic-opioid deaths rose from roughly 3,000 in 2013 to over 70,000 by 2022, and illegally made fentanyls were involved in 75.1% of U.S. overdose deaths by 2023 (Centers for Disease Control and Prevention, 2026), in most cases mixed with other illicit drugs.

The first two waves operated through the formation and redirection of demand: widespread prescription of medical opioids initially cultivated a dependent population, which later transitioned to illicit heroin following regulatory tightening. Rather than altering the underlying consumer pool, the third wave transformed the product itself. A cheap, fully synthetic opioid roughly fifty to a hundred times more potent than heroin entered the established distribution network, frequently

reaching consumers as an undetected adulterant in heroin or pressed into counterfeit prescription pills. Consequently, the fentanyl shock was driven by changes in the chemical composition and lethality of the illicit supply rather than by shifts in user demographics.

This transition was mainly driven by a shift in production and distribution technology, so its geography was shaped by which organizations could execute it earliest, at scale, and at lowest cost. That is the question this paper takes up. The fentanyl era remains the deadliest phase of the epidemic. Understanding how this illicit drug reached the communities it did, earlier and more severely in some places than others, is essential to anticipating the next supply-side shock.

2.2. The Supply of Illicit Opioids and the Sinaloa Cartel

The supply side of U.S. illicit opioid markets has long been dominated by Mexican drug trafficking organizations (DTOs). They produce and traffic most of the heroin and methamphetamine, and, since the mid-2010s, nearly all of the fentanyl consumed in the United States (DEA, 2020; Drug Enforcement Administration, 2013). Mexican DTOs control production in Mexico, the smuggling corridors across the southwest border, and wholesale distribution hubs throughout the country.

Nearly all fentanyl consumed in the United States entered through the Mexican supply chain, moving through the same corridors Mexican DTOs already used for heroin and methamphetamine: pressed into counterfeit pills or mixed into heroin and smuggled across the southwest border (DEA, 2016, 2018).⁴ What made this transition favor some organizations over others was the economics of the drug itself. As a fully synthetic opioid, fentanyl decoupled production from agriculture: it required no poppy cultivation, could be scaled within days rather than over growing seasons, and cut the cost per morphine-equivalent dose by roughly 98 percent, sharply lowering concealment and seizure risk per dose (DEA, 2020). The same potency made fentanyl a profitable cutting agent. Buyers in illicit retail markets cannot observe purity before consuming, so sellers can dilute or adulterate the product while keeping much of their clientele (Galenianos et al., 2012). A small

⁴A secondary channel of direct-to-consumer shipments of high-purity fentanyl and precursors from Chinese exporters operated in the early period, exploiting gaps in international mail screening (DEA, 2018; Mars et al., 2019; Moore et al., 2023). It carried far smaller quantities than the southwest-border flow and remained geographically diffuse (U.S. Postal Service Office of Inspector General, 2018). It contracted further after China's 2019 class-wide scheduling of fentanyl-related substances and the expansion of U.S. mail and customs screening, as synthesis consolidated within Mexico-based organizations working from legal trade flows (DEA, 2020).

quantity of fentanyl could stand in for a much larger quantity of heroin at a fraction of the cost, which helps explain why most fentanyl reached consumers mixed into heroin and other drugs. Which Mexican organization would come to dominate the U.S. fentanyl market was mostly settled by which one already had the capacity to synthesize it at scale.

Among Mexican DTOs, the Sinaloa Cartel was best positioned to fill that role, for three reasons. First, it had the production capacity. Sinaloa had operated clandestine synthetic-drug laboratories for more than two decades, giving it long-standing lab infrastructure and technical expertise that plant-based competitors lacked. Records from the Mexican Ministry of National Defense (SEDENA) show that over 2007–2013, before fentanyl entered U.S. markets, the Sinaloa Cartel accounted for 36 percent of all clandestine laboratories raided in Mexico, the largest share of any single organization (Appendix Figure A1), with seizures concentrated in the states under its territorial control along the Pacific corridor (Appendix Figure A2).⁵

Second, it controlled the precursor supply chain. Sinaloa's methamphetamine operations had already established procurement relationships with Chinese chemical suppliers, the same suppliers that would later provide fentanyl precursors (DEA, 2016, 2024). Nearly all SEDENA seizures of the methamphetamine precursor phenylacetone (P2P) over 2011–2013 were attributed to Sinaloa (Appendix Figure A3). The same dominance later reappeared for fentanyl precursors: over 2020–2023, seizures of the primary fentanyl precursors (NPP and ANPP) in the state of Sinaloa exceeded 2,000 kg, roughly forty times the quantity seized in Jalisco, the home territory of the rival CJNG (*Cartel Jalisco Nueva Generación*; Figure 3).

Third, its strategic geographic positioning provided a critical advantage (Figure 2). Sinaloa's control of Mexico's northern Pacific corridor gave it direct access to the Pacific-coast ports of entry through which precursor shipments from Asia arrived (Mazatlán and Topolobampo, both within Sinaloa's home territory), and situating laboratories along that corridor minimized the distance between synthesis sites and the U.S. border (DEA, 2020). Consistent with this, during the early diffusion period (2014–2018), fentanyl seizures in Mexico were concentrated almost entirely in the Sinaloa-controlled Pacific states of Baja California, Sonora, and Sinaloa (Appendix Figure A4).

Together, these three advantages gave Sinaloa a decisive head start. It was the first Mexican organization to produce and distribute fentanyl at scale, and this precedence is visible in the historical record: Sinaloa-linked fentanyl activity

⁵I thank María Montoya-Aguirre for generously sharing the SEDENA seizure data.

appears in DEA field reporting, and in specialized press coverage of the trade, years before comparable activity is documented for rival organizations such as the CJNG (DEA, 2016, 2024). Fentanyl fit within Sinaloa's existing synthetic-drug production model instead of requiring a new line of business, so the organization could introduce it into U.S. markets earlier than rival cartels while keeping lower marginal production costs.

Early fentanyl distribution followed Sinaloa's established wholesale networks instead of expanding into new markets. Federal assessments consistently document that fentanyl distribution initially overlapped with the counties where Sinaloa already ran heroin and methamphetamine wholesale operations, reflecting continuity in logistics, not geographic expansion (DEA, 2009, 2018). This path dependence in illicit supply chains (new drugs introduced through established networks) motivates the empirical strategy that follows: a county's pre-fentanyl exposure to Sinaloa should predict the timing and intensity of its fentanyl wave.

This advantage was not permanent, and it eroded on two separate margins. On the Mexican side, the CJNG began building rival infrastructure: according to specialized press and law-enforcement reports, by 2018 the CJNG was able to produce and distribute fentanyl as well. Yet as of the 2015 NDTA, the intelligence record I use to measure pre-fentanyl exposure, the CJNG had not yet been designated a Consolidated Priority Organization Target and was explicitly excluded from the report's cartel territorial maps (Drug Enforcement Administration, 2015): its emerging control of Mexican trafficking routes had not yet translated into comparable U.S. wholesale distribution capacity. That translation came later, on the U.S. side. After 2018, the CJNG replicated Sinaloa's synthetic-production model and built out its own U.S. distribution channels, eroding Sinaloa's distributional edge (DEA, 2018, 2020). Whether the mortality effect of early Sinaloa presence attenuates or persists in later years is theoretically ambiguous. Widespread entry should dissipate the initial supply shock, but counties with longer exposure may have developed entrenched local dependence that sustains elevated mortality even as market share equalizes. The net effect is an empirical question.

3. Measurement of Sinaloa Presence and Empirical Strategy

3.1. Measuring Sinaloa Cartel Presence

Measuring the geographic footprint of a transnational criminal organization is difficult: cartels operate through intermediaries, deliberately obscure their presence, and leave no administrative paper trail. No single source provides systematic, high-coverage data on local trafficking activity in the United States.

My primary measure comes from the 2015 National Drug Threat Assessment (NDTA), which compiles intelligence from DEA field offices (Figure 4); my estimation sample covers the 78 counties these offices map to after the restrictions described below. For each field office, I observe the share of drug cases attributed to each trafficking organization. I use this county-level variation in cartel presence to identify counties with a high presence, or robust dominance, of the Sinaloa Cartel. Let c index counties and $o(c)$ the DEA office covering county c . I observe $\text{SinaloaShare}_{o(c)}$, the share of drug cases in that office linked to Sinaloa, and define a county treatment indicator equal to one when Sinaloa accounts for a majority of those cases:⁶

$$\text{Sinaloa}_c = 1(\text{SinaloaShare}_{o(c)} > 0.5) . \quad (1)$$

Each office is assigned to the county that contains its city; the main specification does not extend treatment to the rest of the metropolitan area (Section 6 reports that extension). Because certain offices are geographically contiguous, the estimation sample excludes treated counties in rival-dominated states and comparison counties in Sinaloa-dominated states, to mitigate spillovers (Appendix Figure A5).⁷

One limitation of this mapping is that most DEA offices sit in metro areas, so it disproportionately excludes rural counties. Still, the retained counties account for roughly 33% of all U.S. opioid overdose deaths in 2010, so the sample captures a substantial share of the mortality burden of interest.

A natural concern is that the NDTA 2015 was published after fentanyl began entering U.S. markets, so fentanyl-era investigations could in principle have shaped cartel attribution. Two considerations favor this baseline indicator. First, cross-validation against 2007–2022 NDTA reports confirms high spatial stability in Sinaloa’s operations (DEA, 2016). Second, the 2015 data offers fine-grained continuous coverage across field offices, allowing a clean dominance threshold that coarser

⁶ $\text{SinaloaShare}_{o(c)}$ is read directly from the NDTA maps, not computed from underlying case counts.

⁷State-level majority is computed weighting each county by population.

historical maps lack. I nonetheless check sensitivity to any residual contamination using an alternative measure drawn entirely from pre-fentanyl intelligence.

That alternative is the 2008 NDTA map of U.S. cities reporting the presence of organizations affiliated with the Sinaloa Federation (compiled from January–April 2008 reporting). I assign treatment to the counties containing those cities and re-estimate on the same sample, so only the assignment of treatment, not the set of counties, differs. Because this baseline strictly predates the fentanyl transition, it addresses potential reverse-causality concerns by design. Given its coarser, presence-only resolution, I employ it primarily to verify the robustness of the baseline estimates. Section 6 shows the two measures yield the same qualitative pattern.

To independently validate the intelligence-based assignment, I cross-check the baseline classification against local media archives. I build a text-based measure of Sinaloa presence following Sobrino (2019) and Coscia and Rios (2012), who use news content to map criminal presence in Mexico. I search Access World News, an archive of local and regional U.S. newspapers, for Sinaloa-related keywords (aliases, leadership names, trafficking terms). Local outlets routinely report cartel-linked arrests and operations, which anchors presence to specific localities.⁸ The measures agree closely. Over the pre-fentanyl period, newspapers in treated states averaged 9.1 Sinaloa-related mentions against 3.8 in control states, a ratio of roughly 2.4 to 1 (Figure 5), and the news-based geography corresponds closely to prior DEA assessments. Given that news coverage is disproportionately concentrated in major media markets, this metric serves primarily as a supplementary validation tool.

3.2. Empirical Strategy

To identify the causal effect of cartel presence on local mortality, I adopt an event-study difference-in-differences design that compares outcomes across counties exposed to pre-existing Sinaloa dominance versus rival cartel coverage. I take 2013 as the last pre-fentanyl year and omit it as the reference period. While CDC surveillance flagged isolated fentanyl-involved deaths before then, large-scale illicit manufacturing and distribution did not consolidate until 2014. Formally, I estimate:

$$Y_{ct} = \sum_{\tau \neq -1} \beta_{\tau} (\text{Sinaloa}_c \times \mathbf{1}[t - 2014 = \tau]) + \alpha_c + \gamma_t + X_{ct}\delta + \varepsilon_{ct}, \quad (2)$$

⁸I thank Eric Lease Morgan of the University of Notre Dame Hesburgh Libraries for performing the text extraction from the Access World News archive.

where Y_{ct} denotes the outcome of interest (in the baseline specification the fentanyl-related overdose mortality rate, and in later specifications other overdose, seizure, crime, and socioeconomic outcomes) in county c and year t ; Sinaloa_c is the treatment indicator defined in Equation (1); α_c are county fixed effects; γ_t are year fixed effects; and X_{ct} is a vector of time-varying demographic and socioeconomic covariates. The baseline vector of covariates, X_{ct} , controls for time-varying demographic composition, including the population shares of non-Hispanic White, non-Hispanic Black, and Hispanic residents, the share female, and an indicator for large (urban) counties; the population shares in the 15–34, 35–54, and 55+ age brackets; and the log of total population.⁹ Imports per capita, excluding oil, enters as an additional control only in the robustness specification that addresses local trade exposure as an alternative explanation (Section 6).

Because Sinaloa_c is time-invariant at the county level, the county fixed effects α_c absorb its level entirely. The treatment effect is identified only from the interaction terms $\text{Sinaloa}_c \times \mathbf{1}[t - 2014 = \tau]$, which vary over time. County fixed effects absorb time-invariant level differences between jurisdictions—such as baseline demographics, local institutions, or reporting infrastructure—so identification comes only from the *change* in mortality within Sinaloa-dominated counties before and after 2014, relative to the same change in comparison counties. Event time $\tau = t - 2014$ indexes years relative to 2014, so $\tau = -1$ corresponds to 2013, the omitted reference year. The coefficients β_τ trace the dynamic evolution of the outcome in Sinaloa-dominated counties relative to comparison counties dominated by rival trafficking organizations. Standard errors are clustered at the state level.

As a complementary and more parsimonious approach, I also estimate a standard difference-in-differences specification with a single post-period indicator:

$$Y_{ct} = \beta (\text{Sinaloa}_c \times \text{Post}_t) + \alpha_c + \gamma_t + X_{ct}\delta + \varepsilon_{ct}, \quad (3)$$

where $\text{Post}_t = \mathbf{1}\{t \geq 2014\}$. This specification collapses the event-time interactions above into a single coefficient β , identified from the same source of variation: the differential change in outcomes after 2014 between Sinaloa-present and comparison counties. It includes the same county and year fixed effects and controls X_{ct} as Equation (2), and standard errors are again clustered at the state level. Both Equa-

⁹The share of the adult population with a college degree is excluded from the baseline: its within-county variation over the sample period is negligible (within SD = 0.003 vs. between SD = 0.013, $T = 11$ years). This pattern is consistent with infrequent ACS 5-year vintage updates, and the variable's inclusion destabilizes the treatment coefficient without adding identifying power.

tion (2) and Equation (3) are estimated weighting each county by its population aged 15 and older.

A potential concern is that the introduction of fentanyl itself altered the geographic footprint of the Sinaloa Cartel, rendering the treatment indicator endogenous to post-2014 market dynamics. Several pieces of evidence suggest this is unlikely. First, the NDTA 2015 reports cartel shares at the field-office level, a resolution that earlier, coarser maps lack. Second, cross-validating against NDTA reports from 2007 to 2022 reveals a highly stable geographic pattern, indicating that Sinaloa's U.S. distribution network was largely fixed prior to the fentanyl wave (DEA, 2016).

This addresses whether the 2015 measurement reflects post-2014 reshuffling. A deeper version of the concern is that Sinaloa's original choice of where to build heroin and methamphetamine infrastructure, made years before fentanyl existed, could itself be correlated with local conditions that predict a steeper fentanyl trajectory for reasons unrelated to Sinaloa. Section 2 speaks to this directly. Sinaloa's advantage rests on geography and infrastructure fixed well before 2013: SEDENA lab-seizure data show Sinaloa accounted for 36% of clandestine-lab seizures over 2007–2013, and its territorial control of the Pacific corridor gave it access to the deepwater ports used for precursor shipments, a position built for heroin and methamphetamine over decades. A trend break coinciding exactly with 2014 would require these pre-existing, geographically anchored features to also predict the timing and magnitude of a synthetic-opioid shock that originated in a supply innovation abroad, not in local drug-market conditions.

