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ABSTRACT

Colombian coca cultivation fell dramatically between 2000 and 2015, a period that saw intense U.S.-backed eradication and interdiction efforts. That progress reversed in 2015, when peace talks and legal rulings in Colombia opened enforcement gaps. Coca cultivation has since increased to record levels, while cocaine-related overdose deaths in the United States also rose sharply. We estimate how much of that rise can be causally attributed to Colombia's new coca boom. Leveraging the unforeseen coca supply shock and cross-county differences in pre-shock cocaine exposure, we find that the surge in supply caused an immediate rise in overdose mortality in the U.S. The implied annual loss in U.S. statistical-life value is about $45,000 per hectare of cultivation in Colombia. We estimate that on the order of 1,000–1,500 additional U.S. deaths per year can be attributed to Colombia's coca boom. At Colombia's 2022 cultivation level (over 230,000 hectares), the cocaine supply shock may impose on the order of $10 billion per year in U.S. costs via overdose fatalities.

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1. Introduction

Illicit drug markets are transnational in scope. Shifts in supply in one part of the world can have profound consequences for public health and safety in consuming regions elsewhere (Dell, 2015; Csete et al., 2016; Castillo, Mejía, and Restrepo, 2020; Moore, Olney, and Hansen, 2023; Donahoe and Soliman, 2025). In the 2000s, with heavy U.S. support, Colombia waged an aggressive campaign against coca cultivation, the raw plant used to make cocaine. Initiatives like Plan Colombia combined aerial herbicide spraying with manual crop eradication to contain cocaine supply at its source. Colombia's coca fields shrank from about 168,000 hectares in 2000 to just 48,000 by 2013, and cocaine became much less available in the United States (Office of National Drug Control Policy, 2012).

However, this progress reversed in the mid-2010s as enforcement efforts weakened. Two major policy changes in 2015–2016 contributed to a rapid resurgence in coca cultivation. First, in May 2015 the Colombian government halted its U.S.-backed aerial fumigation program for coca crops after health authorities warned that the spray chemical glyphosate was likely carcinogenic. Second, Colombia's government signed a landmark peace accord in late 2016 with the Revolutionary Armed Forces of Colombia (FARC) guerrilla group, ending a 52-year civil conflict. While peace was welcome, it had unintended consequences for the drug trade. For years, the FARC had tightly controlled and taxed coca production in the areas it dominated. When the rebels demobilized, a power vacuum formed in remote coca-growing regions. A variety of other armed groups, including dissident FARC factions, the ELN, and Mexican trafficking organizations, expanded into these territories. DEA reported that some FARC elements encouraged coca growers to expand planting (Drug Enforcement Administration, 2017; United Nations Peacekeeping, 2018). At the same time, the peace deal introduced a coca crop substitution program that promised stipends and development aid to farmers who eradicated their coca. This well-intentioned plan backfired. Farmers quickly realized they needed to have coca plants in the ground to qualify for compensation, which led many to start new coca plots or expand existing ones in hopes of securing the promised subsidies (Gelvez and Serrano, 2023; Prem, Vargas, and Mejía, 2023).

By 2022, Colombia's coca cultivation area and potential cocaine output were more than three times their 2015 levels (Figure 1, panel A).[1] DEA assessments indicated that in 2016–2017 cocaine availability and purity were increasing in multiple U.S. markets (Drug Enforcement Administration, 2017). Consistent with those reports, Drug Enforcement Administration (DEA) data show that after 2015 the average size of cocaine seizures jumped markedly, while seizures of other drugs did not follow the same pattern (Figure 1, panel B).

The surge in supply was accompanied by an alarming increase in U.S. drug deaths. After remaining stable from 2009 through 2013, the rate of drug overdose deaths involving cocaine nearly tripled by 2018 (Jalal et al., 2018). Policymakers attributed the increase in overdose deaths to Colombia's coca expansion. As one U.S. State Department official warned: "Between 2013 and 2016, coca cultivation in Colombia increased by more than 130 percent and cocaine production by more than 200 percent. The explosion in Colombian cocaine is driving an increase in cocaine use and overdose deaths in the United States and threatening Colombia's internal security and successful implementation of its peace accord" (Glenn, 2018).

Of course, simply observing that Colombian coca surged and U.S. overdoses spiked does not prove causality. Overdose trends are influenced by many factors, ranging from the spread of fentanyl to broader socioeconomic shifts, so rigorous analysis is needed to isolate the impact of Colombia's supply boom from other developments. To our knowledge, no prior study has quantitatively estimated how much of the recent U.S. drug overdose crisis can be attributed to Colombia's cocaine resurgence. In this paper,

[1] Potential production refers to the amount of cocaine that would be produced if all the leaves harvested from the area under coca cultivation in one year would be converted into 100% pure cocaine hydrochloride (United Nations Office on Drugs and Crime, 2010).

we provide the first causal evidence on this link by exploiting the natural experiment created by Colombia's 2015–2016 policy pivot.

Our research design leverages an exogenous increase in Colombia's aggregate coca production and differences in local exposure to Colombian cocaine across U.S. counties. Before the shock, some U.S. counties were major transshipment hubs or had high cocaine consumption, while others saw minimal cocaine traffic. When the Colombian coca surge hit, areas more entwined with the cocaine trade experienced a larger increase in supply than less-exposed areas. We combine detailed data on Colombia's coca production with U.S. county-level overdose death records and compare changes in overdose rates in high-exposure versus low-exposure counties after 2015. This approach allows us to isolate the effect of the Colombian supply shock on U.S. overdose mortality, under the assumption (which we confirm in tests) that absent the shock those counties would have followed similar overdose trends.

Our results show a clear pattern: starting in 2016, overdose death rates rose significantly faster in the U.S. counties most exposed to Colombian cocaine than in otherwise similar counties with little exposure. This divergence was not apparent before 2015, which strengthens the case that the Colombian supply shock, rather than any pre-existing local trend, was responsible for the change. By 2017–2018, the hardest-hit areas were suffering dozens of extra overdose deaths per million residents each year relative to low-exposure areas. Aggregating these local impacts, we estimate that Colombia's coca boom caused on the order of 1,500 additional U.S. drug overdose deaths per year in the late 2010s and early 2020s. For context, U.S. cocaine overdose fatalities surpassed 20,000 per year in the early 2020s, so a significant share of the recent surge in cocaine deaths can be linked to Colombia's policy reversal.

We conduct a variety of robustness and falsification checks. The result holds under alternative definitions of the coca boom (using different measures of cultivation and production, or simply a post-2015 indicator) and is consistent when we use different data sources to measure pre-shock drug exposure. We find no comparable increase in overdose deaths involving drugs other than cocaine, i.e., the effect is specific to cocaine's supply surge. We also run falsification tests by applying our methodology to "placebo" shocks (for example, pretending that another drug or an unrelated factor experienced a similar supply jump). These placebo tests produce no effect, which supports that the coca surge is behind the observed mortality increase.

Our empirical findings carry several policy lessons. First, they highlight how lapses in international drug enforcement can produce health spillovers. Colombia's halt to aerial eradication and the peace accord's unintended consequences created a health externality abroad. Even well-intended domestic policies like ending a civil war can generate serious cross-border repercussions when they undermine drug supply control. This episode shows that a breakdown in supply-side enforcement abroad can swiftly undercut public health progress at home.

Second, our results suggest that U.S. investments in overseas counter-narcotics efforts may have delivered large domestic benefits. If American counter-narcotics support helped drive Colombia's coca decline in the 2000s, our estimates imply that this aid prevented many American overdose deaths. For example, since the launch of Plan Colombia in 1999, the U.S. government has invested over $10 billion in counternarcotics efforts in Colombia (U.S. Government Accountability Office, 2018). Now that those gains have been reversed, the reversal is associated with substantial costs to the United States. We estimate that Colombia's current coca boom is inflicting on the order of $10 billion per year in damages to the U.S. in the form of overdose fatalities. In concrete terms, each additional hectare of coca cultivated in Colombia ultimately causes roughly $45,000 per year in harm to the United States when valuing the statistical lives lost to drug overdose. By this empirical benchmark, our estimates suggest that programs reducing coca cultivation could generate substantial health benefits.

Third, we find that a major surge in drug supply can drive up overdose deaths even without any increase in domestic demand, and that U.S. demand-side measures did not appear to mitigate the mortality impact of the supply increase. In our data, the effect of Colombia's supply shock on U.S. mortality was rapid: overdose death rates in highly exposed areas began climbing within about a year of the coca surge. Domestic drug treatment, education, and enforcement efforts did not significantly blunt this rise. This implies that when supply explodes, it can overwhelm even a stable demand base.

Related Literature. We are most directly related to the literature on the economics of cocaine markets. Prior research has analyzed the effects of Plan Colombia drug eradication policies both on production (Mejía and Posada, 2008; Camacho and Mejía, 2017; Mejía, Restrepo, and Rozo, 2017; Gelvez and Serrano, 2023; Prem, Vargas, and Mejía, 2023) and the downstream effects on user markets (Mejía and Restrepo, 2016). We are most related to Mejía and Restrepo (2016), who combine predictions from a calibrated model with time-series evidence to show that reductions in supply under Plan Colombia had limited effects on cocaine prices and retail availability. To our knowledge, we are not aware of prior work that directly estimates the downstream health consequences of cocaine using a quasi-experimental framework, and we find these health effects to be substantial.

It is important to note that our exercise focuses exclusively on measuring health spillovers to the U.S. In concurrent work, Daniele, Soliman, and Vargas (2026) exploit the same post-2015 Colombian supply expansion and trace its propagation through global maritime trade networks. They show that violence increased sharply at logistical chokepoints along the cocaine supply chain and spilled into Ecuador, where homicide rates rose nearly fivefold once centralized criminal control fragmented. Our analysis complements theirs by focusing on a different downstream margin (overdose mortality in the United States) and by estimating the per-hectare welfare cost of coca cultivation.

Increased drug supply can also generate broader local effects. For example, both the expansion and suppression of drug markets have been shown to impose substantial costs on local communities through increased violence (Angrist and Kugler, 2008; Mejía and Restrepo, 2013; Dube and Naidu, 2015; Dube, García-Ponce, and Thom, 2016), natural resource depletion (Negret et al., 2019; Dávalos et al., 2011), and weakened state capacity (Acemoglu, Robinson, and Santos, 2013).