4. Data

4.1. CDC Mortality Data

I assemble a county-by-year panel of overdose mortality from the CDC Multiple Cause-of-Death Restricted Use Files, from which I construct annual counts of fentanyl-related deaths (T40.4), heroin deaths (T40.1), cocaine deaths (T40.5), and mixed-drug overdoses. Following standard NCHS coding practice (Hedegaard et al., 2021), I define opioid-involved deaths using multiple-cause ICD-10 codes T40.0–T40.4 and classify polydrug overdoses using all contributing causes listed on the certificate.¹⁰

¹⁰I depart from the broadest NCHS definition in excluding T40.6 (other and unspecified narcotics): the ICD-10 nomenclature assigns this code to poisonings by narcotics that cannot be identified as

Other studies correcting or defining opioid-involved mortality similarly restrict to an opioid-specific subset of the T40 codes instead of the full NCHS aggregate (Ruhm, 2018). Table 1 presents summary statistics for the mortality rates over the sample period, comparing 2010 with 2020. Appendix Table A1 reports the same rates separately for treated and control counties in the pre-treatment period (2010–2013). The estimation sample spans 2010–2020. I end the panel in 2020 to avoid confounding the fentanyl supply shock with the onset of the COVID-19 pandemic, which independently disrupted illicit drug markets.

Correcting for incomplete drug specification

A well-documented limitation of CDC mortality data is that a non-trivial share of overdose death certificates does not identify the specific drug involved: the underlying cause is coded as a drug poisoning, but no contributing substance is listed in the multiple-cause fields. Counting only deaths with an explicit fentanyl (T40.4) or opioid code therefore undercounts true mortality. Nationally, roughly 17% of overdose deaths between 1999 and 2020 lacked any drug classification, and applying a correction adds on the order of 28% more opioid-involved deaths relative to the uncorrected counts (Drake & Ruhm, 2023; Ruhm, 2018). Critically for a county-level design, the degree of under-specification varies substantially across geography and over time, reflecting differences in toxicological capacity and in coroner versus medical-examiner systems. If specification completeness differs systematically between Sinaloa-presence and comparison counties, the raw counts could bias the estimated treatment effect.

To address this, I implement the preferred regression correction of Drake and Ruhm (2023). I restrict to the overdose universe (UCD codes X40–X44, X60–X64, X85, Y10–Y14) and partition deaths into those with at least one specified substance and those with none. For each substance and each year, I estimate a probit of the substance indicator on decedent characteristics (sex, age, education, marital status, race/ethnicity, place of death, and day of the week), together with state fixed effects, using only the drug-specified sample. The estimated model is then used to predict the probability of each substance’s involvement for the drug-unspecified deaths, which are assigned a fractional involvement in $[0, 1]$ instead of being discarded.¹¹

a specific opioid, as well as by psychodysleptics (hallucinogens), so a share of T40.6-coded deaths cannot be reliably attributed to opioid involvement at all.

¹¹Where a probit cannot be estimated or predicted (e.g., perfect prediction or sparse cells), I fall back to the year-by-state and then year-level share of the substance among specified deaths.

The resulting fractional corrected outcomes are aggregated to the county-year level and used to re-estimate the main specifications. Because the procedure is estimated separately by year, it accommodates the changing composition of overdose deaths across the sample period.

4.2. Drug Seizure Data

I use drug seizure data as a first-stage check on the proposed mechanism: if pre-fentanyl exposure to Sinaloa increased local fentanyl availability, treated areas should show a post-2014 rise in fentanyl-positive seizures, independently of the mortality data. My source is the National Forensic Laboratory Information System (NFLIS-Drug), which compiles laboratory-confirmed identifications of seized substances from roughly 284 state and local forensic laboratories, covering an estimated 98% of the national drug caseload. The dataset reports the number of identifications by substance and by state, including fentanyl, heroin, cocaine, and methamphetamine. Figure 6 plots raw trends in identifications per 100,000 by substance for treated and comparison states. Fentanyl identifications diverge sharply in treated states after 2014, while heroin and cocaine do not. A limitation is that NFLIS is aggregated at the state level, which rules out a direct county mapping and likely weakens its observed correlation with local mortality outcomes.

4.3. Additional Outcomes and Controls

Beyond mortality, I examine whether the fentanyl supply shock propagated to local public safety using longitudinal crime data from the Uniform Crime Reporting (UCR) program. I use the concatenated files of Kaplan (2021) for the “Offenses Known and Clearances by Arrest” series for 2010–2022, a standardized record of crimes reported to law enforcement agencies nationwide. I focus on two aggregates: the *Index Violent Crimes* (murder, robbery, aggravated assault, and rape) and the *Index Property Crimes* (burglary, motor vehicle theft, and larceny-theft). To ensure consistency with the mortality analysis, I aggregate agency-level reports to the county-year level and apply the same treatment definition based on pre-fentanyl Sinaloa presence.¹²

To ensure the estimated effects of Sinaloa trafficking infrastructure are not confounded by other concurrent shifts in the opioid market or local economic con-

¹²Instead of restricting to agencies that report in every month, which would drop counties and risk selecting on reporting capacity correlated with local conditions, I retain the full county panel and adjust for coverage using Kaplan’s months-missing indicators (Kaplan, 2021).

ditions, I integrate several additional sources used in the robustness specifications. First, I account for the historical legal environment of opioid supply using the triplicate prescription requirement identified by Alpert et al. (2022a), which predated the 1990s marketing of OxyContin and predicts lower initial prescription-opioid penetration and subsequent shifts to illicit markets. Second, I incorporate the timing of Mandatory Access Prescription Drug Monitoring Programs (PDMPs) compiled by Kim (2021), controlling for state-level policy interventions aimed at curbing the crisis. Finally, following Moore et al. (2023), I use state-level trade exposure constructed from the Census Bureau's *USA Trade Online* portal, isolating the cartel's organizational capacity from broader trends in international trade and the "China shock" (Autor et al., 2016) that may independently correlate with overdose mortality.

Population denominators. Mortality rates per 100,000 are computed using county-level intercensal population estimates from the Census Bureau. The heterogeneity analyses by education and race/ethnicity use group-specific denominators from NHGIS. The panel spans 2010–2020.

5. Effects on Overdose Mortality

I begin by documenting the geographic patterns that characterized the fentanyl wave. Figure 7 maps county-level fentanyl mortality over time: essentially absent in 2010, fentanyl deaths first emerge around 2014 in New Mexico and Appalachia, spread by 2018 through Appalachia, the Northwest, and parts of the Midwest, and blanket the country by 2022. Figure 8 shows that the areas hit earliest and hardest by fentanyl are precisely those with elevated overdose mortality in 2010–2014, consistent with fentanyl diffusing through pre-existing drug markets. By 2022, however, overdose mortality is far more geographically dispersed. One potential explanation is that fentanyl was increasingly mixed with stimulants as well as heroin, especially after 2017. This would have extended the risk to areas with little prior heroin exposure but established cocaine and methamphetamine markets.

Turning to the effect of exposure to the Sinaloa Cartel, I show the raw trajectories of fentanyl and non-fentanyl-opioid mortality in Figure 9, Panels (a) and (c). Counties with and without Sinaloa presence follow similar trajectories of fentanyl and non-fentanyl-opioid mortality before 2014. Once fentanyl production took off, the series diverge sharply. Between 2013 and 2020, fentanyl mortality rose by 26.1 deaths per 100,000 in Sinaloa-presence counties against 13.4 in comparison counties,

an increase nearly twice as large, while non-fentanyl-opioid mortality followed comparable or slightly lower paths in both groups.

The event-study estimates confirm this pattern. Sinaloa-dominated counties followed trends statistically indistinguishable from those of comparison counties dominated by other cartels before 2014. I find no evidence of differential pre-trends in fentanyl mortality (Figure 9, Panel (b)) or in non-fentanyl-opioid mortality (Panel (d)). After 2014, the fentanyl coefficients are positive, grow over time, and become statistically significant by the end of the period. By contrast, overdose deaths involving opioids other than fentanyl remain flat or decline slightly in both groups, with event-study coefficients statistically indistinguishable from zero throughout (Panel (d)).

Sinaloa presence is associated with a large and statistically significant increase in fentanyl mortality of 5.52 deaths per 100,000 in the post-2014 period, a 742% increase relative to the pre-treatment mean of 0.74, from the pooled difference-in-differences estimates of Equation (3) (Table 2, Column 1). Fentanyl deaths with no co-occurring cocaine, heroin, or methamphetamine (Column 6) rise by 2.41 deaths per 100,000 ($p < 0.10$), roughly 44% of the overall fentanyl effect, indicating that mixing with other drugs accounts for the majority of Sinaloa's impact on fentanyl mortality. This matches the underlying composition of the data: nationally, about 52% of fentanyl-involved deaths in 2020 also involved cocaine, heroin, or methamphetamine, a share that is nearly identical in Sinaloa-present and comparison counties.

An immediate alternative reading treats this as substitution: fentanyl may simply have displaced heroin or prescription-opioid deaths that would have occurred anyway, leaving total overdose mortality in Sinaloa-present counties unchanged. The paper's own results speak against this. Other-opioid mortality excluding fentanyl shows no significant response (Column 2), and single-substance heroin, cocaine, and methamphetamine mortality are all null (Appendix Table A2). The all-opioid effect of 6.13 deaths per 100,000 (Section 6) is close to the fentanyl-specific estimate of 5.52, consistent with addition to overall mortality.

Polydrug mortality combining fentanyl with cocaine, heroin, or methamphetamine (Columns 3–5) rises by a statistically significant amount for fentanyl-with-cocaine (1.66) and fentanyl-with-heroin (1.48), with a smaller but statistically significant increase for fentanyl-with-methamphetamine (0.67, $p < 0.05$).

The fentanyl-methamphetamine coefficient is modest by comparison, and its timing differs from the other combinations: event-study estimates for this outcome are indistinguishable from zero through 2016 and only turn marginally significant start-

ing in 2017. This delayed pattern is more consistent with demand-driven mixing, sometimes called a “goofball,” than with a supply-side push tied to Sinaloa’s earlier shift to P2P methamphetamine production. Deaths involving methamphetamine combined with any opioid were already common before 2014, and the vast majority of that pre-treatment combination involved opioids other than fentanyl; after 2014, that combination’s prevalence stays roughly constant, and what shifts is which opioid it involves. The two markets are also geographically complementary: fentanyl entered largely through the East Coast heroin markets described in Donahoe and Soliman (2025), while methamphetamine mortality has historically been concentrated in the West and rural South. Scaled by the pre-2014 mean of methamphetamine mortality, the fentanyl-methamphetamine coefficient represents a 52% increase, smaller than the 64% increase for fentanyl-with-heroin relative to the pre-2014 mean of heroin mortality, confirming that methamphetamine’s weaker response holds up on a proportional basis as well.

The corresponding single-substance estimates in Appendix Table A2 show fentanyl mortality rising sharply while cocaine and methamphetamine mortality alone show no statistically significant change, consistent with the fentanyl-cocaine poly-drug result above: cocaine mortality rises when fentanyl is also present, not as an independent expansion of the stimulant market.

To quantify the contribution of Sinaloa’s distribution infrastructure to the fentanyl wave, I compare the estimated treatment effect to the observed excess fentanyl mortality. The population-weighted fentanyl mortality rate increased by 18.59 deaths per 100,000 between 2014 and 2020 in my sample. The estimated effect of Sinaloa presence, 5.52 deaths per 100,000, represents approximately 29.7% of that increase.¹³

The mortality burden is heavily concentrated among less-educated populations. For individuals without a college degree, Sinaloa presence raises fentanyl mortality by 15.1 deaths per 100,000 in the post-period (more than ten times the pre-treatment group mean), against 1.5 among the college-educated (Appendix Table A4). The gradient holds across every opioid outcome. Heroin-involved mortality also rises for the non-college group. Because this outcome includes deaths with fentanyl co-involvement, the increase plausibly reflects contamination of the heroin supply, not an expansion of heroin use itself, consistent with the null effects on heroin-only mortality reported above.

¹³This compares the estimated treatment effect $\hat{\beta} = 5.52$ directly to the observed increase $\Delta \bar{Y} = 18.59$ deaths per 100,000 between 2014 and 2020.

The burden also varies by race and ethnicity. Appendix Table A5 repeats the exercise by race/ethnicity of the decedent. The largest and most precisely estimated effects are among Hispanic populations: fentanyl mortality rises by 6.7 deaths per 100,000 in the post-period, roughly eighteen times the group's pre-treatment mean, and Hispanic heroin-involved mortality also increases. Effects for non-Hispanic Whites are sizable (4.9 additional fentanyl deaths per 100,000) though less precisely estimated, while estimates for non-Hispanic Blacks are too imprecise to be informative, reflecting small Black populations in many sample counties. This pattern is difficult to reconcile with the leading alternative account of the opioid epidemic's third wave: that it stemmed from unintended consequences of prescription-opioid supply restrictions. That channel predicts increases concentrated among Whites, who report the highest rates of prescription-opioid misuse (Case & Deaton, 2015). Sinaloa's fentanyl-adulterated heroin instead reached buyers across each county's existing heroin market irrespective of race, so its mortality toll fell on populations largely unexposed to prescription opioids, a pattern that echoes the heroin-quality channel documented by Donahoe and Soliman (2025). It also matches the prediction of Mulligan (2024), whose model of choice between prescription and illicit sources implies that cheaper illicit opioids raise mortality most among groups with limited prior access to prescriptions, a mechanism he uses to explain why Black opioid mortality overtook White mortality in the late 2010s.

A third margin is decedent age. Appendix Table A6 splits the same outcomes into three brackets: 15–34, 35–54, and 55 and older. The effect concentrates among the working-age population: fentanyl mortality rises by 6.6 deaths per 100,000 among 15–34 year-olds and 8.1 among 35–54 year-olds, both statistically significant, while the coefficient for residents 55 and older (2.3) is not distinguishable from zero. The all-opioid outcome follows the same pattern. Heroin-involved mortality rises only for the 35–54 group, and only at the 10% level. The fentanyl-heroin combination is the exception, rising significantly across all three brackets; the 55+ coefficient there is measured against a pre-treatment mean of just 0.017 deaths per 100,000, so the implied percentage increase is not a meaningful magnitude.

6. Robustness and Threats to Identification

In this section I provide evidence in support of the identification strategy, explore alternative explanations for the findings, and test the sensitivity of the results.

A key identifying assumption is the absence of differential pre-trends in fentanyl

mortality between treated and comparison counties. Figures 9 and A6 support it: pre-2014 coefficients are small and statistically indistinguishable from zero across all outcomes, showing that comparison counties, where fentanyl distribution networks were less developed than Sinaloa's, did not differ in mortality trends before the fentanyl wave.

Permutation test. I assess how unusual the treated-county increases are relative to overall variability in opioid mortality. For each outcome, I randomly reassign Sinaloa presence across counties and re-estimate Equation (3) 10,000 times, on the same estimation sample used in Table 2. Each draw assigns placebo treatment to a random set of counties of the same size as the actual treated group, without regard to state boundaries, and keeps standard errors clustered at the state level. Figure 10 plots the resulting placebo distributions. The estimated fentanyl effect (5.52) and the all-opioid effect (6.13) both exceed the 99th percentile of their placebo distributions ($p = 0.006$ and $p = 0.009$, respectively), and the fentanyl-with-heroin effect exceeds the 95th percentile ($p = 0.026$). The estimated effects are thus highly unlikely to arise from the random assignment of treatment.

Wild cluster bootstrap. Inference is clustered at the state level throughout, but the sample contains only 37 state clusters, unbalanced between 22 treated and 15 comparison states, a setting in which analytical cluster-robust standard errors can over-reject (Cameron et al., 2008). As a check, I re-estimate Equation (3) and compute wild cluster bootstrap- t p -values and confidence intervals for each coefficient, using the six-point weights of Webb (2014) and the null imposed (Roodman et al., 2019), a procedure appropriate for this small and unbalanced number of clusters. Appendix Table A3 reports the full results. The main fentanyl estimate remains statistically significant at the 10% level under this more conservative procedure (bootstrap $p = 0.096$, 95% CI $[-1.01, 10.14]$). The polydrug coefficients retain the same sign and comparable magnitude, though they are estimated somewhat less precisely under the bootstrap (p between 0.09 and 0.18).