Our work also adds to the understanding of several features of illicit trade markets. First, measurement of illicit trade is difficult by nature, and thus the study of such markets is intrinsically challenging (Fisman and Wei, 2009; Moore, Olney, and Hansen, 2023). Our framework instead exploits how a common shock lands differentially across counties with varying degrees of prior exposure to the drug. Although we cannot measure cocaine use directly, our strategy does produce estimates of the overdose fatality cost per hectare of cultivation at the source, which is the policy-relevant variable. Second, drug production and trade adapt strongly to enforcement changes and often have transboundary spillover effects (Miron and Zwiebel, 1995; Dobkin and Nicosia, 2009; Dell, 2015; Galenianos and Gavazza, 2017; Chimeli and Soares, 2017; Bellégo and Drouard, 2024; Donahoe and Soliman, 2025). Colombia's post-2015 experience provides another example of a rapid resurgence of a globally traded drug as enforcement gaps emerge. Finally, drug diffusion has profound health ramifications, although in the prior literature, much more evidence is available on the opioid crisis (e.g., Alpert, Powell, and Pacula, 2018; Evans, Lieber, and Power, 2019; Balestra et al., 2021; Alpert et al., 2022; Cornaggia et al., 2022; Eichmeyer and Zhang, 2022; Evans, Harris, and Kessler, 2022) and less so on other imported drugs such as cocaine (Evans, Garthwaite, and Moore, 2016)

Before proceeding, it is worthwhile to highlight scholarly work that offers alternative perspectives on *Plan Colombia* and on supply-side drug regulation more generally, and to clarify how our conclusions would be affected if those perspectives are correct. First, in our policy discussion we implicitly treat *Plan Colombia* as a major contributor to Colombia's coca decline between 2000 and 2013. This interpretation is motivated by the close alignment between the timing of the decline and the period of intensive U.S.-backed eradication, and by the fact that most large-scale crop suppression efforts during this period were supported by U.S. funding and technical assistance under *Plan Colombia*. That said, the academic literature on the effectiveness and efficiency of *Plan*

Colombia is considerably more nuanced. For example, Mejía (2016) documents diminishing effectiveness of eradication efforts over time, and some studies argue that *Plan Colombia* may have been largely ineffective in reducing aggregate coca cultivation (Moreno-Sanchez, Kraybill, and Thompson, 2003; Rincón-Ruiz et al., 2016). If U.S. interventions were not in fact the primary driver of the coca decline between 2000 and 2013, this would not affect our econometric analysis of how the post-2015 coca boom translated into health costs in the United States. It would, however, limit our ability to interpret earlier supply suppression efforts under *Plan Colombia* as having saved lives.

Second, prior work argues that historical supply-side interventions can be inefficient and costly. For example, Mejía and Restrepo (2008) show that reductions in cultivated area need not translate proportionally or contemporaneously into reductions in cocaine production. Related work highlights displacement or "balloon" effects, whereby eradication shifts cultivation and trafficking across regions rather than reducing overall supply (Mejía, 2016; Mejía and Restrepo, 2008). Other studies document additional externalities of eradication policies, including adverse health impacts from aerial spraying and forced displacement of rural households (Dion and Russler, 2008; Camacho and Mejía, 2017). Mejía (2016) further argues that demand-side policies such as interdiction may be more cost-effective by avoiding supply-side adaptation and unintended consequences. Our findings show that large positive supply shocks generate substantial health externalities in consumer markets. Although it follows logically that reductions in cocaine supply could reduce these harms, our analysis does not speak to the relative effectiveness of alternative policy instruments. Nor do we claim that a return to *Plan Colombia*–style interventions would necessarily be the most efficient or welfare-maximizing approach.

Section 2 continues with institutional background and data description. Section 3 presents our main empirical analysis. Section 4 discusses policy implications and then concludes the paper.

2. Background and Data

2.1 Background

History of Colombian coca cultivation, conflict, and U.S. involvement. Colombia's coca cultivation has long been entwined with its internal conflict. Starting in the 1980s, cocaine cartels and guerrilla groups like the Revolutionary Armed Forces of Colombia (FARC) built their operations around the coca economy. FARC rebels taxed coca-growing farmers in areas under their control and played a major role in trafficking. For decades, cocaine profits financed armed groups on both sides of Colombia's civil conflict: leftist guerrillas and right-wing paramilitaries alike financed their fighting through the drug trade. In response, the United States stepped in as a key partner in Colombia's counter-narcotics efforts. The most prominent initiative was Plan Colombia, launched in 2000 and backed by over $10 billion in U.S. aid. Plan Colombia combined military operations with aggressive coca eradication campaigns ranging from aerial herbicide spraying to manual crop destruction on the ground in an effort to weaken insurgents and curtail cocaine production. Colombia's coca cultivation area fell from roughly 168,000 hectares in 2000 to about 48,000 hectares by 2013, which was a historic low.

The reversal of coca's decline. These gains reversed beginning in 2015 due to a convergence of policy shifts and peace developments. In May 2015, the Colombian government suspended its U.S.-backed aerial fumigation program for coca fields. Aerial spraying of glyphosate herbicide over coca fields had been a central counter-narcotics tactic for over two decades. By the mid-2010s, however, mounting health and legal concerns put this practice under scrutiny, and the International Agency for Research on Cancer had classified glyphosate as "probably carcinogenic to humans." In response, President Santos suspended the aerial fumigation program in May 2015, removing a key constraint on large-scale coca planting. The United Nations recorded a 39% surge in Colombia's coca cultivation in 2015 compared to the previous year (UNODC, 2016). The government vowed to replace aerial spraying with manual eradication brigades, but those on-the-ground efforts proved too limited to stem the rebound.

A second upheaval followed with Colombia's historic peace accord with the FARC in late 2016, which formally ended a 52-year insurgency. Roughly 14,000 former FARC combatants entered the reintegration process after the peace accord (United Nations Peacekeeping, 2018). The peace deal was rightly celebrated for reducing violence: conflict-related killings plummeted in many areas after FARC units laid down their arms. However, the transition also upended the established order in Colombia's remote coca-growing zones, leading to unintended consequences for the drug trade. For years, the FARC had imposed its own kind of control in these rural territories: it decided who could grow coca and how much, taxed the trade, and kept out rival traffickers. When the FARC withdrew, a power vacuum opened across vast swaths of the countryside. Other actors quickly moved in. FARC dissidents who rejected the peace, the ELN guerrilla group, former paramilitary crime networks (i.e., the BACRIM), and even Mexican cartels rushed into the void and seized control of coca fields and trafficking routes. Coca cultivation expanded rapidly in areas formerly managed by the FARC. In the absence of both FARC control and effective state presence, many farmers expanded coca cultivation, often under pressure from newly arrived armed groups.

The peace accord also reshaped the government's approach to coca growers. As part of the accord's rural reforms, the Colombian government agreed to treat coca cultivation more as a livelihood issue than a crime. It launched a National Program for the Substitution of Illicit Crops (PNIS), promising tens of thousands of farming families cash stipends and development assistance if they voluntarily uprooted their coca bushes. However, this well-intentioned program created perverse incentives: only those cultivating illicit crops stood to benefit from the subsidies. Anticipating compensation, many farmers began planting new coca or expanding their plots in 2016–2017 to qualify for the payouts (Prem, Vargas, and Mejía, 2023; Gelvez and Serrano, 2023). Subsequent studies confirmed this response. By 2017 Colombia's coca crop surged to over 171,000 hectares, which at that time was a record high (UNODC, 2018).

Coca's resurgence. By 2017–2018, these factors jointly drove a sustained expansion in coca cultivation. The end of aerial spraying, the post-conflict power vacuum, and the new subsidy incentives collectively propelled Colombia into a coca boom. This trend continued into the 2020s. After a brief plateau in 2018–2019, coca cultivation reached new historic highs during the COVID-19 pandemic and its aftermath. A UNODC survey in 2022 found Colombia's coca fields spanned an unprecedented 230,000 hectares, the largest area ever recorded. Given global coca cultivation of 355,000 hectares in 2022, Colombia's 230,000 hectares represented roughly 65 percent of world coca cultivation in that year (UNODC and Government of Colombia, 2022). Cocaine production soared as well: the UN estimated Colombia's cocaine output at 1,738 tons in 2022, another all-time high. Cultivation remains heavily concentrated in a few traditional hotspots. The border region of Catatumbo in Norte de Santander and the southern departments of Nariño and Putumayo together account for about 65% of the country's coca acreage (UNODC and Government of Colombia, 2022).

Policy debates. The post-2015 coca expansion prompted divergent policy responses. The Santos administration (2010–2018) emphasized voluntary substitution and rural development over forced eradication, consistent with the peace accord's framing of coca cultivation as a livelihood issue. The U.S. government, by contrast, pressed for more aggressive enforcement; in 2017, the Trump administration threatened to decertify Colombia as a counter-narcotics partner, citing the suspension of aerial spraying and the sharp rise in cultivation. President Duque (2018–2022) shifted toward intensified manual eradication, yet cultivation remained near historic highs throughout his term, as replanting by armed groups offset eradication gains. Most recently, the Petro administration (2022–present) has moved away from forced eradication of smallholder plots, instead prioritizing interdiction of trafficking networks and voluntary crop substitution backed by rural development investment. Throughout these shifts, coca cultivation has continued to expand, underscoring the difficulty of reversing supply-side trends once enforcement gaps have opened. The persistence of high cultivation levels across administrations with markedly different strategies provides additional context for interpreting our empirical results, which capture the downstream health costs of the initial supply shock rather than the effectiveness of any particular policy response.

2.2 Data

Colombia coca cultivation. We obtain data on coca cultivation in Colombia from the United Nations Office on Drugs and Crime (UNODC), which compiles annual metrics on illicit coca crop production. These data are based on surveys and official reports from coca-growing countries, which provides estimates of the area under coca cultivation (in hectares) for 1995–2022. In addition, we draw on data from the White House Office of National Drug Control Policy (ONDCP), which reports potential cocaine production (in metric tons) for 2010-2021. Using these sources, we construct annual series of the total cultivated coca area and estimated cocaine production in Colombia.