Alternative explanations. Figure 11 adds controls for leading alternative drivers: (i) the “triplicate” prescription requirement (Alpert et al., 2022a), (ii) mandatory-access PDMPs (Kim, 2021), (iii) international trade exposure (Moore et al., 2023), and (iv) baseline intensity of the earlier waves, including high pre-treatment OxyContin marketing (Alpert et al., 2018) and high initial heroin prevalence. The post-treatment

coefficients remain stable and mostly statistically significant across specifications.

Alternative measure of cartel presence. A potential concern is that the NDTA 2015 market-share measure might reflect post-fentanyl cartel realignments. To address this concern, I re-estimate the model assigning treatment from the 2008 NDTA—a map established well before the fentanyl wave. In this report, the Sinaloa Cartel is designated as the Sinaloa Federation and, due to reporting limitations, the measure captures Sinaloa presence rather than majority dominance. On the common sample, this alternative definition yields the same qualitative pattern: no differential pre-trends and a post-2014 divergence (Appendix Figure A7), implying 4.55 additional deaths per 100,000, compared with 5.52 under the baseline 2015 measure.¹⁴

Alternative comparison group. Appendix Figure A8 re-estimates the model, defining the control group as all remaining DEA-office counties, including the within-state spillover counties excluded from the main analysis. Because these plausibly experienced within-state spillovers, the estimates are conservative. The post-2014 fentanyl divergence remains evident, while non-fentanyl-opioid mortality shows no comparable pattern. These excluded counties include Los Angeles, San Diego, and Riverside, three of the largest methamphetamine markets in the country, so the fentanyl-methamphetamine estimates in Table 2 speak only to markets outside these three metro areas.

Metropolitan-area treatment. The baseline assigns each DEA office to the county that contains its city. To test whether the effect reaches the surrounding metropolitan area, I assign each office's treatment to every county in its 2013 core-based statistical area (CBSA) and apply the same within-state spillover rule. A satellite county is dropped when its CBSA contains offices with different treatment status or an office excluded from the main sample. The expanded sample has 364 counties: the 78 office counties and 286 satellite counties. The two-period estimate for fentanyl falls from 5.52 to 3.80 deaths per 100,000 ($p = 0.07$), and satellite counties on their own show 1.80 ($p = 0.31$), with no differential pre-trends in either sample (Appendix Figure A9). Non-fentanyl opioid mortality shows no effect in any of the three samples. The effect is concentrated in the counties where the DEA observed Sinaloa's operations, which fits a mechanism that runs through wholesale

¹⁴Both estimates use the same sample and specification as Table 2.

distribution hubs. Extending treatment to suburban counties adds units where the measure carries more error, and the estimate attenuates.

Incomplete drug specification. I re-estimate Equation (2) on outcomes corrected for incomplete drug specification following Drake and Ruhm (2023) (Appendix Figures A10–A14). The corrected fentanyl coefficients are indistinguishable from zero in the pre-treatment period and grow from 5.56 per 100,000 in 2016 to 11.93 by 2020, tracking the uncorrected path at somewhat larger magnitudes. Corrected all-opioid mortality follows the same shape (5.35 in 2016 to 8.45 in 2020), and the corrected fentanyl-heroin outcome is positive and statistically significant from 2017 onward (2.43 by 2020, $p < 0.05$). Because the imputation assigns fractional involvement to drug-unspecified deaths, it widens the confidence intervals, and the corrected point estimates for the individual substances are less precise in the final years. Still, the consistent sign, timing, and growth of the corrected series show that the divergence is not an artifact of how unspecified overdose deaths are coded.

7. Downstream Effects

Having established the mortality effect, I examine whether the fentanyl supply shock propagated to local crime, labor markets, and social insurance programs.

7.1. Crime

Transitions in illicit drug markets can be accompanied by local violence when distributors must establish control over a new market by force. The clearest U.S. precedent is the crack cocaine epidemic. Grogger and Willis (2000) show that the introduction of crack sharply raised violent crime, with no corresponding change in property crime, as distributors fought to establish territorial control in a newly emerging market, and Evans et al. (2016) document that, historically, higher crack-market violence translated directly into more homicides across U.S. cities. This pattern reflects a general feature of drug-market violence: it tends to arise when dealers must contest entry (Fagan & Chin, 1989; Moore & Pacula, 2021). Crack retail markets were also unusually fragmented, with many small dealers competing over street corners. The data are consistent with a different structure for Sinaloa's fentanyl distribution, which moved through a network that was already large and established, suggesting that entry may not have required displacing incumbent rivals. Analogous dynamics appear on the Mexican side of the fentanyl trade, where

cartels contest production zones and smuggling corridors (De Haro Lopez, 2023; Dell, 2015; Lindo & Padilla-Romo, 2018).

I therefore examine whether the U.S. fentanyl transition was accompanied by an increase in local crime, and if so, through which channel. Two channels could generate a response: territorial conflict between traffickers over market entry, of the kind documented above for crack, or acquisitive crime among users driven by economic hardship and addiction. A review of quasi-experimental evidence on the broader opioid crisis finds a weaker link to crime than the one documented for crack (Maclean et al., 2022), consistent with differences in how the two drugs are used and distributed. Violent crime speaks to the territorial channel; property crime, the more common marker of user-driven acquisitive crime, speaks to the economic-hardship channel.

In line with previous literature, I find no evidence of either channel operating at the local level. Using the Violent and Property Crime Indices from the UCR (Kaplan, 2021) (Figure 12), I find the coefficients on violent crime small and indistinguishable from zero throughout the post-period (Panel (a)), while the property crime coefficients are negative but not statistically significant (Panel (b)). The absence of a violent-crime response is consistent with a transition carried by Sinaloa's pre-existing U.S. wholesale network rather than a contest to establish one; the absence of a rise in property crime is inconsistent with the fentanyl wave inducing acquisitive crime among affected users.

7.2. Labor Markets and Social Insurance

Beyond crime, families and communities affected by the fentanyl wave may face economic strain through lost earnings, caregiving burdens, and the health and mortality toll of addiction (Buckles et al., 2023; Case & Deaton, 2015). This strain could raise demand for public transfer programs (Arteaga & Barone, 2026; Park & Powell, 2021), or it could instead be masked in local aggregates if the crisis removes affected working-age individuals from the population. I therefore examine labor-market outcomes and social-insurance caseloads to trace how the mortality shock propagated to the economic well-being of the surrounding community.

Labor-market aggregates show no evidence of deterioration. Appendix Figure A15 reports event studies for employment, unemployment, labor-force participation, and poverty from the ACS. Unemployment and poverty are flat throughout. If anything, employment point estimates are uniformly positive (though statistically insignificant) and non-participation declines. These results should be read

jointly with the mortality results: fentanyl deaths concentrate among prime-age, non-college individuals with weaker labor-force attachment, so mortality selection mechanically removes affected individuals from county aggregates and can mask individual-level harm.

Social insurance caseloads show a more nuanced pattern. Appendix Figure A16 reports effects on Supplemental Security Income receipt by category. Working-age (18–64) and blind/disabled caseloads show no change, indicating that the crisis did not translate into higher disability rolls. In contrast, aged SSI receipt (65+) rises in treated counties. While I cannot pin down the channel, this is consistent with economic strain among elderly households, including lost support from prime-age family members and assumed caregiving responsibilities, a margin documented by Buckles et al. (2023).

8. Conclusion

This paper examines the supply-side origins of the third wave of the opioid crisis by exploiting variation in the pre-fentanyl geographic footprint of the Sinaloa Cartel across U.S. counties. Using a difference-in-differences design that compares counties with documented Sinaloa presence in the 2015 DEA assessment to counties where other organizations dominated, I find that Sinaloa-presence counties experienced a large and statistically significant increase in fentanyl-related overdose mortality after 2014, on the order of 5.52 additional deaths per 100,000 residents aged 15 and older. This amounts to a 742% increase relative to the pre-treatment mean and accounts for roughly 30% of the observed rise in fentanyl mortality in my sample between 2014 and 2020.

The effect is specific to fentanyl. I find no statistically significant increase in non-fentanyl opioid mortality and no effect on stimulant mortality, indicating that the result operates through opioid supply channels rather than a general expansion of drug-market activity. Most of this effect, however, is not fentanyl acting alone: fentanyl-involved deaths with no co-occurring cocaine, heroin, or methamphetamine account for only 44% of the overall increase, and the remaining 56% comes from fentanyl mixing into the existing cocaine, heroin, and methamphetamine supplies of affected areas, extending overdose risk to users of those substances. This split mirrors the raw composition of fentanyl mortality nationally, where roughly 52% of fentanyl-involved deaths by 2020 also involve cocaine, heroin, or methamphetamine, with a nearly identical share in Sinaloa-present and

comparison counties.

The dynamics in later years are consistent with the changing structure of the market. After 2018, rival organizations, most notably the CJNG, replicated synthetic production and built their own precursor channels, so the initial Sinaloa supply advantage would be expected to attenuate. Yet the estimated effects keep growing through 2020 instead of reversing, consistent with entrenched local dependence sustaining elevated mortality even as the organization's distributional edge fades. I find no accompanying surge in violent or property crime, which underscores that the fentanyl transition was primarily a supply-side reorganization operating through pre-existing criminal infrastructure, with minimal territorial conflict on the U.S. side of the border.

These results fit price-based accounts of the opioid market. Mulligan (2024) shows that cheaper illicit opioids raise consumption most where prescription access was limited, and Sinaloa's distribution network is one concrete channel through which illicit opioids became cheaper and easier to obtain. One limitation of this paper is that the analysis covers residents aged 15 and older. Mulligan documents that opioid mortality among minors diverged from adult mortality after 2010, so the effect of Sinaloa's fentanyl supply on younger populations remains an open question.

These findings have implications for policy. They suggest that the rapid diffusion of illicitly manufactured fentanyl across U.S. drug markets was not an exogenous event but was channeled through pre-existing trafficking infrastructure. Disrupting those networks, not just focusing on demand- or prescription-side interventions, may be a critical part of reducing fentanyl-related mortality. The results also underscore the value of timely, granular supply-side surveillance. The fentanyl signal was visible in forensic seizure data through NFLIS well before federal authorities treated it as a first-order threat: although fentanyl began reaching U.S. markets in 2013–2014, it was not until around 2018 that it was widely recognized as the central driver of overdose mortality. As late as 2015, the DEA suggested in its National Drug Threat Assessment that fentanyl was unlikely to capture a significant share of the opioid market, anticipating that it would remain a limited adulterant consumed mostly without users' knowledge (Drug Enforcement Administration, 2015). The subsequent trajectory of the crisis decisively overturned that forecast. Systems like NFLIS, which track the composition of the drug supply in near real time, offer exactly the kind of early-warning capacity that this episode shows to have been missing. Sustaining and strengthening such supply-side surveillance may be

valuable for detecting the next shift in illicit drug markets before it is reflected in mortality.

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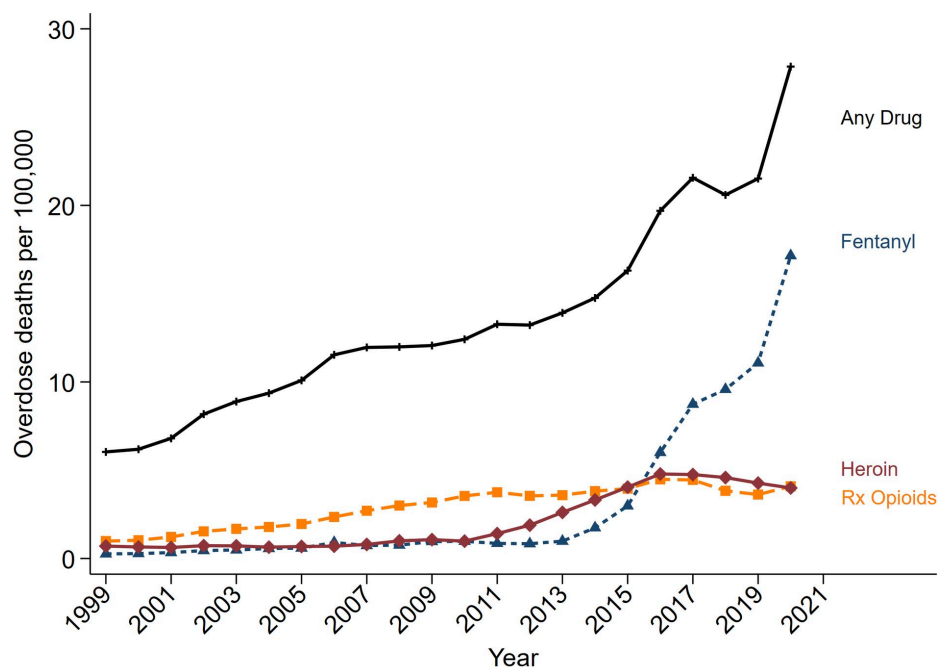
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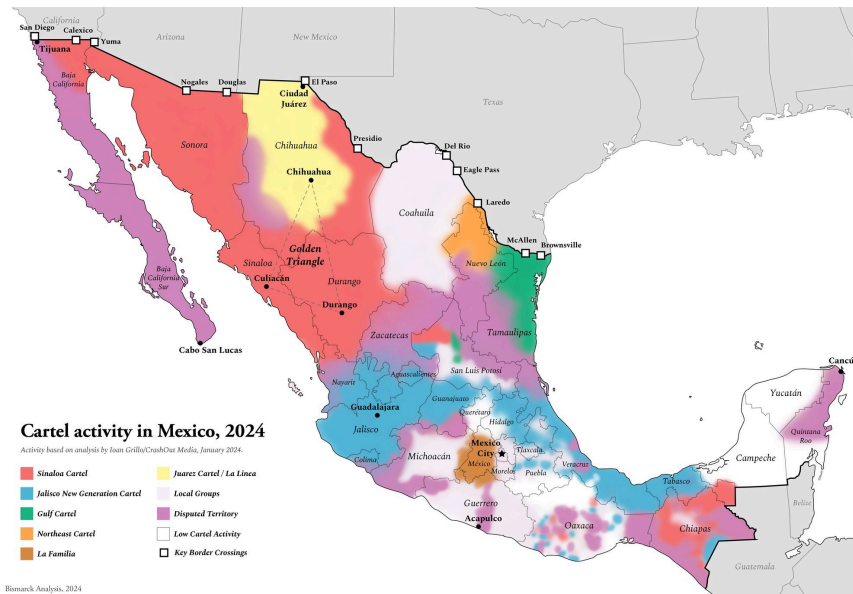
Figures

Figure 1: The Three Waves of the U.S. Opioid Crisis, 1999–2020



Notes: This figure plots national drug poisoning death rates per 100,000 by substance. Poisoning deaths are defined as those with drug poisoning as the underlying cause (ICD-10 X40–X44, X60–X64, X85, Y10–Y14). Substance involvement is identified through multiple-cause codes: T40.2 for prescription and semi-synthetic opioids, T40.1 for heroin, and T40.4 for synthetic opioids other than methadone (fentanyl). Source: CDC Multiple Cause of Death files, WONDER, 1999–2020.