U.S. state-level drug use. To measure drug consumption in the United States, we use data from the National Survey on Drug Use and Health (NSDUH), an annual nationally representative household survey of substance use prevalence and incidence. We extract state-by-year measures of cocaine use – specifically, the share of the population reporting cocaine use (or initiation) in the past year – as well as analogous measures for other substances (e.g., alcohol and tobacco) for context.

We supplement NSDUH data with the National Forensic Laboratory Information System (NFLIS), a DEA-managed database that aggregates results from forensic drug laboratories nationwide. This administrative system records drug reports – instances where a seized substance is chemically identified – from state and local crime labs across the country. We use NFLIS data to obtain the number of cocaine drug reports in each state by year.

U.S. state-level drug seizures. To complement drug use data with more detail on drug supply characteristics, we use the DEA's Laboratory Information Management System (LIMS). LIMS is an internal database of forensic analyses conducted by DEA labs, recording attributes of drug evidence such as weight, purity, and estimated price of samples tested. These data are available by drug type, reported at the state-month level, which we aggregate to annual levels for analysis. From LIMS, we construct state-year indicators of cocaine supply quality – for example, the average net weight of cocaine seized in a state, the purity of cocaine samples, and price-related metrics. Together, these variables provide insight into the nature of cocaine being trafficked (e.g., typical shipment sizes and purity) and help us detect changes in the cocaine market following changes in coca production abroad.

U.S. county-level drug-related crime. Data on drug-related crime in the U.S. come from the FBI's National Incident-Based Reporting System (NIBRS), an administrative dataset of detailed police incident reports. NIBRS records each reported crime incident along with attributes such as the type of offense and any property or contraband seized. We focus on drug possession and trafficking offenses and specifically identify incidents where cocaine was reported as a seized substance. Using the incident-level microdata, we aggregate counts of cocaine-related offenses at the county-year level during 2010–2014. In particular, we construct variables for the number of drug offense incidents per county and the subset of those involving cocaine, which serve as indicators of local cocaine involvement in crime. These NIBRS-derived measures help characterize the distribution of cocaine demand and enforcement encounters within states, and they feed into our county-level exposure index (described below) that links local areas to the Colombian cocaine supply.

U.S. county-level demographics. We incorporate demographic controls and community characteristics from the U.S. Census Bureau's American Community Survey (ACS). For each county and year, we extract total population figures (used in rate calculations and as a size control) and information on residents' country of birth. In particular, we use the ACS to determine the number of county residents who were born in Colombia, from which we compute the Colombian-born population share in the county from 2010-2014. This variable serves as a proxy for local ties to Colombian networks (for example, through immigration or trade links).

U.S. county-level overdose deaths. We use mortality data from the National Vital Statistics System (NVSS) from 2001-2020, which compiles official death records for the entire United States. NVSS mortality microdata include every death certificate, and we observe the date and county location of death, decedent characteristics, and the cause(s) of death as certified by medical authorities.

Our primary outcome is the *cocaine-related* overdose death rate per 100,000 residents in each county-year. We classify a death as cocaine-related if the underlying cause of death falls into any of the following ICD-10 categories: F14.0–F14.9 (mental and behavioral disorders due to cocaine use), T40.5 (poisoning by cocaine), R78.2 (cocaine found in blood), X42 and X62 (accidental or intentional self-poisoning by narcotics and psychodysleptics), and Y12 (poisoning by narcotics and psychodysleptics of undetermined intent). We then compute the county-year mortality rate as the number of cocaine-related overdose deaths divided by the county's population in that year.

Death certificates do not always specify the substances involved in an overdose, so our definition of cocaine-related overdoses may contain errors (CDC, n.d.). As a robustness exercise, we use the subset of deaths that do report specific drugs in the multiple–cause-of-death (MCOD) section, where substances are coded using ICD-10 T-codes (e.g., T40.5 for cocaine; T40.1–T40.4 for heroin, natural opioids, methadone, and synthetic opioids such as fentanyl). Using these MCOD indicators, we construct cocaine-specific (and other drug-specific) overdose mortality rates for robustness checks.

U.S. county-level drug control measures. We count the number of substance-abuse treatment facilities at the county level using data from the U.S. Census Bureau's County Business Patterns (CBP). The CBP reports the annual number of substance-abuse treatment clinics by county for both outpatient and residential facilities. For our analysis, we extract county-level facility counts for 2001–2020.

We also use data from the High Intensity Drug Trafficking Areas (HIDTA) program, administered by the Office of National Drug Control Policy (ONDCP), which designates U.S. counties with high levels of drug trafficking and related criminal activity. HIDTA identifies counties that function as centers of illegal drug production, manufacturing, importation, or distribution. We use these data as a geographic indicator of enforcement intensity and trafficking risk.

3. Evidence

3.1 Empirical Framework

We use the surge in Colombian coca cultivation after 2015 as an exogenous shock to cocaine supply. This coca boom was driven by policy changes and conflict dynamics within Colombia and was largely unrelated to local conditions in the United States. Few, if any, U.S. policymakers or communities could have anticipated or influenced Colombia's decision to halt aerial coca eradication or to sign the 2016 peace accord with FARC. Consequently, Colombia's supply shock is plausibly independent of U.S. county-level trends, except through the channel of increased cocaine availability. Our aim is to isolate the causal impact of this foreign supply shock on U.S. drug overdose mortality.

We implement a difference-in-differences research design to link the Colombian shock to U.S. outcomes. In this framework, the "first difference" is the sharp post-2015 increase in Colombia's coca production, which represents a large overall rise in cocaine supply. The "second difference" is each U.S. county's baseline exposure to the cocaine market, which determines how strongly the supply shock "lands" in that county. We construct a predicted shock variable by multiplying these two components: the total coca cultivated in Colombia in a given year and each county's pre-2015 cocaine exposure share.

This predicted shock varies by year and by county. We then estimate its effect on county-level overdose mortality by regressing the cocaine-related overdose death rate on the predicted shock, controlling for other factors.

Specifically, we construct a predicted shock variable as the product of these two components: Colombia's coca cultivation in year t × county i's baseline 2010-2014 exposure share. This measure varies over time and across counties. We then regress county-level drug mortality rates on this predicted shock measure, controlling for other factors, to estimate the causal effect. Formally, we estimate the following regression model:

$$\text{Cocaine overdose mortality}_{it} = \beta \cdot \text{Coca}_t \times \text{Exposure}_i + \alpha_i + \alpha_t + \varepsilon_{it} \quad (1)$$

and we specify our cocaine exposure measure to be:[2]

$$\text{Exposure}_i = \underbrace{\underbrace{\overbrace{\frac{\text{Cocaine use}_s}{\text{Population}_s}}^{\text{State cocaine use prevalance}} \times \overbrace{\frac{\text{DrugCrime}_i}{\text{DrugCrime}_s}}^{\text{County drug crime prevalance}}}_{\text{Proxy for county cocaine use prevalance}} \times \overbrace{\frac{\text{ColombianPopulation}_i}{\text{Population}_i}}^{\text{County Colombian connection}}}_{\text{Proxy for county cocaine use attributed to Colombian imports}}$$

The dependent variable in the regression is the cocaine-related overdose mortality rate as defined in Section 2.2. The regression specification includes county fixed effects α_i to account for any time-invariant differences between counties, such as poverty or baseline drug environment, and year fixed effects α_t to account for nationwide trends affecting all counties, such as the overall opioid epidemic or economic changes. Regressions are weighted using county-year-level population. Standard errors are clustered at the county level.

Our key independent variable is the interaction of Colombia's coca supply and the county's baseline cocaine exposure. To measure a county's exposure share, we start with state-level cocaine use prevalence (the share of the population using cocaine) and

[2] In our main analyses, we use a multiplicative specification for the exposure measure. In appendix, we show that our findings are robust to using an additive specification instead.

apportion it to counties based on local drug activity. In practice, because cocaine use data are only available at the state level, we multiply each state's cocaine use rate by each county's share of drug-related crimes in that state to proxy the county-level cocaine prevalence. We consider several alternative exposure measures for robustness. One variant uses self-reported cocaine use rates from the National Survey on Drug Use and Health (NSDUH) for 2010–2014, with state-level rates apportioned to counties via local drug-related crime shares. Another uses data from the National Forensic Laboratory Information System (NFLIS), specifically the fraction of drug samples from each county that tested positive for cocaine before 2015. Yet another measure draws on the National Incident-Based Reporting System (NIBRS), using the rate of cocaine-related offenses in the county. All of these serve as proxies for how heavily the county was involved in cocaine markets prior to the shock. Appendix Figure 1 maps the geographic distribution of these exposure components and the resulting county-level exposure index.

Importantly, we verify that our results are not driven by spurious correlations by constructing placebo exposure measures. For example, we replace the cocaine prevalence data with the prevalence of other drugs, or we replace the Colombian-born population share with the non-Colombian-born population share in the exposure measure. Reassuringly, when we use these "fake" exposure variables, we find no effect of the coca shock on overdose deaths. In other words, counties do not experience a mortality increase when they are defined as "exposed" based on unrelated characteristics (such as high usage of other drugs or a large immigrant population). This supports the interpretation that our exposure measure is capturing something specific about cocaine.

Our baseline exposure index uses the county-level Colombian population share to capture local exposure to the Colombian-cocaine distribution network. As a robustness check, we replace this share with an independent proxy based on Colombian perishable imports—flowers (HS 06) and fruit (HS 08), the two largest categories of Colombian non-cocaine exports. The motivation is that perishables shipments are a well-documented vehicle for cocaine smuggling: traffickers exploit the rapid clearance and cold-chain infrastructure of perishable cargo to conceal narcotics inside fresh fruit and flower shipments routed through Atlantic and Gulf ports (Daniele, Soliman, and Vargas, 2026). Counties downstream of customs districts handling large volumes of Colombian perishables therefore plausibly receive larger inflows of Colombian cocaine through the same logistics network. To operationalize this proxy, for each U.S. customs district we compute the 2010–2014 share of Colombian perishable imports in total imports (the Miami district leads at 1.3%, followed by Philadelphia at 0.3%); we then assign each county a perishables exposure equal to a gravity-weighted average of district shares using a 500 km bandwidth. We re-estimate our main specification using this perishables proxy in place of the Colombian population share. Construction details, including the gravity-decay specification and a sensitivity analysis on the bandwidth, are provided in Appendix A.