Figure 2: Cartel Territorial Control in Mexico



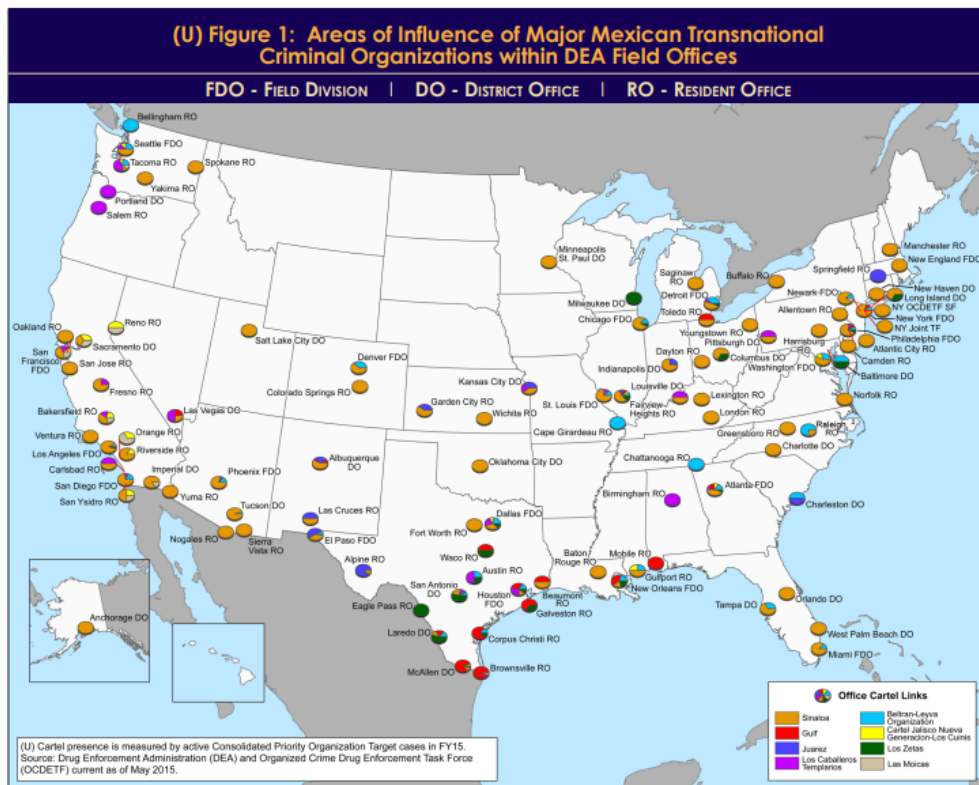
Notes: Approximate territorial control of major Mexican drug-trafficking organizations. Author's elaboration.

Figure 3: Fentanyl Precursor Seizures by Mexican State



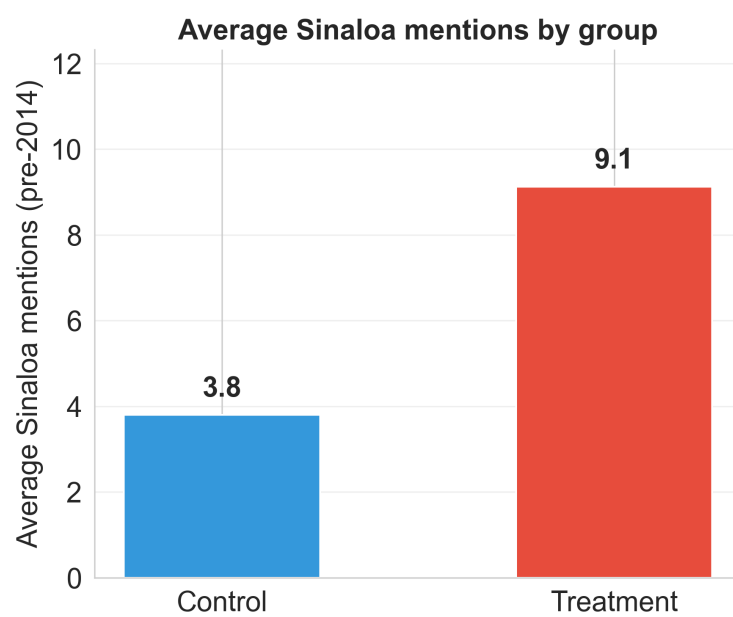
Notes: Fentanyl precursor (NPP and ANPP) seizures by Mexican state, sourced from SEDENA. Bubble size proportional to total kg seized.

Figure 4: Areas of Influence of Mexican Transnational Criminal Organizations, DEA NDTA 2015



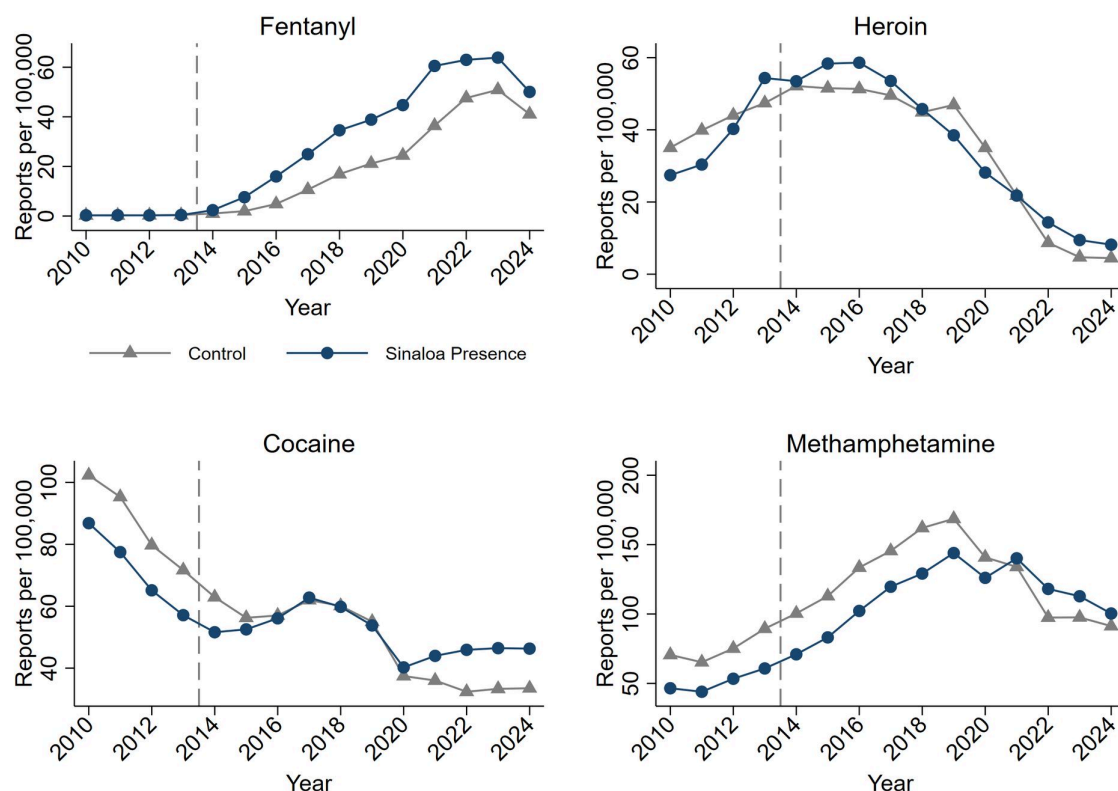
Notes: The map reports the share of drug cases linked to each organization at the DEA field-office level (Field Divisions, District Offices, and Resident Offices), as published in the 2015 National Drug Threat Assessment. Cartel presence is measured by active Consolidated Priority Organization Target cases in FY15. This is the primary source underlying the county-level treatment definition in Equation (1). Source: DEA and OCDETF, National Drug Threat Assessment 2015.

Figure 5: Validation of NDTA Treatment Definition Against Newspaper-Based Measure



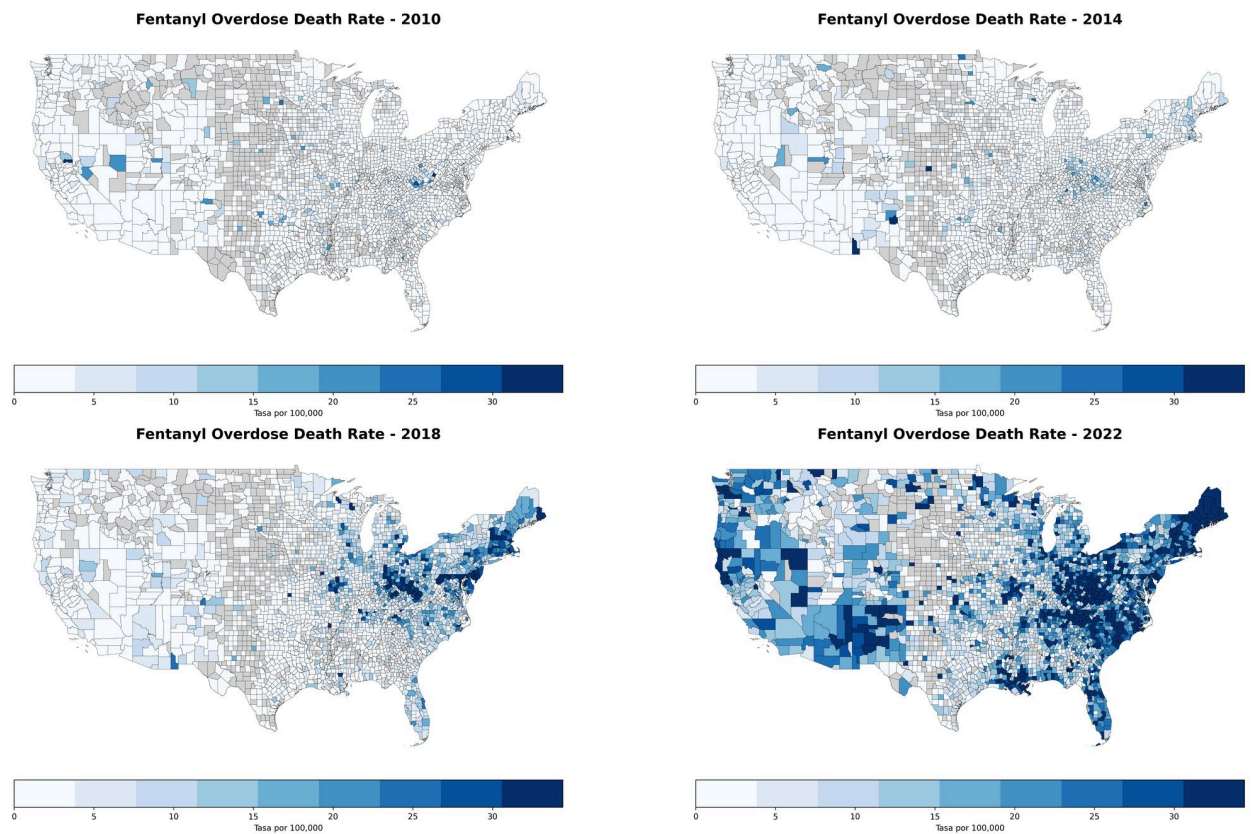
Notes: Average number of Sinaloa Cartel-related mentions in local newspapers per state over the pre-fentanyl period (2010–2013), by NDTA 2015 treatment status. Mentions are extracted from Access World News, an archive of local and regional U.S. newspapers, using organization-level keywords including cartel aliases, leadership names, and associated trafficking terms. Counties classified as Sinaloa-treated under the NDTA definition exhibit roughly 2.4 times more Sinaloa-related coverage than control counties. Source: Access World News; DEA National Drug Threat Assessment 2015.

Figure 6: Trends in Drug Identifications from NFLIS-Drug



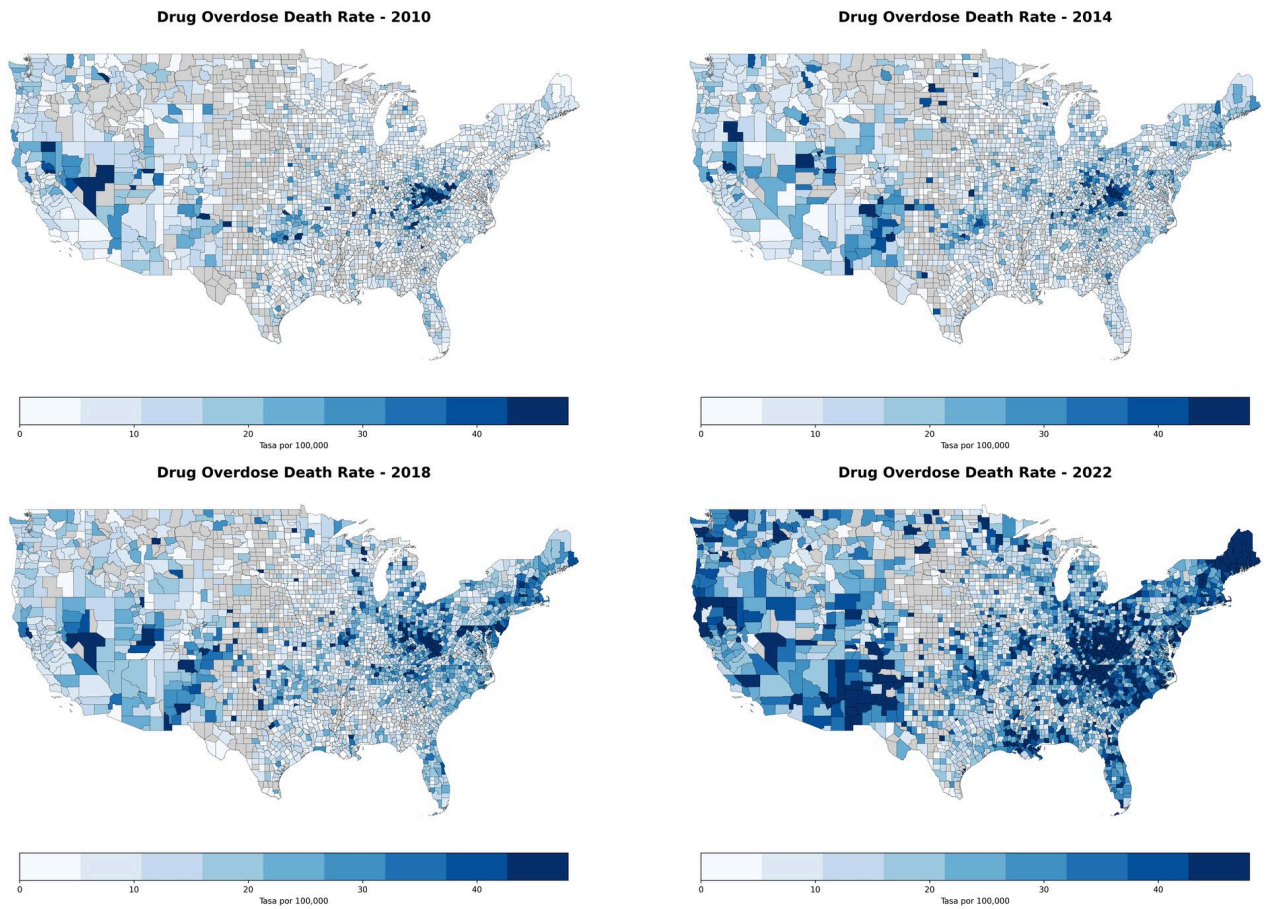
Notes: The figure shows drug identifications per 100,000 residents in treated (Sinaloa presence) and comparison states, reported by forensic laboratories participating in the National Forensic Laboratory Information System (NFLIS-Drug). NFLIS-Drug collects results from chemical analyses of substances seized in law enforcement operations and represents approximately 98 percent of the U.S. forensic drug caseload. Counts reflect the number of laboratory reports in which a given substance was identified and do not correspond to quantities or purity. NFLIS-Drug is reported only at the state level, so this comparison is between states rather than counties. Source: NFLIS-Drug public data and annual reports, U.S. Drug Enforcement Administration.