We also experiment with alternative definitions of the Colombian supply shock. Instead of using the hectares of coca cultivation, one specification uses Colombia's estimated cocaine production volume (in metric tons) as the shock indicator. Another uses a simple dummy variable for the post-2015 period, treating all years from 2015 onward as "after the shock". For comparability, we standardize our main treatment variable to have mean zero and standard deviation one. This way, the coefficient β can be interpreted as the change in the cocaine overdose mortality rate associated with a one standard deviation increase in the Colombian supply shock. We will later translate this effect into more concrete units (such as the number of U.S. overdose deaths per additional hectare of coca) to illustrate its practical magnitude.

3.2 Threats to Identification

Our identification strategy relies on the assumption that, absent Colombia's coca boom, U.S. counties with higher exposure and those with lower exposure would have followed similar overdose mortality trends. In other words, any divergence in outcomes after 2015 should be attributable to the supply shock rather than to pre-existing differences. We present several pieces of evidence to support this parallel-trends assumption and to rule out alternative explanations.

Parallel pre-trends. We first conduct a visual check for parallel trends. Figure 2, panel B divides U.S. counties into four groups: those with zero cocaine exposure and, among the exposed counties, the bottom, middle, and top terciles of exposure. We then plot the cocaine-related overdose death rate for each group over time. Prior to 2015, the overdose trends for the low-, medium-, and high-exposure counties track each other closely. Their levels are also similar. After 2015, however, the high-exposure counties exhibit a much steeper increase in cocaine overdose mortality. By 2020, the overdose death rate in the top-exposure tercile is roughly 20% higher than in the middle- or low-exposure groups. Notably, even counties with no exposure (which serve as a baseline) begin to see a small uptick around 2013, but the post-2015 surge is far more pronounced in the counties that were heavily exposed to cocaine trafficking.

We also perform a formal event-study analysis to test for any pre-shock differences in trends. We augment our main regression equation (1) with a full set of year-specific shock variables, as described in equation (2) below.

$$\text{Cocaine overdose mortality}_{it} = \sum_{\tau \in [-5,4]} \beta_\tau \cdot 1(t=\tau)_t \times \text{Exposure}_i + \alpha_i + \alpha_t + \varepsilon_{it} \quad (2)$$

where $1(t=\tau)_t$ is an event-year dummy that equals 1 if year t is the τ-th year since 2015 (i.e., event year 0 is year 2015). We omit the event year dummy for $\tau = -1$ from the regression. That is, we use 2014 (the year immediately preceding the onset of the coca boom) as the reference year. The β_τ coefficients trace the evolution of the relationship between cocaine-related overdose mortality and county-level coca exposure over time. If the parallel-trends assumption holds, the β_τ coefficients on the exposure – year interactions should be near zero in the years before the shock, and then turn positive once the shock occurs.

Falsification tests with placebo exposures. We next check whether some unobserved factor might be driving our results. One concern is that high-exposure counties could share other characteristics (for example, being large urban centers) that experienced their own shocks unrelated to Colombian coca. To assess this, we construct "fake" exposure measures that have no direct connection to Colombian cocaine. For example, we use each county's pre-2015 share of non-cocaine drug offenses, or the county's share of non-Colombian population, as alternative exposure variables. We then re-run our analysis using these placebo exposure definitions. If our original result were driven by a spurious correlation (say, if high-exposure counties were simply big cities hit by a different drug epidemic), we would expect to see a false "effect" with these fake exposures as well.

Falsification tests with permutation. We further test the robustness of our result using a permutation approach. Essentially, this is a more generalized version of our falsification tests above with manually defined placebo exposures: here, we randomly shuffle the assignment of the exposure share variable $\mathrm{Exposure_i}$ across counties within each state and re-estimate the model 1,000 times. This generates a distribution of "placebo" estimates that would arise if the geographic pattern of exposure were effectively random. If the actual estimate lies far out in the upper tail of this null distribution, then this exercise would confirm that our result is unlikely to have emerged by chance or due to generic within-state trends, broadly diffused policies, or other spatial patterns that are not accounted for by our fixed effects. The permutation test is especially powerful because it preserves any state-level factors (each shuffle keeps the overall exposure totals in each state the same) while breaking the specific county-by-county alignment between exposure and outcomes.

Concurrent drug epidemics. Finally, we consider whether the rise of other drugs—most notably fentanyl—could be confounding our results. The mid-2010s saw a nationwide surge in fentanyl and other synthetic opioids, which might have independently raised overdose mortality. However, fentanyl's spread was rapid and pervasive across the entire country, with no reason to expect it would concentrate in cocaine-exposed counties. In fact, opioid abuse was often higher in rural areas that had low cocaine exposure, the opposite pattern to cocaine. We conduct several checks to verify that the cocaine supply shock is driving our findings, rather than coincident opioid trends. First, we re-estimate equation (1) using the overdose death rate excluding cocaine-related deaths as the outcome to see whether we find an effect on this non-cocaine overdose mortality measure. Second, we augment equation (1) with placebo difference-in-differences terms that interact the coca boom with county's fentanyl exposure share. If fentanyl were driving the results, this additional term would also predict higher overdose deaths when interacted with the shock, and it should reduce the coefficient on the cocaine-shock interaction. Third, we use a more stringent definition of cocaine overdose deaths based only on cases where the death certificate explicitly names cocaine (see Section 2.2 for details). Fourth, we estimate parallel specifications using overdose deaths explicitly attributed to fentanyl and other non-cocaine drugs as placebo outcomes.

3.3 Results

Main effect. The surge in cocaine supply from Colombia led to a pronounced increase in U.S. cocaine-related overdose deaths. Table 1 presents our core regression results estimating equation (1). In Panel A, column 1, the coefficient indicates that a one standard deviation increase in a county's exposure to the Colombian supply boom raised the cocaine overdose mortality rate by 0.387 deaths per 100,000 population per year. This is about a 6% increase relative to the sample's mean cocaine-related mortality rate of roughly 6.3 per 100,000. The effect is precisely estimated and statistically significant at the 1% level.

In columns 2–4 of Panel A, we test the robustness of this result to alternative exposure measures. In column 2, we use a more targeted proxy for pre-shock cocaine prevalence (cocaine-specific offense rates, rather than overall drug offenses, derived from

NIBRS data). In columns 3 and 4, we use the NFLIS drug lab data for exposure, which cover a different set of counties, including many rural areas underrepresented in NSDUH. Across all these variations, the estimated effect remains positive, statistically significant, and of similar magnitude to the baseline in column 1. This consistency suggests that our findings are not sensitive to the exact way we measure a county's pre-2015 cocaine exposure.

Panels B and C of Table 1 report results using different definitions of the Colombian supply shock. Panel B uses Colombia's estimated cocaine production (in tons) instead of coca cultivation area as the shock metric, while Panel C uses a simple indicator (dummy) for years after 2015, i.e., treating the post-2015 period as the "treatment" years uniformly. The results are again qualitatively similar: higher exposure counties experienced a significant rise in overdose deaths after the shock. The magnitude in Panel B is nearly identical to Panel A, and in Panel C it is somewhat attenuated; the coefficient is roughly half as large as in Panel A, likely because a coarse post-2015 dummy captures the timing of the shock less precisely. All specifications confirm that the Colombian coca boom had a meaningful effect on U.S. cocaine mortality.

Temporal pattern and lag. We next examine the timing of the effects. As discussed earlier, Figure 2 provides a visual comparison of raw trends of cocaine-related overdose in high- vs. low-exposure counties. We formalize the parallel-trends test in Figure 3 with an event-study analysis outlined in equation (2). Here, we repeat the estimation underlying Table 1, panel C, column 1, but instead of a single post-2015 indicator, we include indicators for each year before and after 2015 (with year 2014 serving as the reference). Figure 3, panel A plots the coefficients for each year using raw overdose death counts as the outcome, and Figure 3, panel B does the same using overdose mortality rates (per 100,000 people, population-weighted). The pattern is clear in both: the estimated exposure effect in years prior to 2015 exhibits insignificant variation around zero, indicating no pre-trend. Starting in 2016, the coefficients become positive and statistically significant. The impact grows for a few years, peaking around 3–4 years after the shock (i.e., 2018–2019), and then plateaus.

These temporal patterns serve as strong parallel trend tests suggesting a lack of substantial difference between trends in overdose deaths in high versus low exposure counties until the coca boom occurred in 2015. These patterns also shed light on how quickly the impact materialized after the supply shock: the mortality effects appeared with only a short lag, within a year of the coca surge. The main policy-relevant point is that the spillover was not delayed by decades or highly attenuated – it was a near-term effect that would be noticeable within policy horizons.

Demographic groups. We examine whether the cocaine supply shock's impact on mortality was concentrated in certain demographic groups. Table 2, Panel A disaggregates the effect by age. The largest proportional response is among adults aged 45–64, for whom a one-standard-deviation increase in exposure raises cocaine-related mortality by 0.904 per 100,000, roughly 9.4% of the age-group baseline. The effect is also statistically significant but smaller in proportional terms for ages 25–44 (0.305, or 2.7% of the baseline) and ages 15–24 (0.071, or 1.6% of the baseline). We find no significant effect for those under 15 or for adults 65 and older. Panel B indicates larger effects among Black and male populations, both in absolute terms and relative to each group's baseline mortality rate.

Price and purity mechanisms. Through what channel did the upstream supply surge translate into downstream access? From the LIMS data, we collapse individual cocaine seizures to the state-year level and compute mean retail price, mean purity, and the standard deviation of purity. Appendix Table 1 regresses each of these on a post-2015 indicator with state fixed effects. The supply shock appears to have operated primarily through purity: after 2015, mean cocaine purity rose by 15.2 percentage points ($p < 0.01$), roughly a 30% increase over its pre-2015 base. By contrast, the estimated price change is small and statistically insignificant. This is consistent with standard models of illegal drug markets in which retail prices are set primarily by distribution risk premia and upstream supply shocks instead pass through as changes in product quality. Because users often cannot precisely observe purity at the point of consumption, higher purity translates mechanically into higher overdose risk, providing a direct link between the Colombian supply shock and the mortality increase documented above.

Specificity to cocaine-related mortality. We next ask whether Colombia's coca boom had spillover effects on drug deaths beyond those involving cocaine. If the shock mainly increased cocaine availability, we would expect the excess deaths to be concentrated in cocaine overdoses, rather than in fatalities from other substances or unrelated causes. The estimates are consistent with this. We detect essentially no change in non-cocaine drug overdose mortality in high-exposure counties relative to low-exposure counties. Table 3 confirms that the estimated coefficients for overdose deaths not involving cocaine are near zero and not statistically significant. We also consider broader categories of mortality that might be indirectly affected by drug abuse or despair. For example, Appendix Table 2 examines "deaths of despair" (suicides, alcohol-related deaths, and drug overdoses) excluding any cocaine overdoses, following the definitions in Ruhm (2018) and Case and Deaton (2021). Again, we find no evidence of an increase; if anything, some estimates are slightly negative. These null results reinforce that the Colombian shock's impact was quite specific to cocaine-related fatalities. The most plausible mechanism is a rise in cocaine consumption due to greater supply: for example, more people using cocaine, or users consuming larger quantities because the drug was more available and affordable.

Table 4 examines "drug-specific" overdose mortality rates, which we construct using the subset of death certificates that explicitly list the drugs involved, as described in Section 2.2. Because this restrictive approach captures fewer total deaths, the baseline mean for cocaine-specific mortality is much lower in this exercise than in our main analysis. We estimate separate regressions for cocaine, fentanyl, heroin, methadone, and other natural or semi-synthetic opioids. Consistent with all earlier findings, the coca shock leads to a rise in cocaine-specific overdose deaths, but we detect no change in deaths involving any other drug category.

Robustness and falsification checks. In the Online Appendix, we report a variety of additional checks showing that our results are robust. First, we show that the main findings are virtually unchanged if we do not weight the regressions by population (Appendix Table 3). Second, the result is similar or stronger when we restrict the sample to only those counties that had some baseline cocaine exposure (i.e. dropping the zero-exposure counties). Appendix Table 4 presents this test: the estimated effect is larger when zero-exposure counties are excluded, albeit less precisely estimated. This pattern is consistent with a small attenuation bias from including many counties that were never "at risk" of cocaine inflows. Third, we conduct falsification tests by altering how the exposure share is constructed (Table 5). In one variant, we use non-cocaine-related offenses as the basis for the county's exposure proxy, and in another we use the county's non-Colombian population share. In both cases we again find no effect, reinforcing that an unrelated exposure proxy does not produce a spurious result. Fourth, we address the concern that NIBRS coverage during the 2010–2014 baseline window was incomplete, which could introduce measurement error into exposure measures built from NIBRS crime data. Appendix Table 5 re-estimates the main specification on the subsample of counties with positive NIBRS-derived cocaine exposure; the results are quantitatively similar to our main estimates. Fifth, we examine the functional form of the exposure index itself. Our baseline index is the product of three standardized share components (drug-use prevalence, drug-related crime share, and Colombian-born population share); Appendix Table 6 re-estimates the main specification replacing this product with an additive index that sums the three components. Across all eight specifications (four share combinations × two functional forms), the estimated effect remains positive and significant at the 1% level, and the additive form in fact produces larger point estimates than the multiplicative baseline, indicating that the multiplicative choice is conservative. Sixth, we replace the Colombian population share with an independent perishables-import proxy (Appendix Table 6, Panel C); the estimated effect remains similar across all four (drug-use, crime) combinations.

Figure 4 reports a more generalized falsification check. It shows spatial permutation test results as we randomly reshuffle county exposure values within state. We find that the true effect size lies far outside the permutation distribution, suggesting again the impact is specific to actual geography of pre-boom cocaine exposure prior to the coca boom.

Table 6 examines more directly whether fentanyl exposure could confound the relationship between Colombian supply shocks and cocaine-related overdose mortality. As fentanyl overdoses have gone up sharply in the past decade, it is important to assess whether counties with higher fentanyl exposure might mechanically generate patterns that resemble cocaine-driven responses. To address this, we estimate a joint specification that includes two interaction terms: the Colombian coca supply shock interacted with the county's cocaine exposure share, and the same supply shock interacted with the county's fentanyl exposure share. Across all three definitions of the $Coca_t$ variable (cultivation-based, production-based, and a post-2015 indicator) the results consistently show a strong and statistically significant effect for the $Coca_t$ × Cocaine Exposure interaction. In contrast, the $Coca_t$ × Fentanyl Exposure interaction is consistently smaller in magnitude than the cocaine-exposure interaction and changes sign across specifications. In several NFLIS-based columns it is positive and significant; nonetheless the cocaine-exposure coefficient remains large and highly significant in every column. Importantly, the inclusion of fentanyl exposure does not meaningfully change the magnitude or significance of the cocaine-exposure effect.

Effectiveness of demand-side mitigation. Our study provides an opportunity to contribute to the ongoing debate about the demand and supply of drugs and their health impacts. Specifically, we examine the role of demand-side mitigation measures, such as counterdrug enforcement and the availability of drug treatment facilities, in the face of an exogenous increase in drug supply. To test this, in Table 7, columns 1 and 2, we estimate heterogeneous exposure effects based on counties' HIDTA (High Intensity Drug Trafficking Area) designation. If demand-side efforts dampened the supply shock's impact, one might expect smaller effects in HIDTA counties. Instead, we find the opposite: HIDTA-designated counties experience greater, not lesser, exposure effects. Column 3 presents a full interaction regression, where we interact the exposure effect term with the HIDTA indicator, and the results remain consistent. In column 4, we use the number of drug treatment facilities in the county as a second measure of demand-side regulatory effort. Again, we find no statistically significant evidence that this measure mitigates the exposure effect. These results should be interpreted with caution since they are correlational (counties with HIDTA status or more clinics may differ in many ways). However, it appears that existing demand-side measures did not meaningfully buffer the mortality impact of the foreign supply shock. Even areas with substantial drug enforcement presence or treatment availability saw pronounced increases in cocaine-related deaths when Colombian supply surged.

Magnitude of effect. Finally, we translate the estimates into aggregate deaths and dollar costs. Using our preferred estimate from Table 1 Panel A, column 1, we can approximate how many additional overdose deaths in the United States were attributable to Colombia's coca expansion. By applying the estimated exposure effect nationally, we calculate that in 2022 (when Colombia's coca cultivation reached roughly 230,000 hectares) the cocaine supply shock led to about 1,483 excess U.S. drug overdose deaths in that year alone. In 2015, before the surge, Colombian cultivation was much lower; our estimates imply that the Colombian supply at that time would have been associated with on the order of 620 U.S. overdose deaths annually. Summing over 2016–2022, the coca boom in Colombia likely resulted in several thousand additional American deaths in total. Put differently, each extra hectare of coca cultivated in Colombia eventually translated into approximately 0.0063 additional overdose deaths per year in the United States. Because our mortality data end in 2020, the 2021–2022 calculations apply our preferred coefficient to those years' cultivation levels and should be interpreted as model-implied extrapolations rather than directly observed mortality.

We can also express this externality in monetary terms. Based on the U.S. Environmental Protection Agency's standard Value of a Statistical Life (about $7.4 million in 2006 dollars; all dollar figures below are expressed in 2006 dollars), the loss of life from one hectare of coca cultivation corresponds to roughly $45,000 in damages per year. In other words, every hectare of Colombian coca imposes an annual cost of about $45,000 on U.S. society through increased overdose fatalities. For perspective, a Colombian farmer's annual profit from one hectare of coca is only a few thousand dollars, which is far below the $45,000 per-hectare cost imposed on the U.S. With roughly 230,000 hectares now under cultivation, that translates into on the order of $10 billion per year in U.S. overdose mortality costs. This calculation highlights the enormous health and economic stakes of international drug supply shocks. We turn to the policy implications of these findings in the next section.

4. Policy Implications and Conclusion

Our empirical findings carry several policy implications, which we discuss in turn. First, the rapid mortality response we document after Colombia's 2015 enforcement disruption underscores how quickly cross-border health spillovers can materialize when drug-control regimes change. Future shifts in international drug policy—whether in source countries, transit corridors, or destination markets—warrant explicit assessment of downstream public-health consequences.

Second, the same elasticity that maps higher Colombian cultivation into U.S. mortality also implies a domestic dividend from earlier supply-side aid: if the 2000s decline in Colombian coca was driven materially by U.S. counter-narcotics support, our estimates imply that such aid prevented a substantial number of overdose deaths annually.

Third, our estimates provide a tractable benchmark for evaluating future interventions: each additional hectare of Colombian coca translates into roughly $45,000 per year in U.S. mortality costs, valued using the standard EPA value of statistical life. While a full cost-benefit accounting of any specific policy lies beyond the scope of this paper, these magnitudes establish a substantial empirical benchmark for prospective coca-reduction investments.

Finally, our results speak to the long-standing question of supply versus demand in drug-mortality policy. We find that upstream supply reductions move U.S. mortality even when domestic demand-side factors are held fixed, and our mechanism evidence points to product purity—not just availability—as a margin through which supply-side interventions can reduce overdose risk.

This paper shows that Colombia's post-2015 coca surge caused a measurable rise in U.S. cocaine overdose deaths. We link the foreign supply shock to larger increases in mortality in U.S. counties that were more exposed before the shock. The timing is tight: effects appear within a year of the surge and persist. Our preferred estimates imply roughly 1,500 excess U.S. overdose deaths per year in the late 2010s and early 2020s attributable to the boom. These results are robust across alternative exposure measures, alternative ways to define the shock, and placebo tests, and they are specific to cocaine-related mortality.

Taken together, these results indicate that even modest reductions in source-country coca cultivation could yield large mortality benefits in the United States, and that lapses in supply-side enforcement abroad warrant explicit accounting for cross-border health spillovers.