Figure 7: Fentanyl Poisoning Deaths by County



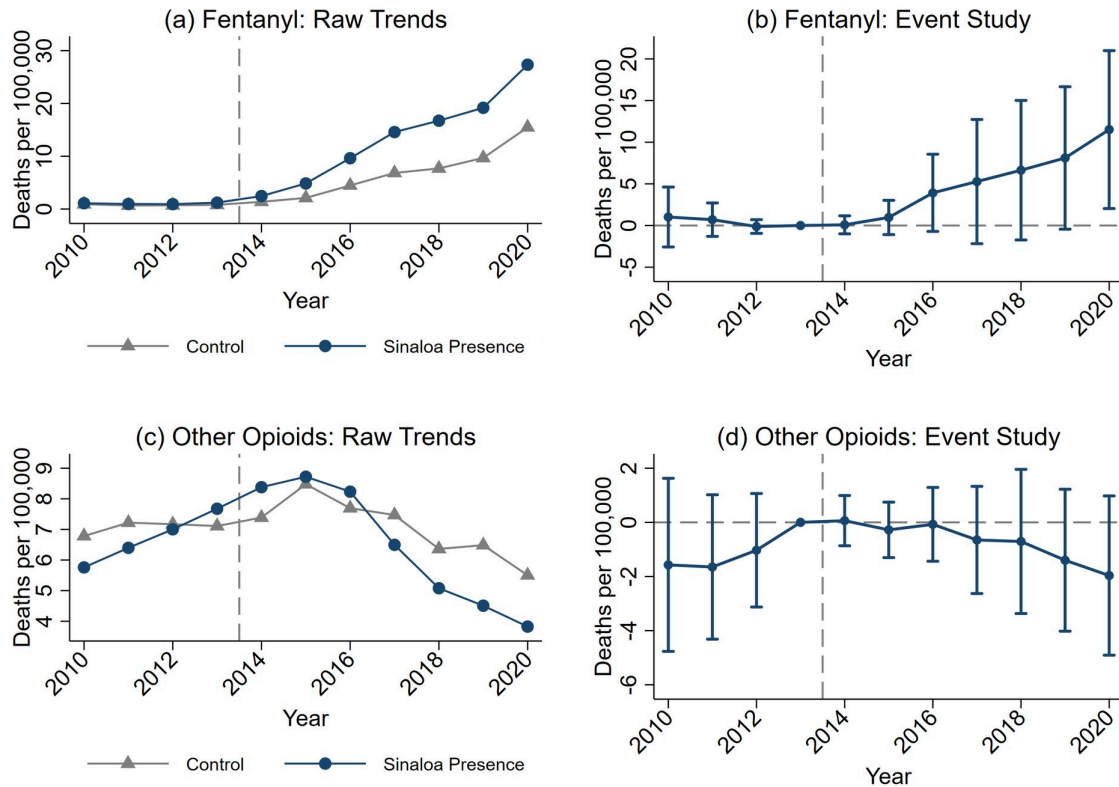
Notes: County-level fentanyl-involved poisoning mortality rates per 100,000 residents aged 15 and older, by year. Source: CDC Multiple Cause of Death files, 2010–2022.

Figure 8: Drug Poisoning Deaths by County



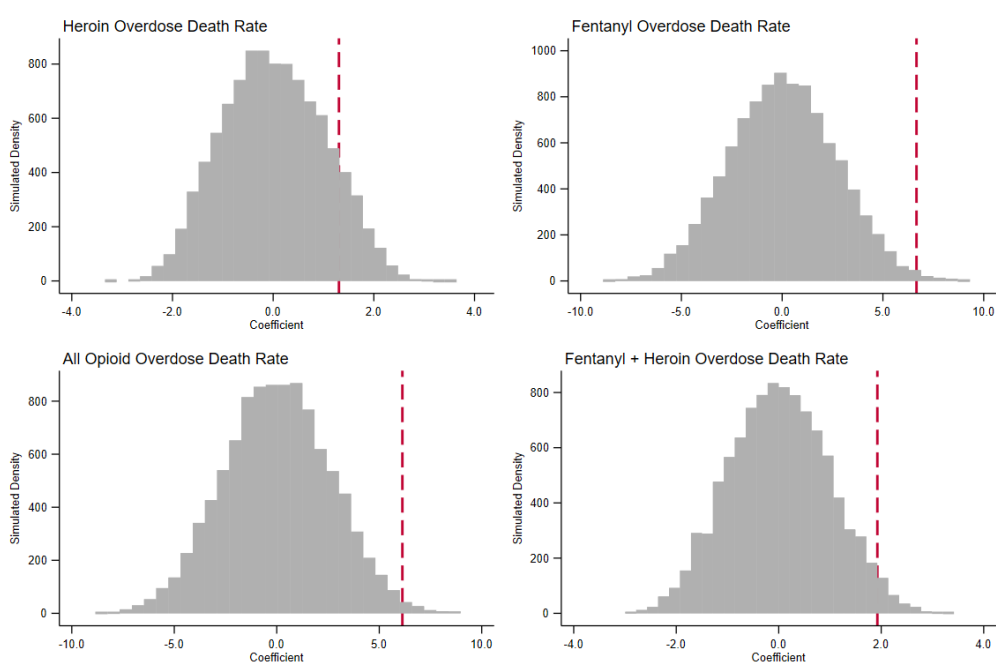
Notes: County-level drug poisoning mortality rates per 100,000 residents aged 15 and older, by year. Drug poisoning deaths are those with an underlying cause in ICD-10 codes X40–X44, X60–X64, X85, or Y10–Y14 and at least one drug class identified among the multiple causes of death. Source: CDC Multiple Cause of Death files, 2010–2022.

Figure 9: Sinaloa Cartel Presence and Opioid Mortality



Notes: The figure presents raw trends and event-study estimates of poisoning mortality at the county-year level from 2010 to 2020. Panels (a) and (b) report raw trends and event-study estimates for fentanyl-involved poisoning deaths; Panels (c) and (d) report the same for poisoning deaths involving opioids other than fentanyl. Raw-trend panels display population-weighted average mortality rates per 100,000 residents aged 15 and older by treatment status. Event-study panels report coefficients from Equation (2), normalized to 2013; the vertical line marks 2014, the onset of the fentanyl wave. All specifications include county and year fixed effects and demographic covariates. Estimates are weighted by county population aged 15 and older, and standard errors are clustered at the state level. Source: CDC Multiple Cause of Death files, 2010–2020.

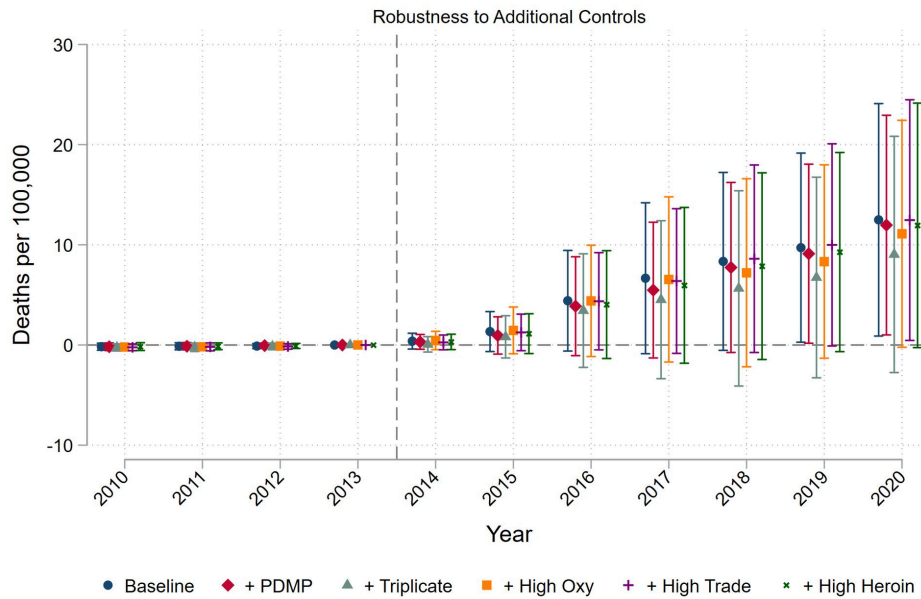
Figure 10: Permutation Tests for Sinaloa Presence



Notes: For these tests, we randomize which county gets 'treated' and then run the TWFE regression 10,000 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.

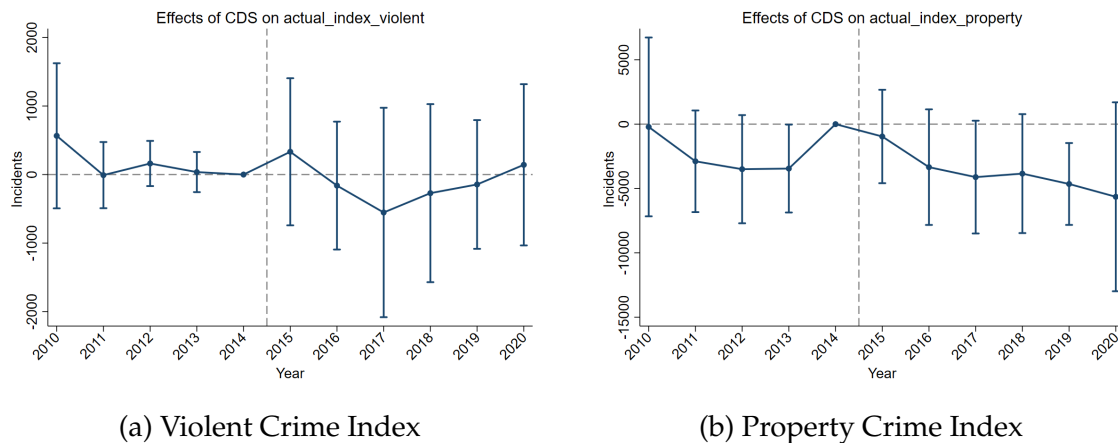
Notes: Placebo distributions from 10,000 random reassignments of Sinaloa treatment status across counties, re-estimating Equation (3) for each outcome. Vertical lines mark the estimated treatment effect from the true assignment. Source: CDC Multiple Cause of Death files, 2010–2020.

Figure 11: Robustness to Alternative Opioid Supply and Policy Explanations



Notes: This figure plots the TWFE coefficients for the interaction between Sinaloa exposure and the 2014–2018 period under different control regimes. The *Baseline* model includes county and year fixed effects plus demographic covariates. Subsequent models add indicators for: *Triplicate* states (Alpert et al., 2022a), *PDMP* implementation dates (Kim, 2021), *Trade* exposure (Moore et al., 2023), and *High Oxy/Heroin* to account for areas with high baseline levels of prescription and illicit opioids prior to the fentanyl wave. Error bars represent 95% confidence intervals clustered at the state level. Source: CDC Multiple Cause of Death files, 2010–2020.

Figure 12: Impact of Sinaloa Presence on Local Crime Rates



(a) Violent Crime Index

(b) Property Crime Index

Notes: This figure presents event-study estimates for the impact of Sinaloa Cartel presence on local crime. Panel (a) shows the effect on the violent crime index, and Panel (b) shows the effect on the property crime index. Crime data are sourced from the UCR as processed by Kaplan (2021). All specifications include county and year fixed effects and demographic covariates. Estimates are relative to 2013, the omitted year. Error bars represent 95% confidence intervals clustered at the state level.

Tables

Table 1: Summary Statistics: Overdose Death Rates per 100,000, 2010–2020

	2010		2020	
	Mean	SD	Mean	SD
Any opioid	7.04	4.61	25.85	19.07
Fentanyl (T40.4)	0.92	0.65	21.23	19.32
Heroin (T40.1)	1.37	1.55	5.50	3.80
Cocaine (T40.5)	2.58	1.59	9.44	7.95
Methamphetamine	0.95	1.19	9.22	6.80
Fentanyl + Cocaine	0.05	0.08	6.21	7.12
Fentanyl + Heroin	0.01	0.04	3.35	4.03
Fentanyl + Methamphetamine	0.02	0.05	3.79	4.10
Fentanyl + Cocaine + Heroin	0.00	0.01	1.02	1.36
Counties	78		78	

Notes: Unit of observation: county-year. Sample: U.S. counties, 2010–2020. Rates calculated as deaths per 100,000 population aged 15+. Any opioid (excl. fentanyl) is defined as any opioid death where fentanyl (T40.4) is not listed among the contributing causes.

Table 2: TWFE Estimates

	(1) Fentanyl	(2) Other Opioids (No Fentanyl)	(3) Fent+Coc	(4) Fent+Her	(5) Fent+Meth	(6) Fentanyl Only
Post*Sinaloa	5.521** (2.284)	0.152 (0.785)	1.662* (0.830)	1.475*** (0.527)	0.672** (0.295)	2.407* (1.282)
Observations	850	848	850	850	850	850
Mean Dep. Var.	0.744	6.264	0.0559	0.0268	0.0266	0.643

Notes: Each column reports coefficients from Equation (3), a county-level difference-in-differences with county and year fixed effects. Outcomes are overdose mortality rates per 100,000 residents aged 15 and older, by substance. Column (2) excludes fentanyl-involved death certificates before counting opioid deaths, so it reflects non-fentanyl opioid mortality in every county-year, not merely county-years unaffected by fentanyl. Column (6) reports fentanyl-involved deaths with no co-occurring cocaine, heroin, or methamphetamine. Regressions are weighted by county population aged 15+ and include demographic controls (race/ethnicity shares, female share, large-county indicator, age-group shares, log population). Share with a college degree is excluded from X_{ct} : its within-county variation over the sample period is negligible (within SD = 0.003 vs. between SD = 0.013), and its inclusion destabilizes the treatment coefficient without adding identifying power. Standard errors, shown in parentheses, are clustered at the state level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

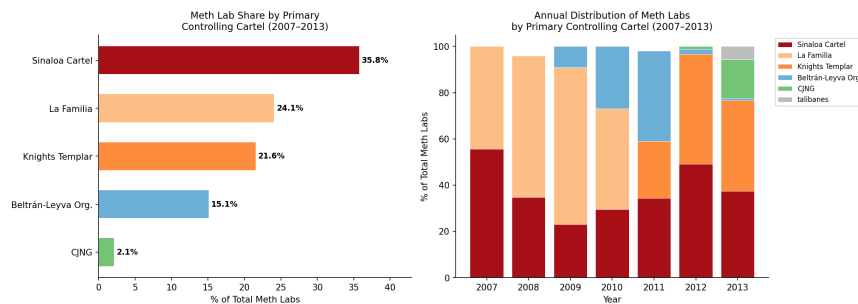
Appendix

Table A1: Summary Statistics: Overdose Death Rates per 100,000, 2010–2013

	Control (No Sinaloa)				Treated (Sinaloa)			
	Mean	SD	Min	Max	Mean	SD	Min	Max
<i>Panel A: Single-drug rates (per 100,000)</i>								
Any opioid	9.30	5.39	0.00	28.89	9.09	4.88	0.00	19.92
Any opioid (excl. fentanyl)	8.57	4.93	0.00	26.37	8.10	4.38	0.00	17.41
Fentanyl (T40.4)	0.73	0.63	0.00	3.14	0.98	0.98	0.00	5.27
Heroin (T40.1)	2.02	1.94	0.00	8.33	2.36	2.46	0.00	10.68
Cocaine (T40.5)	2.19	1.56	0.00	6.70	2.20	1.60	0.00	8.05
Methamphetamine	1.50	1.77	0.00	9.04	0.74	1.12	0.00	5.83
<i>Panel B: Polydrug rates (per 100,000)</i>								
Fentanyl + Cocaine	0.04	0.09	0.00	0.46	0.08	0.18	0.00	1.27
Fentanyl + Heroin	0.02	0.06	0.00	0.59	0.06	0.20	0.00	1.69
Fentanyl + Methamphetamine	0.04	0.09	0.00	0.46	0.03	0.15	0.00	1.69
Fentanyl + Cocaine + Heroin	0.00	0.01	0.00	0.15	0.01	0.05	0.00	0.42
Observations	139				167			

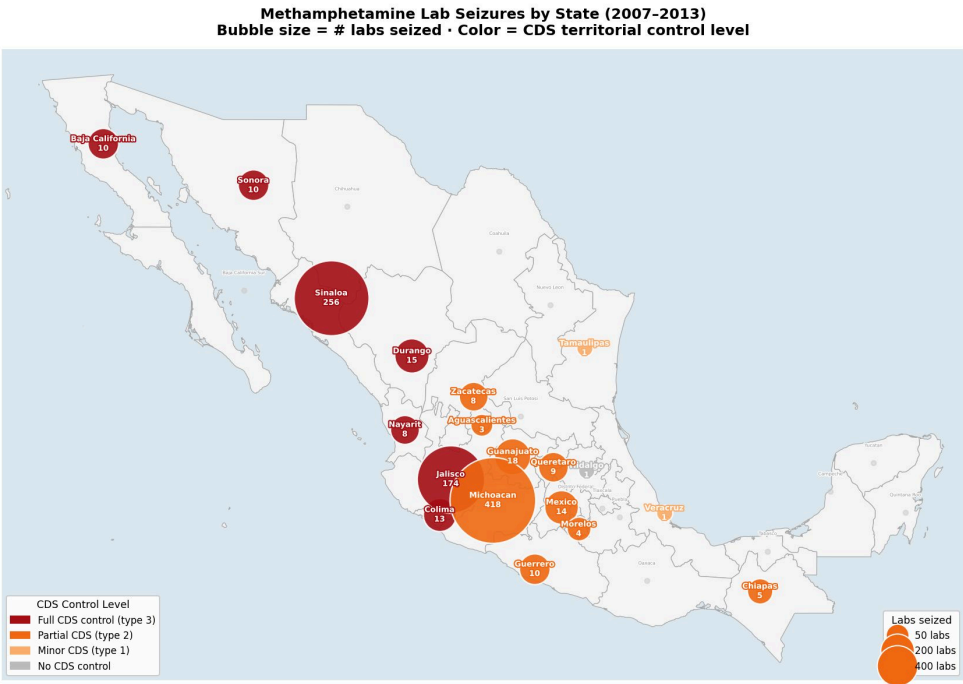
Notes: Unit of observation: county-year. Sample: U.S. counties, pre-treatment period 2010–2013. Rates calculated as deaths per 100,000 population aged 15+. Any opioid (excl. fentanyl) is defined as any opioid death where fentanyl (T40.4) is not listed among the contributing causes. Sinaloa presence is defined as a Sinaloa share above 50% of cartel-attributed drug cases in the DEA field office, based on NDTA 2015.

Figure A1: Clandestine Laboratory Seizures by Organization, 2007–2013



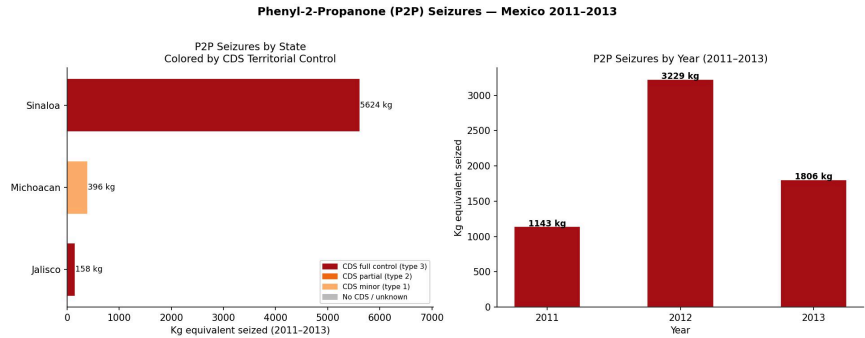
Notes: Share of clandestine drug laboratories raided by the Mexican Army over 2007–2013, by the criminal organization to which the laboratory was attributed. Author's elaboration based on SEDENA records.

Figure A2: Methamphetamine Laboratory Seizures by Mexican State, 2007–2013



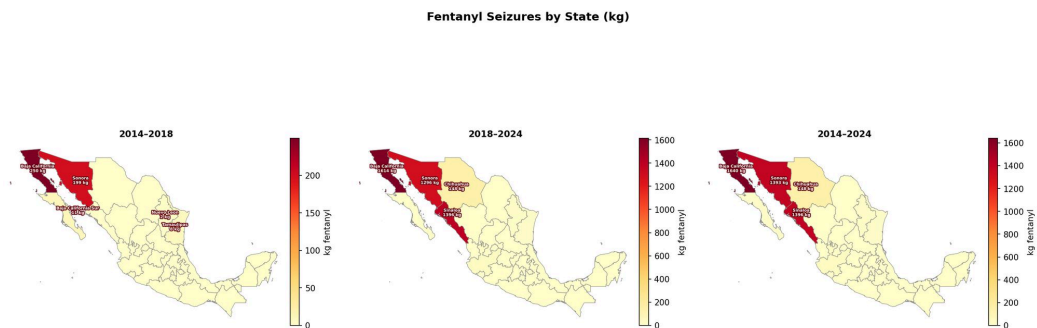
Notes: Clandestine methamphetamine laboratory seizures by Mexican state over 2007–2013, prior to the entry of illicitly manufactured fentanyl. Bubble size is proportional to the number of laboratories seized; color indicates the degree of Sinaloa Cartel (CDS) territorial control. States under full CDS control accounted for roughly half of all laboratory seizures nationwide. The largest single concentration, Michoacán, was dominated by La Familia Michoacana and the Caballeros Templarios, organizations largely dismantled by 2013–2015—before the consolidation of fentanyl production in Mexico. Source: SEDENA.

Figure A3: P2P Precursor Seizures by Organization, 2011–2013



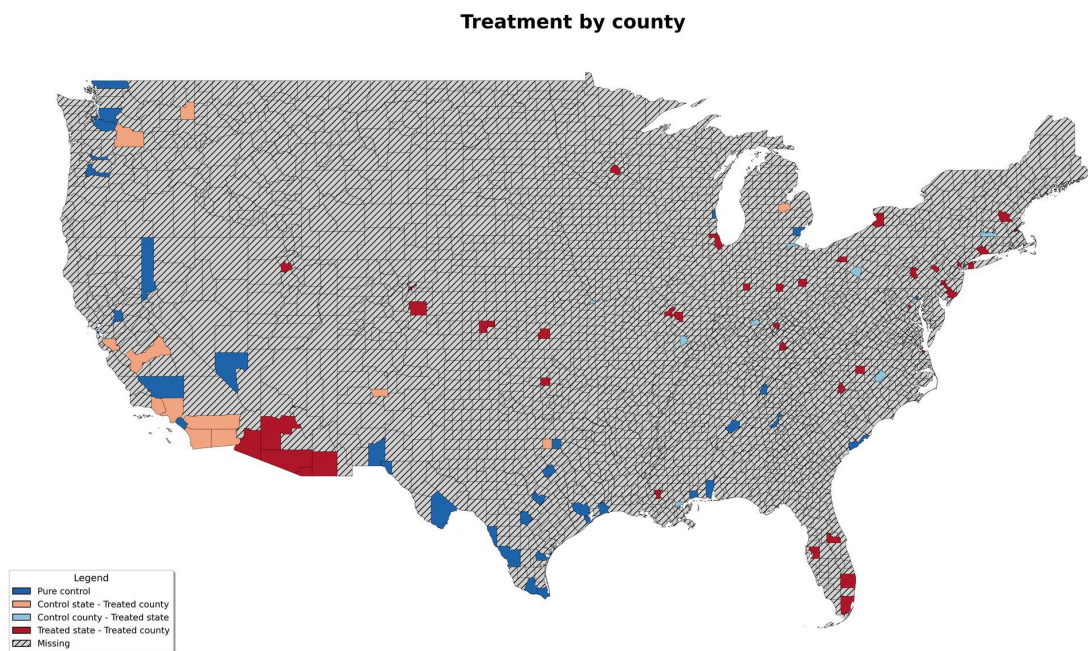
Notes: Seizures of phenylacetone (P2P), the principal methamphetamine precursor, attributed to each criminal organization over 2011–2013. Author’s elaboration based on SEDENA records.

Figure A4: Fentanyl Seizures by Mexican State, 2014–2024



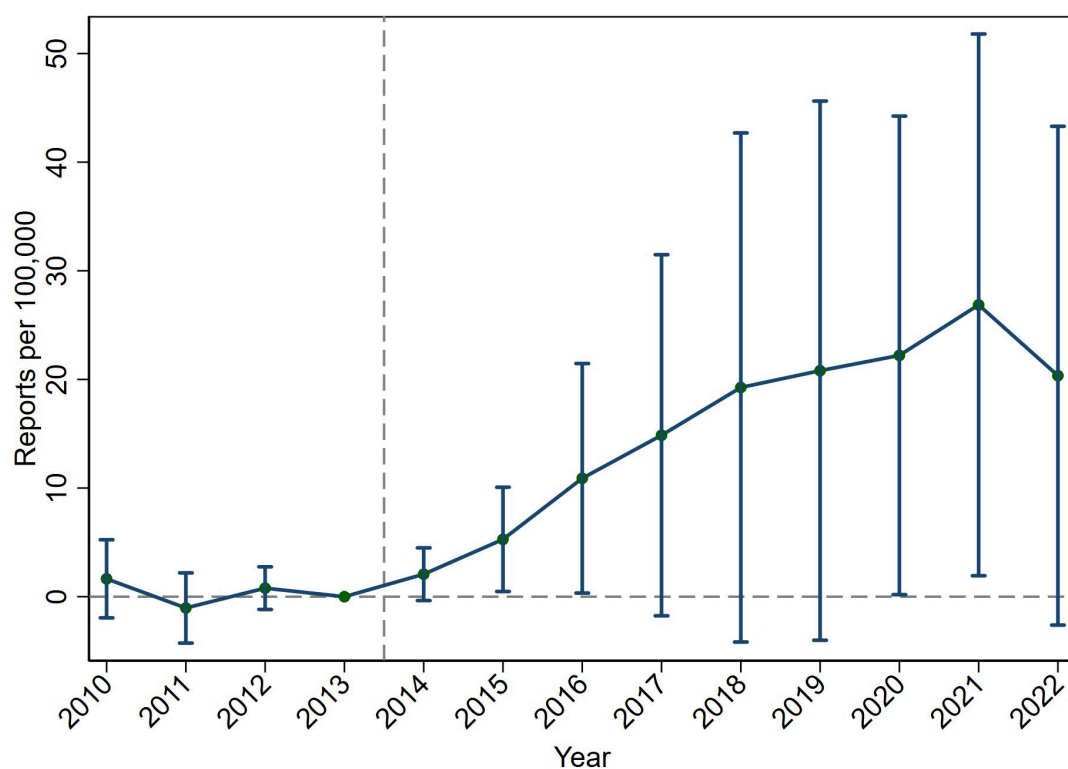
Notes: Fentanyl seizures (kg) by Mexican state across three periods. Source: SEDENA.

Figure A5: County-Level Treatment Assignment



Notes: Counties classified as treated (Sinaloa share of drug cases above 50 percent) and control under the NDTA 2015 definition, after excluding treated counties in comparison states and comparison counties in treated states. See Figure 4 for the underlying DEA field-office map.

Figure A6: Event Study Estimates for Fentanyl Seizures



Notes: Event-study estimates for NFLIS-Drug reports. The outcome is the rate of reports of a given drug per 100,000. Estimates are relative to 2013, the omitted year. Confidence intervals use standard errors clustered at the state level. Source: NFLIS-Drug public data, U.S. Drug Enforcement Administration.

Table A2: TWFE Estimates: Single-Substance Outcomes

	(1)	(2)	(3)	(4)
	Fentanyl	Heroin	Cocaine	Meth
Post*Sinaloa	5.521** (2.284)	1.303 (0.824)	1.220 (0.933)	-0.311 (1.030)
Observations	850	850	850	850
Mean Dep. Var.	0.744	2.246	2.282	1.512

Notes: Each column reports coefficients from Equation (3), a county-level difference-in-differences with county and year fixed effects. Outcomes are single-substance overdose mortality rates per 100,000 residents aged 15 and older. Regressions are weighted by county population aged 15+ and include demographic controls (race/ethnicity shares, female share, large-county indicator, age-group shares, log population); share with a college degree is excluded (negligible within-county variation). Standard errors, shown in parentheses, are clustered at the state level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

Table A3: Wild Cluster Bootstrap Inference

	(1) Fentanyl	(2) Other Opioids (No Fentanyl)	(3) Fent+Coc	(4) Fent+Her	(5) Fent+Meth	(6) Fentanyl Only
Post*Sinaloa	5.521** (2.284)	0.152 (0.785)	1.662* (0.830)	1.475*** (0.527)	0.672** (0.295)	2.407* (1.282)
Wild boot. p	0.0959	0.868	0.150	0.0861	0.132	0.176
Wild boot. 95% CI	[-1.01, 10.14]	[-1.90, 2.55]	[-0.63, 3.47]	[-0.25, 2.56]	[-0.23, 1.36]	[-1.21, 4.94]
Observations	850	848	850	850	850	850
Mean Dep. Var.	0.744	6.264	0.0559	0.0268	0.0266	0.643

Notes: Each column reports coefficients from the same specification as Table 2, Equation (3), a county-level difference-in-differences with county and year fixed effects. Column (2) excludes fentanyl-involved death certificates before counting opioid deaths. Column (6) reports fentanyl-involved deaths with no co-occurring cocaine, heroin, or methamphetamine. Regressions are weighted by county population aged 15+ and include demographic controls (race/ethnicity shares, female share, large-county indicator, age-group shares, log population); share with a college degree is excluded (negligible within-county variation). Standard errors, shown in parentheses below each coefficient, are clustered at the state level. "Wild boot." rows report wild cluster bootstrap- t p -values and 95% confidence intervals for the coefficient directly above (Cameron et al., 2008; Roodman et al., 2019), using Webb (2014) six-point weights (Webb, 2014), the null imposed (WCR), and 19,999 replications, clustered on state (37 states; 22 treated, 15 control). This addresses the small and unbalanced number of clusters in the sample.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$, based on the analytical cluster-robust standard errors.

Table A4: Heterogeneity by Educational Attainment

Outcome	Group	Pre-mean (per 100k)	2014+ $\hat{\beta}$ (SE)	% of mean (2014+)
Any Opioid	No College	12.625	15.368*** (2.780)	121.7%
	College	3.282	1.945*** (0.382)	59.3%
Fentanyl	No College	1.423	15.051*** (3.241)	1057.5%
	College	0.513	1.457** (0.548)	284.2%
Fent. + Heroin	No College	0.083	3.507*** (0.982)	4213.9%
	College	0.003	0.351** (0.170)	n.m. [†]
Heroin	No College	4.247	3.911*** (1.320)	92.1%
	College	0.638	0.515** (0.194)	80.7%

Notes: Rates per 100,000 population aged 25 and older in each education group, since educational attainment is defined for the adult population; this differs from the 15+ denominator used elsewhere in the paper. Estimates from Equation (3), with standard errors shown in parentheses, computed separately by educational attainment of the decedent. Pre-mean is the population-weighted mean in treated counties before 2013. Standard errors clustered at the state level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

[†] Percent-of-mean not reported (n.m.) for pre-means below 0.05 deaths per 100,000: the implied percentage is not an informative magnitude.

Table A5: Heterogeneity by Race and Ethnicity

Outcome	Group	Pre-mean (per 100k)	2014+ $\hat{\beta}$ (SE)	% of mean (2014+)
Any Opioid	NH White	10.004	4.799** (2.119)	48.0%
	NH Black	4.190	4.224 (3.957)	100.8%
	Hispanic	3.600	7.123*** (1.090)	197.9%
Fentanyl	NH White	1.194	4.923** (2.327)	412.4%
	NH Black	0.370	3.462 (3.937)	935.6%
	Hispanic	0.366	6.706*** (1.132)	1833.8%
Fent. + Heroin	NH White	0.069	0.931 (0.629)	1346.1%
	NH Black	0.046	1.756 (1.079)	n.m. [†]
	Hispanic	0.013	1.597*** (0.366)	n.m. [†]
Heroin	NH White	3.361	0.196 (1.042)	5.8%
	NH Black	2.165	1.777 (1.470)	82.1%
	Hispanic	1.336	2.046*** (0.268)	153.2%

Notes: Estimates from Equation (3), with standard errors shown in parentheses, computed separately by race/ethnicity of the decedent, using group-specific population denominators (deaths in group over county population share of the group, per 100,000). Pre-mean is the population-weighted mean in treated counties before 2013. Standard errors clustered at the state level.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

[†] Percent-of-mean not reported (n.m.) for pre-means below 0.05 deaths per 100,000: the implied percentage is not an informative magnitude.