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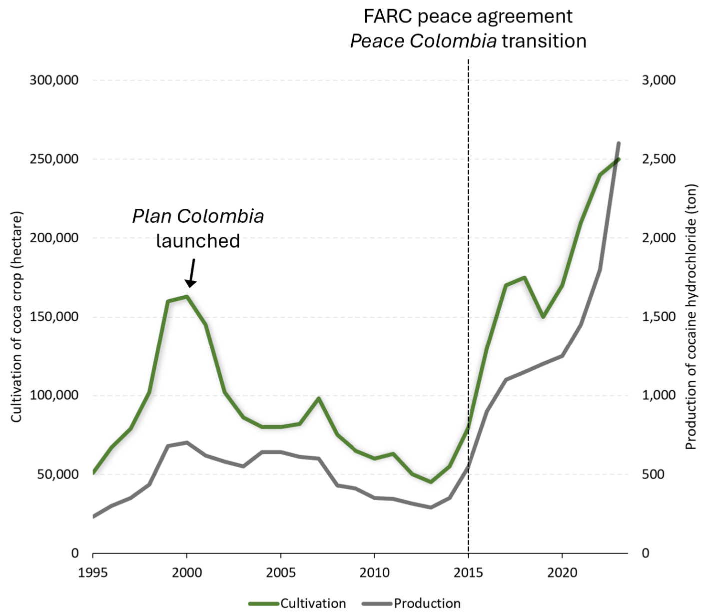
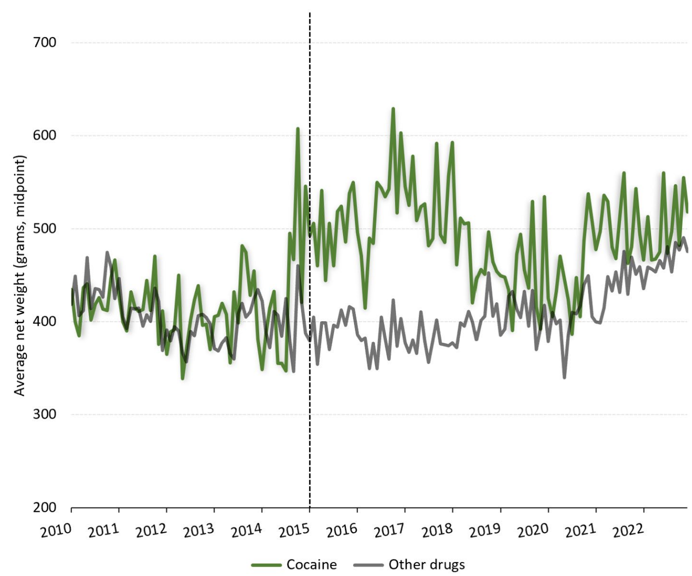
Figures and Tables

Figure 1. Trends in Colombia's Coca Cultivation and U.S. Cocaine Seizures

A. Coca Cultivation and Potential Production in Colombia



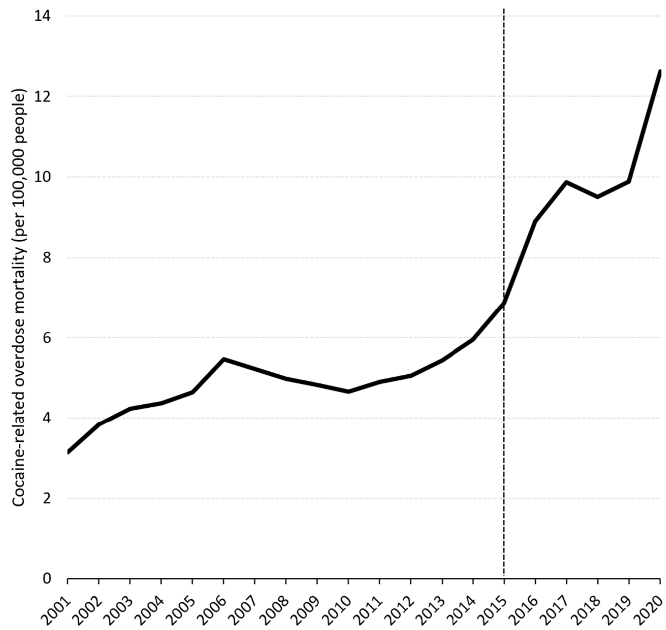
B. Average Net Weight of Cocaine and Other Drug Seizures in the United States



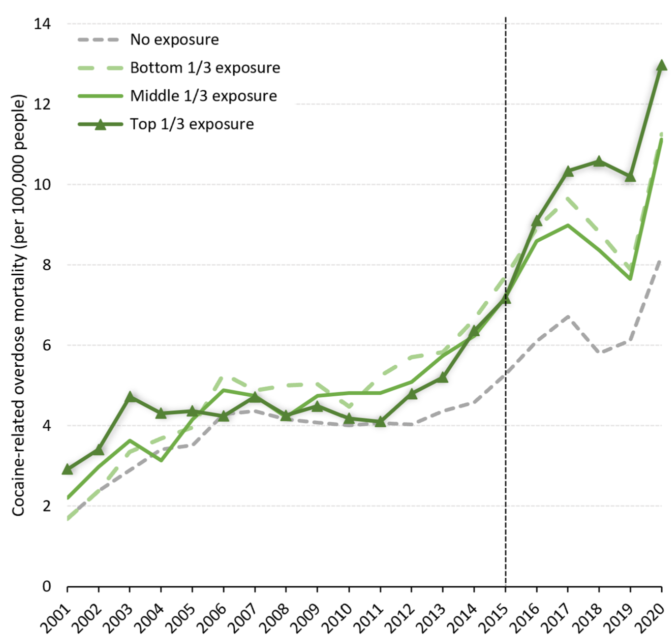
Notes: Panel A plots total coca cultivation area (in hectares, left axis) and potential cocaine production (in metric tons, right axis) in Colombia from 1995–2022, based on United Nations Office on Drugs and Crime (UNODC) annual estimates. Panel B plots monthly average net weight (grams, midpoint of DEA brackets) of drug seizures reported in the DEA Laboratory Information Management System (LIMS). The green line shows cocaine samples; the gray line shows all other drug types. The vertical dashed line in both panels marks the start of year 2015.

Figure 2. Trends in U.S. Cocaine-Related Overdose Mortality and Cross-County Differences

A. National Cocaine-Involved Overdose Mortality



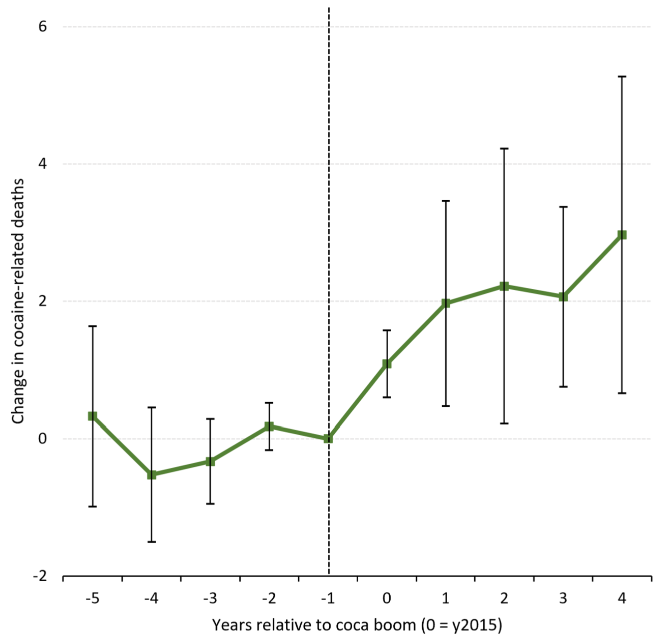
B. Overdose Mortality in High- versus Low-Exposure Counties



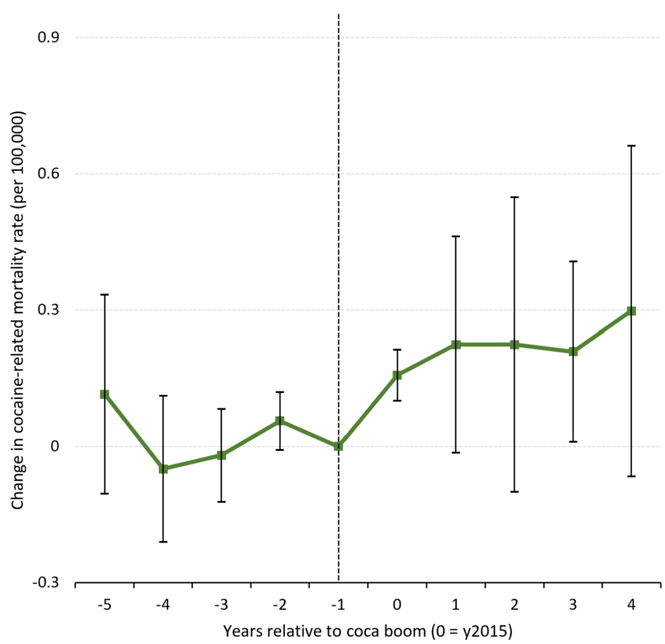
Notes: Panel A plots the national rate of cocaine-involved overdose deaths (per 100,000 people) in the United States. Panel B separates counties by their pre-2015 exposure to Colombian cocaine, corresponding to the top, middle, and bottom terciles of the Cocaine Exposure Index, with counties recording zero exposure shown separately.

Figure 3. Event Study Estimates of the Cocaine Supply Shock on U.S. Cocaine Overdose Mortality

A. Raw Death Counts

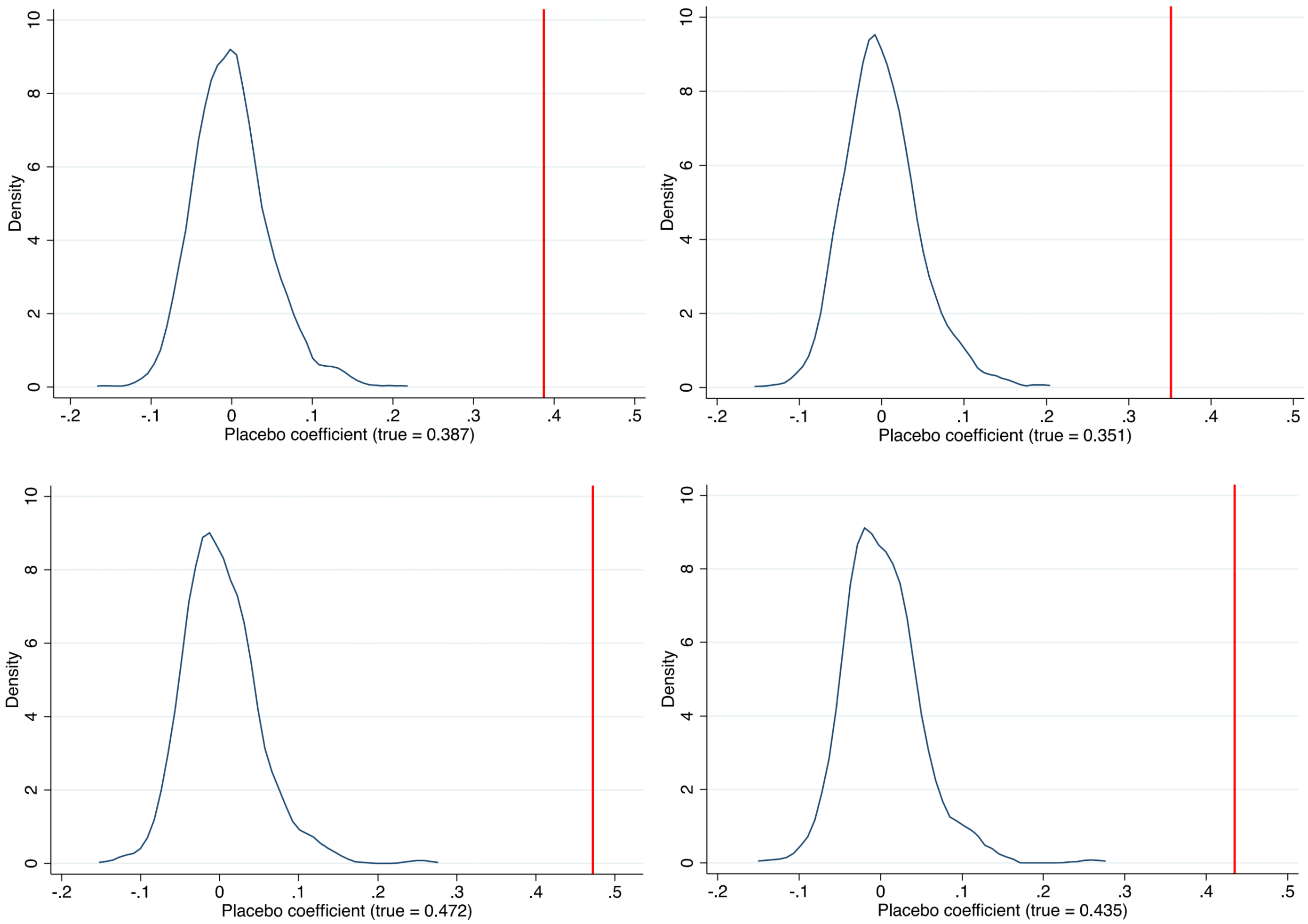


B. Cocaine-Related Overdose Mortality Rate



Notes: Each panel plots coefficients from an event-study regression estimating the dynamic effects of exposure to Colombia's coca boom on U.S. cocaine-involved overdose outcomes. The horizontal axis shows event time centered on 2015 (defined as event year 0). The vertical dashed line marks one year prior to the onset of the supply shock. Bars show 95 percent confidence intervals.

Figure 4. Spatial Permutation Tests for the Cocaine Exposure Effect



Notes: Each panel shows the distribution of placebo coefficients from 1,000 spatial permutation tests in which the pre-2015 county exposure index ($Exposure_i$) is randomly reassigned among counties within the same state. The blue curve shows the density of estimated coefficients under these placebo reassignments; the red vertical line marks the true estimated coefficient from the actual exposure geography (corresponding to Table 1, Panel A), ranging from 0.351 to 0.472 across the four panels (Panel A: 0.387; Panel B: 0.351; Panel C: 0.472; Panel D: 0.435).

Table 1. The Effect of the Cocaine Supply Shock on U.S. Cocaine-Related Overdose Mortality

A. Cultivation area as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.387***	0.351**	0.472***	0.435**
	(0.131)	(0.137)	(0.104)	(0.177)
Observations	59096	59096	59096	59096
Y-mean	6.320	6.320	6.320	6.320
Y-sd	6.441	6.441	6.441	6.441
B. Production as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.402***	0.366**	0.495***	0.460**
	(0.150)	(0.157)	(0.124)	(0.209)
Observations	29561	29561	29561	29561
Y-mean	7.946	7.946	7.946	7.946
Y-sd	7.812	7.812	7.812	7.812
C. Post 2015 as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.170***	0.157***	0.204***	0.191**
	(0.057)	(0.059)	(0.047)	(0.079)
Observations	59096	59096	59096	59096
Y-mean	6.320	6.320	6.320	6.320
Y-sd	6.441	6.441	6.441	6.441
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: Each panel reports estimates from county-by-year regressions of cocaine-related overdose mortality on exposure to Colombia's coca expansion. The key variable, $Coca_t \times Exposure_i$, is the interaction between Colombia's coca shock (measured variously as cultivation area, potential production, or a post-2015 indicator) and each county's pre-2015 exposure to Colombian cocaine. Exposure is proxied using pre-shock measures from the National Survey on Drug Use and Health (NSDUH) and the DEA's National Forensic Laboratory Information System (NFLIS), based on drug-offense or cocaine-specific property crime shares. The dependent variable is the cocaine-related overdose death rate per 100,000 population. All models include county and year fixed effects; standard errors (in parentheses) are clustered at the county level. ***, **, * denote statistical significance at the 1, 5, and 10 percent levels, respectively.

Table 2. Heterogeneity of Exposure Effects by Demographic Groups

A. By Age

	Mortality rate (#deaths per 100,000)				
	<15	15–24	25–44	45–64	65+
$Coca_t \times Exposure_i$ (std)	0.0003	0.0710***	0.3050**	0.9040*	0.4786
	(0.0044)	(0.0230)	(0.1490)	(0.5120)	(0.3786)
Observations	10,475	59,096	59,096	59,096	31,755
Y-mean	0.127	4.489	11.259	9.618	1.764
Y-sd	0.818	6.444	13.302	11.546	3.610
County, Year FEs	Y	Y	Y	Y	Y
Drug use	NSDUH	NSDUH	NSDUH	NSDUH	NSDUH
Crime	Drug offense	Drug offense	Drug offense	Drug offense	Drug offense

B. By Race and Gender

	Mortality rate (#deaths per 100,000)				
	White	Black	Other race	Male	Female
$Coca_t \times Exposure_i$ (std)	0.2653***	0.8428**	0.0737	0.6296***	0.1654***
	(0.0382)	(0.3681)	(0.0481)	(0.2164)	(0.0597)
Observations	46904	46699	46904	46904	46904
Y-mean	6.746	8.404	1.578	9.866	3.621
Y-sd	6.413	11.322	5.961	9.866	4.044
County, Year FEs	Y	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NSDUH	NSDUH	NSDUH
Crime measure	Drug offense	Drug offense	Drug offense	Drug offense	Drug offense

Notes: This table estimates the heterogeneous effects of Colombia's coca expansion on U.S. cocaine-involved overdose mortality by demographic group. The dependent variable is the cocaine-involved overdose death rate (per 100,000 population). Panel A disaggregates the analysis by age group (<15, 15–24, 25-44, 45–64, and 65+), while Panel B separates results by race and gender. The key regressor, $Coca_t \times Exposure_i$, interacts Colombia's coca cultivation with each county's pre-2015 cocaine exposure share (proxied by NSDUH drug-offense data). All specifications include county and year fixed effects; standard errors (in parentheses) are clustered at the county level. ***, **, * denote significance at the 1%, 5%, and 10% levels, respectively.

Table 3. Effects on Non-Cocaine-Related Overdose Mortality

A. Cultivation area as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.029	0.016	0.106	0.095
	(0.085)	(0.080)	(0.072)	(0.092)
Observations	59096	59096	59096	59096
Y-mean	8.196	8.196	8.196	8.196
Y-sd	6.553	6.553	6.553	6.553
B. Production as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.001	-0.006	0.089	0.079
	(0.099)	(0.091)	(0.096)	(0.114)
Observations	29561	29561	29561	29561
Y-mean	10.372	10.372	10.372	10.372
Y-sd	7.178	7.178	7.178	7.178
C. Post 2015 as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.007	0.005	0.034	0.031
	(0.038)	(0.035)	(0.036)	(0.047)
Observations	59096	59096	59096	59096
Y-mean	8.196	8.196	8.196	8.196
Y-sd	6.553	6.553	6.553	6.553
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: Each panel reports estimates from county-by-year regressions of non-cocaine-related overdose mortality on exposure to Colombia's coca expansion. The key variable, $Coca_t \times Exposure_i$, is the interaction between Colombia's coca shock (measured variously as cultivation area, potential production, or a post-2015 indicator) and each county's pre-2015 exposure to Colombian cocaine. Exposure is proxied using pre-shock measures from the National Survey on Drug Use and Health (NSDUH) and the DEA's National Forensic Laboratory Information System (NFLIS), based on drug-offense or cocaine-specific property crime shares. The dependent variable is the non-cocaine-related overdose death rate per 100,000 population. All models include county and year fixed effects; standard errors (in parentheses) are clustered at the county level. ***, **, * denote statistical significance at the 1, 5, and 10 percent levels, respectively.

Table 4. Effects on Drug-Specific Overdose Mortality

	Mortality rate (#deaths per 100,000)				
	Cocaine	Fentanyl	Heroin	Nat./semi-synth	Methadone
$Coca_t \times Exposure_i$ (std)	0.0186**	-0.0007	-0.0012	-0.0002	0.0000
	(0.0073)	(0.0005)	(0.0014)	(0.0016)	(0.0001)
Observations	50436	50436	50436	50436	50436
Y-mean	0.138	0.063	0.371	0.129	0.063
Y-sd	0.661	0.262	0.371	0.672	0.423
County, Year FEs	Y	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NSDUH	NSDUH	NSDUH
Crime measure	Drug offense	Drug offense	Drug offense	Drug offense	Drug offense

Notes: This table reports estimates from county-by-year regressions of drug-specific overdose mortality on exposure to Colombia's coca expansion. The key variable, $Coca_t \times Exposure_i$, is the interaction between Colombia's coca shock (measured as cultivation area) and each county's pre-2015 exposure to Colombian cocaine. Exposure is proxied using pre-shock measures from the National Survey on Drug Use and Health (NSDUH) based on drug-offense data. The dependent variable is drug-specific overdose death rate per 100,000 population. All models include county and year fixed effects; standard errors (in parentheses) are clustered at the county level. ***, **, * denote statistical significance at the 1, 5, and 10 percent levels, respectively.