Table A6: Heterogeneity by Age Group

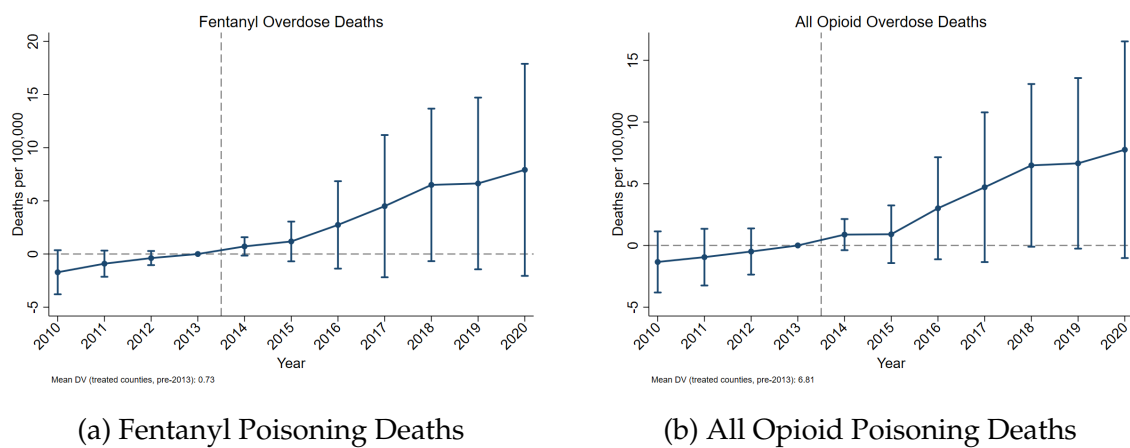
Outcome	Group	Pre-mean (per 100k)	2014+ $\hat{\beta}$ (SE)	% of mean (2014+)
Any Opioid	15-34	6.702	6.529*** (1.725)	97.4%
	35-54	10.358	8.240*** (2.894)	79.6%
	55+	4.088	2.621 (1.947)	64.1%
Fentanyl	15-34	0.616	6.593*** (1.924)	1070.5%
	35-54	1.329	8.090** (3.330)	608.9%
	55+	0.625	2.290 (1.941)	366.3%
Fent. + Heroin	15-34	0.046	1.229** (0.496)	n.m. [†]
	35-54	0.056	2.265*** (0.743)	4022.1%
	55+	0.017	1.112** (0.485)	n.m. [†]
Heroin	15-34	2.933	1.056 (0.751)	36.0%
	35-54	3.059	2.104* (1.221)	68.8%
	55+	0.800	0.942 (0.691)	117.6%

Standard errors in parentheses

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$

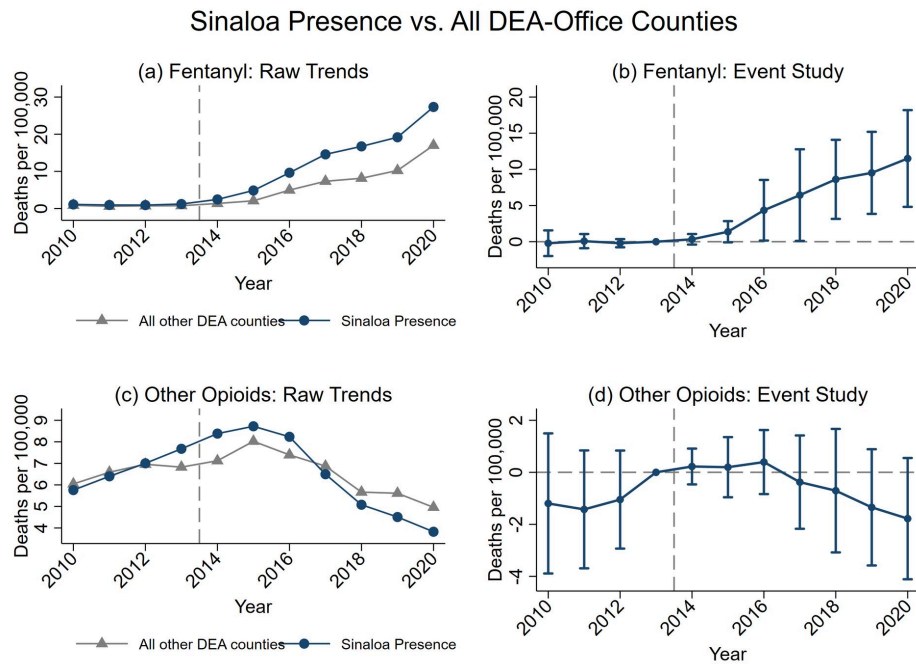
[†] Percent-of-mean not reported (n.m.) for pre-means below 0.05 deaths per 100,000: not an informative magnitude.

Figure A7: Event-Study Estimates Using 2008 Sinaloa Federation Presence



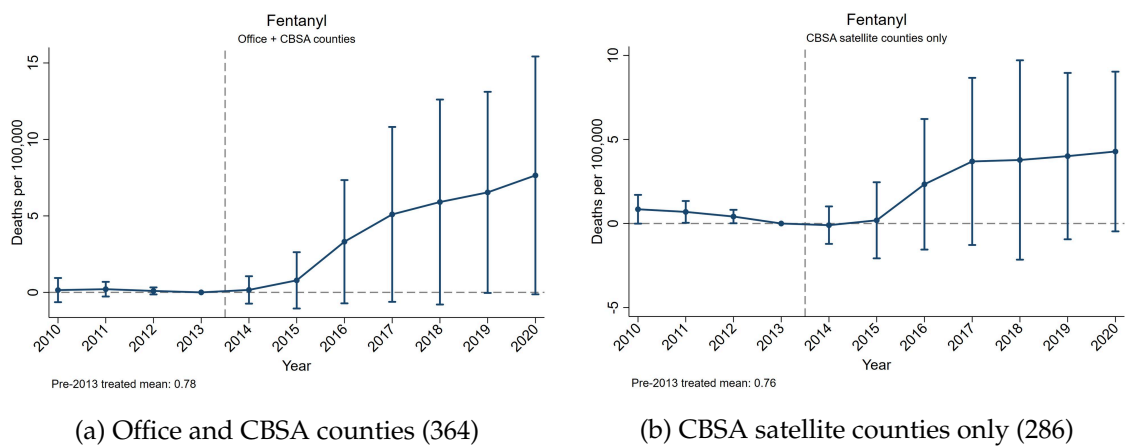
Notes: Event-study estimates of the effect of 2008 Federation (Sinaloa-proxy) presence on poisoning mortality, estimated on the same sample as the main NDTA 2015 analysis. Treatment is assigned to counties containing cities reporting Sinaloa-led Federation presence in the 2008 NCIC map (reporting window January–April 2008). Estimates are relative to 2013, the omitted year; the vertical line marks the 2014 onset of the fentanyl wave. Rates are per 100,000 residents aged 15 and older. Confidence intervals use standard errors clustered at the state level. Source: CDC Multiple Cause of Death files, 2010–2022.

Figure A8: Robustness: Sinaloa Presence vs. All DEA-Office Counties



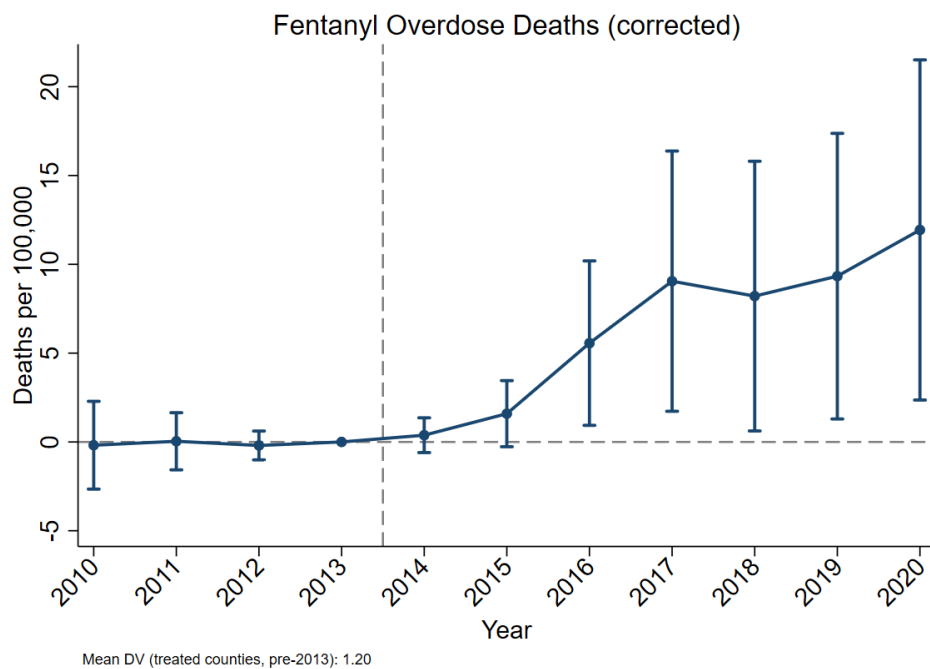
Notes: This figure replicates the main four-panel analysis using an expanded control group. Treatment is defined as counties with Sinaloa dominance at both the county and state level (the main treatment group); the control group consists of all remaining counties covered by DEA field-office jurisdictions, including the 20 within-state spillover counties excluded from the main specification (see Appendix A). Panels (a) and (b) report raw trends and event-study estimates for fentanyl-involved poisoning deaths; Panels (c) and (d) report the same for poisoning deaths involving opioids other than fentanyl. Event-study estimates are relative to 2013, the omitted year, and include county and year fixed effects and demographic covariates. Rates are per 100,000 residents aged 15 and older. Estimates are weighted by county population aged 15 and older; standard errors are clustered at the state level. Because the expanded control group includes counties plausibly exposed to Sinaloa distribution through within-state spillovers, these estimates are conservative relative to the main specification. Source: CDC Multiple Cause of Death files, 2010–2020.

Figure A9: Robustness: Treatment Extended to the Office's Metropolitan Area



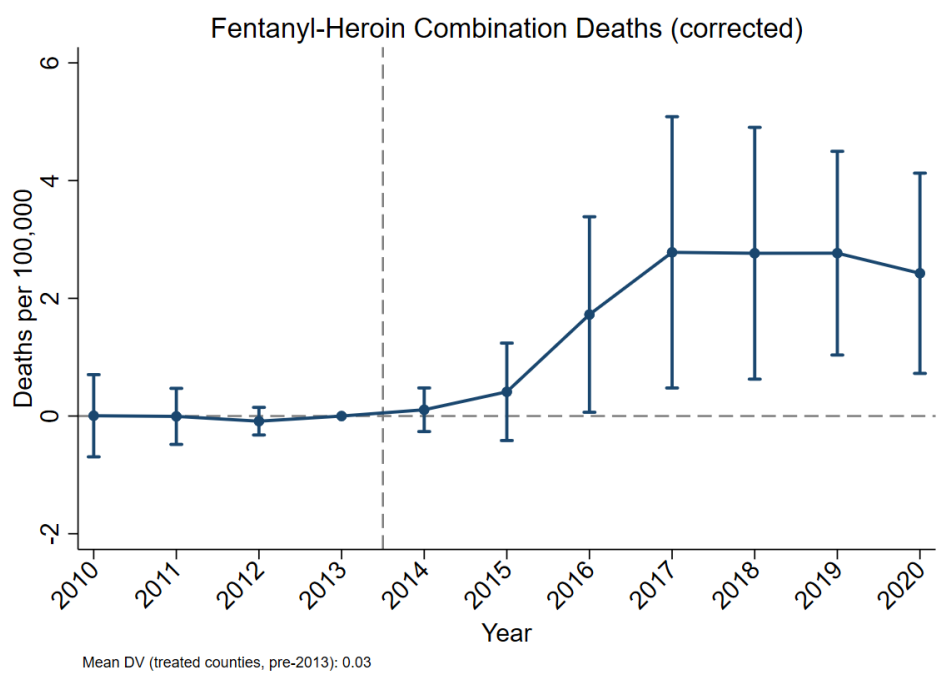
Notes: Event-study estimates of fentanyl-involved poisoning deaths per 100,000 residents aged 15 and older. Each DEA office's treatment is assigned to every county in its 2013 core-based statistical area (Census delineation). Satellite counties are dropped when their CBSA contains offices with different treatment status or an office excluded from the main sample, and when their state's retained offices have a different treatment status (the within-state spillover rule of the main specification). Panel (a) pools the 78 office counties with 286 satellite counties; Panel (b) uses the satellite counties alone. Estimates are relative to 2013 and include county and year fixed effects and demographic covariates. County-years with no recorded deaths are set to zero. Estimates are weighted by county population aged 15 and older; standard errors are clustered at the state level. The corresponding two-period estimates are 3.80 (s.e. 2.03) in Panel (a) and 1.80 (s.e. 1.73) in Panel (b), compared with 5.52 (s.e. 2.28) in the main specification. Source: CDC Multiple Cause of Death files, 2010–2020.

Figure A10: Event Study Estimates on Drake-Ruhm Corrected Outcomes: Fentanyl



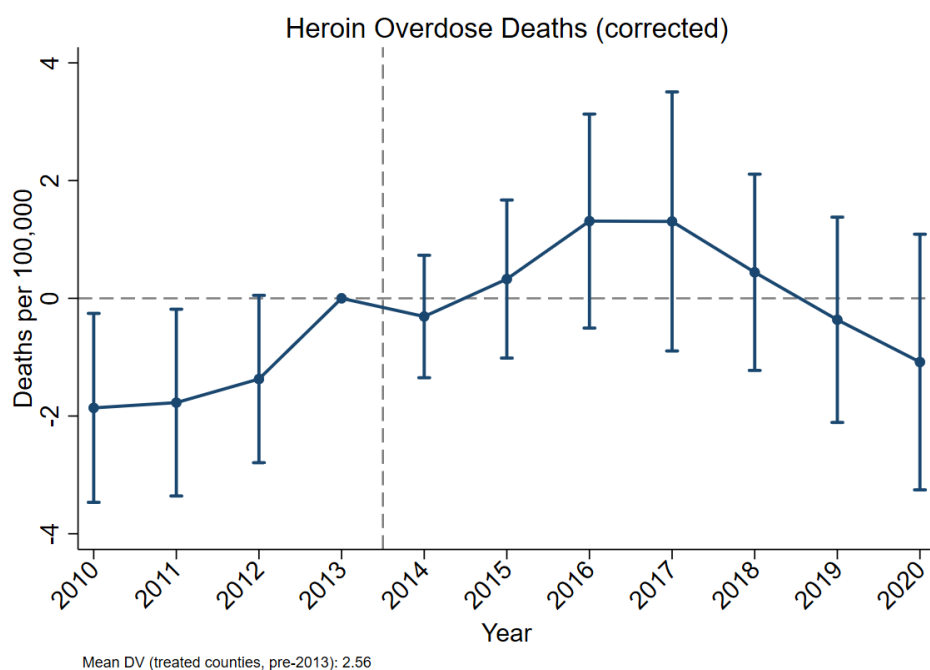
Notes: Event-study estimates from Equation (2) estimated on poisoning outcomes corrected for incomplete drug specification following Drake and Ruhm (2023). Drug-unspecified poisoning deaths are assigned fractional substance involvement via year-by-year probit imputation on decedent characteristics and state fixed effects. Outcomes are rates per 100,000 residents aged 15 and older. Estimates are relative to 2013, the omitted year. Confidence intervals use standard errors clustered at the state level. Source: CDC Multiple Cause of Death, 2010–2022.

Figure A11: Event Study Estimates on Drake-Ruhm Corrected Outcomes: Fentanyl + Heroin



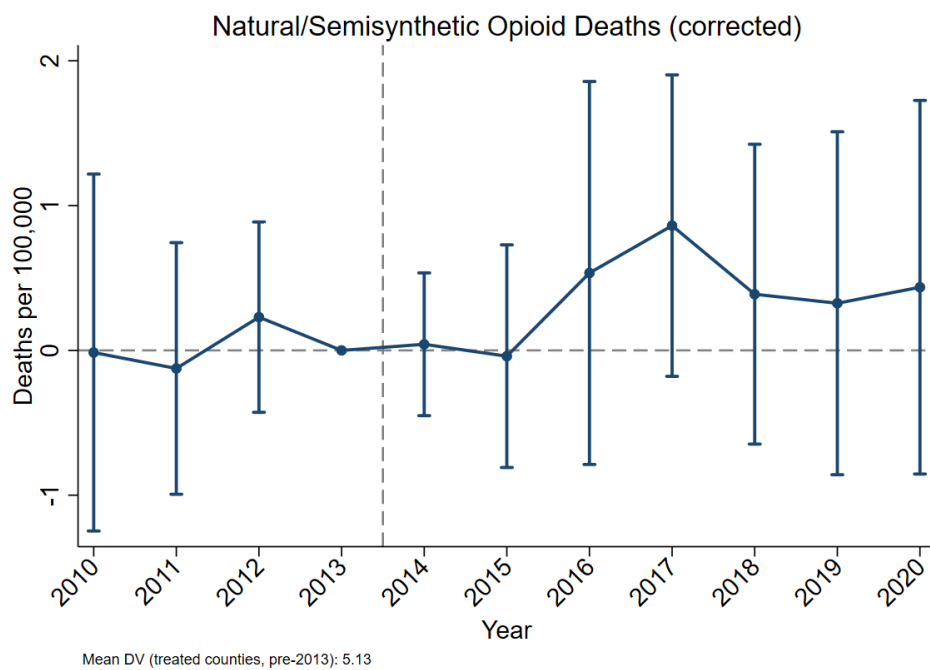
Notes: Event-study estimates from Equation (2) estimated on poisoning outcomes corrected for incomplete drug specification following Drake and Ruhm (2023). Drug-unspecified poisoning deaths are assigned fractional substance involvement via year-by-year probit imputation on decedent characteristics and state fixed effects. Outcomes are rates per 100,000 residents aged 15 and older. Estimates are relative to 2013, the omitted year. Confidence intervals use standard errors clustered at the state level. Source: CDC Multiple Cause of Death, 2010–2022.

Figure A12: Event Study Estimates on Drake-Ruhm Corrected Outcomes: Heroin



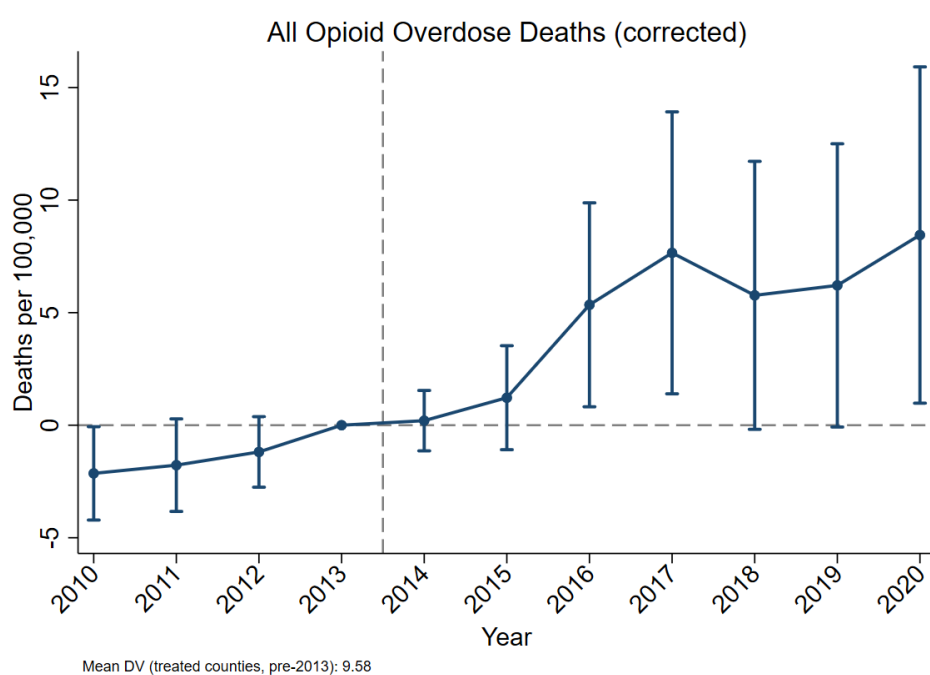
Notes: Event-study estimates from Equation (2), estimated on poisoning outcomes corrected for incomplete drug specification following Drake and Ruhm (2023). Drug-unspecified poisoning deaths are assigned fractional substance involvement via a year-by-year probit imputation on decedent characteristics and state fixed effects; the corrected outcomes are then aggregated to the county-year level and used in Equation (2), which includes county and year fixed effects. Outcomes are rates per 100,000 residents aged 15 and older. Estimates are relative to 2013, the omitted year. Confidence intervals use standard errors clustered at the state level. Source: CDC Multiple Cause of Death, 2010–2022.

Figure A13: Event Study Estimates on Drake-Ruhm Corrected Outcomes: Natural and Semi-Synthetic Opioids



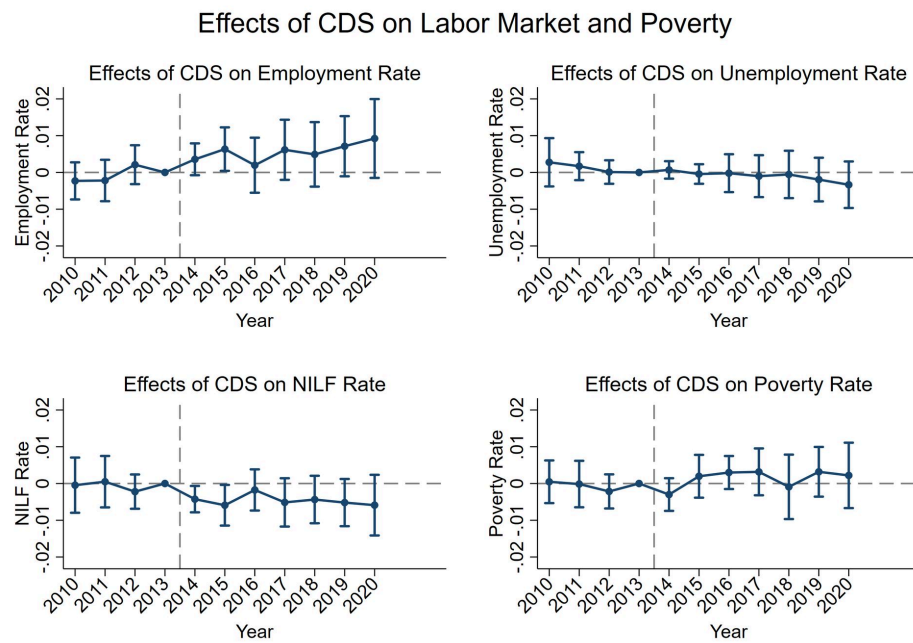
Notes: Event-study estimates from Equation (2), estimated on poisoning outcomes corrected for incomplete drug specification following Drake and Ruhm (2023). Drug-unspecified poisoning deaths are assigned fractional substance involvement via a year-by-year probit imputation on decedent characteristics and state fixed effects; the corrected outcomes are then aggregated to the county-year level and used in Equation (2), which includes county and year fixed effects. Outcomes are rates per 100,000 residents aged 15 and older. Estimates are relative to 2013, the omitted year. Confidence intervals use standard errors clustered at the state level. Source: CDC Multiple Cause of Death, 2010–2022.

Figure A14: Event Study Estimates on Drake-Ruhm Corrected Outcomes: Any Opioid



Notes: Event-study estimates from Equation (2) estimated on poisoning outcomes corrected for incomplete drug specification following Drake and Ruhm (2023). Drug-unspecified poisoning deaths are assigned fractional substance involvement via year-by-year probit imputation on decedent characteristics and state fixed effects. Outcomes are rates per 100,000 residents aged 15 and older. Estimates are relative to 2013, the omitted year. Confidence intervals use standard errors clustered at the state level. Source: CDC Multiple Cause of Death, 2010–2022.

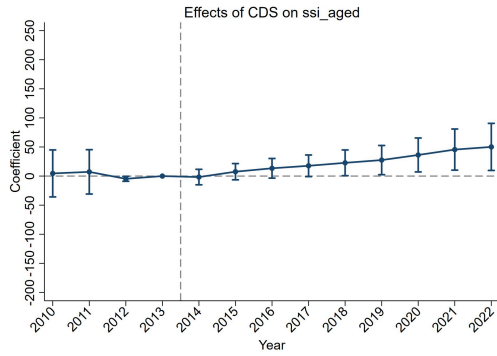
Figure A15: Effects of Sinaloa Presence on Labor Market and Poverty Outcomes



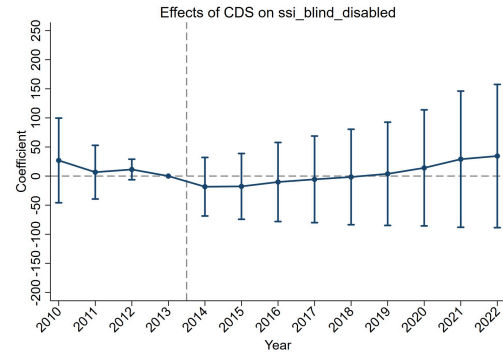
County + year FE. SE clustered at state. Weighted by pop 15+.

Notes: Event-study estimates from Equation (2) for employment, unemployment, labor-force participation, and the poverty rate. Estimates are relative to 2013, the omitted year. Confidence intervals use standard errors clustered at the state level. Source: American Community Survey.

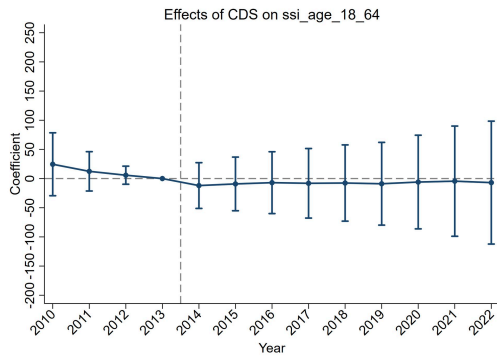
Figure A16: Effects of Sinaloa Cartel Presence on SSI Caseloads



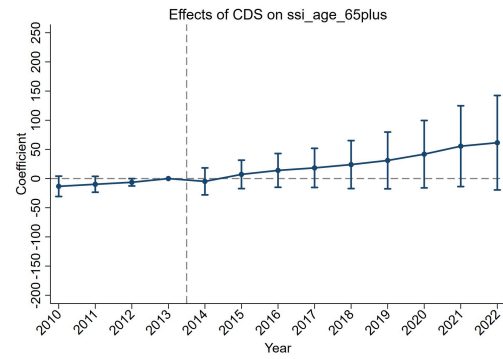
(a) SSI Aged Recipients



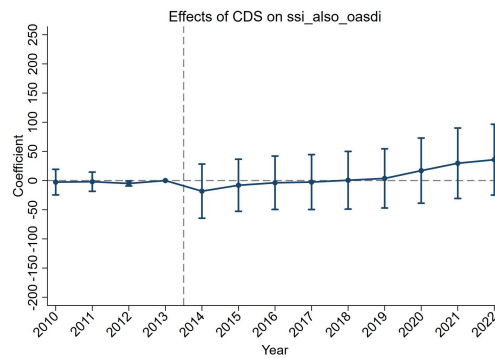
(b) SSI Blind and Disabled Recipients



(c) SSI Recipients Ages 18–64



(d) SSI Recipients Ages 65 and Older



(e) SSI Recipients Also Receiving OASDI

Notes: Each panel reports event-study estimates from Equation (2). The dependent variable is the number of SSI recipients or amount spent per 100,000 population in the indicated category. Estimates are relative to 2013, the omitted year. Shaded bands represent 95% confidence intervals. Standard errors are clustered at the state level. CDS stands for Cartel de Sinaloa, the Spanish name of the Sinaloa Cartel. Source: Social Security Administration.

A. Sample Construction

This appendix provides additional detail on the construction of the main estimation sample. Section A.1 describes how I combine the mortality, population, and cartel-presence data sources. Section A.2 documents the restrictions that take the sample from the set of counties covered by DEA field-office intelligence to the county-year panel used in the main analysis.

A.1. Merge Between Mortality Data and Cartel Presence Measures

I combine three primary data sources at the county-year level. Mortality outcomes come from the CDC Multiple Cause-of-Death (MCOB) Restricted Use “All Counties” files, 2010–2022, described in Section 4.1. From the fixed-width death-certificate records I construct annual county-level counts of overdose deaths, defined as cases in which the underlying cause of death is drug poisoning (ICD–10 codes X40–X44, X60–X64, X85, or Y10–Y14). Substance-specific indicators are built from the multiple-cause fields: “any opioid” requires at least one T40.0–T40.4 code, “fentanyl” corresponds to T40.4, “heroin” to T40.1, “cocaine” to T40.5, and “meth” to T43.6. Because the restricted-use files include county FIPS codes for all decedents regardless of county population, the mortality counts are not subject to the small-cell suppression that affects the public-use WONDER data.

Population denominators come from the Census Bureau county-level intercensal estimates. I merge these to the mortality counts using county FIPS codes and compute mortality rates per 100,000 residents aged 15 and older for each substance category.

Treatment status is assigned from the DEA National Drug Threat Assessment (NDTA) cartel-presence maps, which report cartel dominance at the level of DEA field-office jurisdictions. Each county is mapped to the DEA field office $o(c)$ covering it, and the county-level treatment indicator equals one when the Sinaloa cartel accounts for more than 50% of cartel-attributed cases in that office (Equation (1) in the main text). Treatment is defined from the 2015 NDTA, which reports cartel shares at the field-office level. As validation, I construct an independent text-based measure of Sinaloa presence from the Access World News local newspaper archive, which agrees closely with the NDTA-based indicator (Figure 5).

A.2. Sample Selection

The starting point consists of the 98 counties that map to the DEA field-office jurisdictions documented in the NDTA 2015 assessment, observed over 2010–2022. Appendix Table A7 reports the selection process.

From this set, I exclude 20 counties to mitigate potential spillover effects arising from the geographic contiguity of certain DEA offices: counties with Sinaloa-majority classification located in states otherwise dominated by rival organizations, and rival-majority counties located in states otherwise dominated by Sinaloa (Appendix Figure A5). Because drug distribution networks operate at a regional scale, retaining these counties would risk contaminating the comparison group with Sinaloa supply exposure (and vice versa). The excluded set is composed predominantly of large metropolitan counties, including Los Angeles, San Diego, and Riverside. One limitation of this restriction is that the resulting sample is composed almost entirely of large urban counties.

The classified sample therefore consists of 78 counties—42 treated and 36 control—observed over 2010–2022. The panel is nearly balanced: 75 of the 78 counties are observed in all 13 years, while three small border counties—Santa Cruz County, AZ, and Brewster and Maverick Counties, TX—are observed for 12, 9, and 10 years respectively. Because the panel is constructed from individual death records, county-years in which these counties record no qualifying overdose deaths do not enter the panel, yielding 1,006 county-year observations (545 treated, 461 control) rather than the 1,014 of a fully balanced panel. The eight absent county-years account for less than 0.1% of sample population.

The main estimation sample additionally restricts to 2010–2020 and to observations with non-missing covariates, yielding 850 county-year observations across the same 78 counties. These counties account for 74.5 million residents aged 15 and older in 2013 (40.7 million in treated counties and 33.7 million in control counties), approximately 29% of the national population aged 15 and older.

Table A7: Sample Sizes After Restrictions

	Counties	County-years
Counties mapped to NDTA 2015 DEA field offices	98	1,266
Exclusion of within-state spillover counties	78	1,006
Estimation sample (2010–2020, non-missing covariates)	78	850

Notes: The table reports the number of counties and county-year observations remaining at each step of the sample-restriction process. County-years are those appearing in the death-record-based panel over 2010–2022. The first restriction drops 20 counties to mitigate spillover effects: Sinaloa-majority counties located in states otherwise dominated by rival organizations, and rival-majority counties located in states otherwise dominated by Sinaloa. The classified panel is unbalanced because three small border counties record no qualifying overdose deaths in a total of eight county-years. The estimation sample restricts to 2010–2020 and drops county-years with missing covariate data.

Table A8: Counties by Treatment Status

	Control	Treated	Total
Number of counties	36	42	78
County-year observations	389	461	850
Population 15+, 2013 (millions)	33.7	40.7	74.5
Share large urban county	0.97	0.98	0.97

Notes: This table reports the distribution of counties in the main estimation sample by treatment status. A county is treated if the Sinaloa cartel accounts for more than 50% of cartel-attributed cases in the DEA field office covering it, based on the NDTA 2015 assessment. County-year observations refer to the 2010–2020 estimation sample. Population is the county population aged 15 and older in 2013. Large urban county is an indicator for counties classified as large metropolitan.