Table 5. Falsification Checks: Regressions with Placebo Share

	Non-cocaine drug	Non-Colombian
A. Cultivation area as $Coca_t$		
	Mortality rate (#deaths per 100,000)	
$Coca_t$ × $Exposure_i$ (std)	1.025	0.379
	(0.681)	(0.492)
Observations	59096	59098
R-square	0.574	0.573
B. Production as $Coca_t$		
	Mortality rate (#deaths per 100,000)	
$Coca_t$ × $Exposure_i$ (std)	1.022	0.441
	(0.705)	(0.547)
Observations	29561	29561
R-square	0.688	0.687
C. Post 2015 as $Coca_t$		
	Mortality rate (#deaths per 100,000)	
$Coca_t$ × $Exposure_i$ (std)	0.436	0.141
	(0.318)	(0.233)
Observations	59096	59098
R-square	0.574	0.573
County, Year FEs	Y	Y
Drug use measure	NFLIS	NFLIS
Crime measure	Drug offense	Drug offense

Notes: This table presents falsification tests using alternative, non-cocaine-related exposure measures. The dependent variable is the cocaine-related overdose death rate per 100,000 population. The key regressor, $Coca_t$ × $Exposure_i$, interacts Colombia's coca cultivation or production (or a post-2015 indicator) with two placebo versions of county exposure: (1) the county's pre-2015 share of non-cocaine drug offenses, and (2) the share of residents born outside Colombia ("Non-Colombian" exposure). Both placebo exposure variables are constructed from DEA's National Forensic Laboratory Information System (NFLIS) and U.S. Census data. All regressions include county and year fixed effects; standard errors (in parentheses) are clustered at the county level.

Table 6. Regressions with Both Cocaine and Fentanyl Exposure Share

A. Cultivation area as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t$ × Cocaine $Exposure_i$ (std)	0.535***	0.508**	0.436**	0.396**
	(0.129)	(0.221)	(0.107)	(0.185)
$Coca_t$ × Fentanyl $Exposure_i$ (std)	-0.153	-0.117	0.139**	0.121*
	(0.102)	(0.157)	(0.045)	(0.063)
Observations	59096	59096	59096	59096
Y-mean	6.320	6.320	6.320	6.320
Y-sd	6.441	6.441	6.441	6.441
B. Production as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t$ × Cocaine $Exposure_i$ (std)	0.560***	0.550**	0.461***	0.421*
	(0.163)	(0.270)	(0.131)	(0.240)
$Coca_t$ × Fentanyl $Exposure_i$ (std)	-0.190	-0.207	-0.130***	-0.087
	(0.136)	(0.205)	(0.041)	(0.040)
Observations	29561	29561	29561	29561
Y-mean	7.946	7.946	7.946	7.946
Y-sd	7.812	7.812	7.812	7.812
C. Post 2015 as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t$ × Cocaine $Exposure_i$ (std)	0.213***	0.214**	0.187***	0.172**
	(0.059)	(0.102)	(0.049)	(0.063)
$Coca_t$ × Fentanyl $Exposure_i$ (std)	-0.052	-0.064	0.067**	0.060**
	(0.059)	(0.080)	(0.022)	(0.018)
Observations	59096	59096	59096	59096
Y-mean	6.320	6.320	6.320	6.320
Y-sd	6.441	6.441	6.441	6.441
County, Year FEs	Y	Y	Y	Y
Cocaine drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Fentanyl drug use measure	NFLIS	NFLIS	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: This table reports regressions that include both cocaine exposure and fentanyl exposure in the same specification. The dependent variable in all columns is the cocaine-related overdose death rate per 100,000 population. Each panel uses a different definition of $Coca_t$: (1) Colombia's coca cultivation share, (2) Colombia's cocaine production share, and (3) a post-2015 indicator for the Colombian coca-boom period. In each specification, $Coca_t$ × Cocaine Exposure is the main interaction of interest, and $Coca_t$ × Fentanyl Exposure serves as a placebo interaction testing whether fentanyl exposure can account for the observed effects. All regressions include county and year fixed effects, and standard errors are clustered at the county level. ***, **, * denote significance at the 1%, 5%, and 10% levels, respectively.

Table 7. Heterogeneity of Exposure Effects by Demand-Side Regulation

	Mortality rate (#deaths per 100,000)			
	HIDTA Sample	non-HIDTA Sample	Full Sample	
$Coca_t \times Exposure_i$ (std)	0.405***	0.222***	0.195***	0.334***
	(0.152)	(0.070)	(0.059)	(0.118)
$Coca_t \times Exposure_i$ (std) × HIDTA			0.219	
			(0.161)	
$Coca_t \times Exposure_i$ (std) × #Facilities (100s)				0.080
				(0.179)
#Facilities (100s)				-1.705***
				(0.102)
Observations	12830	46266	59096	59096
Y-mean	6.622	5.627	6.320	6.320
Y-sd	6.015	7.275	6.441	6.441
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NSDUH	NSDUH
Crime measure	Drug offense	Drug offense	Drug offense	Drug offense

Notes: Columns 1–2 split counties by their HIDTA (High Intensity Drug Trafficking Area) designation. Columns 3–4 test for heterogeneity by HIDTA and the number of drug treatment facilities. $Coca_t \times Exposure_i$, is the interaction between Colombia's coca shock (measured as 2022 cultivation area) and each county's pre-2015 exposure to Colombian cocaine. All regressions include county and year fixed effects. Standard errors (in parentheses) are clustered at the county level. ***, **, and * denote statistical significance at the 1, 5, and 10 percent levels, respectively.

Appendix A. Construction of the Perishables Exposure Proxy

This appendix details the construction of the Colombian-perishables exposure proxy used in the Panel C robustness check of Appendix Table 6. The motivation is that perishable cargo from Colombia—principally cut flowers and fresh fruit—is a known smuggling vehicle for cocaine trafficking, with the same Atlantic and Gulf air- and sea-freight infrastructure used by both legal perishable shipments and illicit narcotics (Daniele, Soliman, and Vargas, 2026). The construction proceeds in four steps.

Step 1: District-level perishables fraction. For each U.S. customs district j, we define the Colombian perishable import fraction as:

$$\mathrm{FracPerish}_j = \frac{\sum_{t=2010}^{2014} \mathrm{Colombian\ Perishable\ Imports}_{jt}}{\sum_{t=2010}^{2014} \mathrm{Total\ Imports}_{jt}}$$

where Colombian perishable imports are the sum of HS chapter 06 (cut flowers) and HS chapter 08 (edible fruit) imports from Colombia through district j, taken from the Census International Trade API. Among U.S. customs districts, Miami has the highest fraction at 1.3%, followed by Philadelphia at 0.3%.

Step 2: District anchor coordinates. Each customs district is anchored at the centroid of its headquarters county. Headquarters counties are listed in the U.S. Customs and Border Protection district reference file (e.g., the Miami district is anchored at Miami-Dade County, FL; the Philadelphia district at Philadelphia County, PA). County centroid coordinates come from the Census 2020 Gazetteer file.

Step 3: Gravity decay to the county level. For each U.S. county c and customs district j, we compute the great-circle distance d_{cj} using the haversine formula. The county-level perishables fraction is the distance-weighted average of district fractions:

$$\mathrm{FracPerish}_c = \frac{\sum_j \exp\left(-\frac{d_{cj}}{D}\right) \mathrm{FracPerish}_j}{\sum_j \exp\left(-\frac{d_{cj}}{D}\right)}$$

with bandwidth D = 500 km in the baseline. Counties geographically close to a high-fraction port load up on that port's value, while inland counties receive a smoothly attenuated mixture of all districts. We vary the bandwidth across {200, 500, 1000} km as a sensitivity check, reported below.

Step 4: Multiplicative exposure index and regression. The original baseline multiplicative exposure index, used in Panel A of Appendix Table 6, is:

$$\text{Exposure}_i^{\text{base}} = \text{DrugUse}_{s(i)} \times \text{Crime}_i \times \text{ColShare}_i$$

where $\text{DrugUse}_{s(i)}$ is the state-level NSDUH cocaine use rate or NFLIS cocaine identification rate, Crime_i is the county-level drug-offense share or cocaine-property crime share, and ColShare_i is the county-level Colombian-born population share from ACS table B03001. Panel C replaces the Colombian population share with the gravity-weighted county-level perishables fraction:

$$\text{Exposure}_i^{C} = \text{DrugUse}_{s(i)} \times \text{Crime}_i \times \text{FracPerish}_i$$

The product is then standardized across counties. For each of the four columns of Panel C, defined by the (DrugUse, Crime) pair following Appendix Table 6's column ordering, we estimate the following county-year panel regression:

$$\text{MortRate}_{it} = \beta \cdot \left(\text{Coca}_t \times \text{Exposure}_i^{C}\right) + \alpha_i + \alpha_t + \varepsilon_{it}$$

where Coca_t is Colombian cultivation area (the cultivation-area shock used to match Appendix Table 6), α_i are county fixed effects, α_t are year fixed effects, observations are weighted by county population, and standard errors are clustered at the county level.

Two construction choices remain worth flagging. First, the perishable-import data are observed only at the customs-district level. The gravity-decay weighting recovers cross-county variation by attenuating each district's fraction with distance, but it imposes the assumption that the downstream distribution of Colombian perishables (and, by analogy, Colombian cocaine routes) follows a smooth distance-decay pattern from the port of entry. Counties served primarily by surface freight from inland hubs may not fit this assumption.

Second, the bandwidth D = 500 km is a tuning parameter. The table below reports estimates under alternative bandwidths D ∈ {200, 500, 1000} km. The point estimates are stable in sign and significance across all three bandwidths.

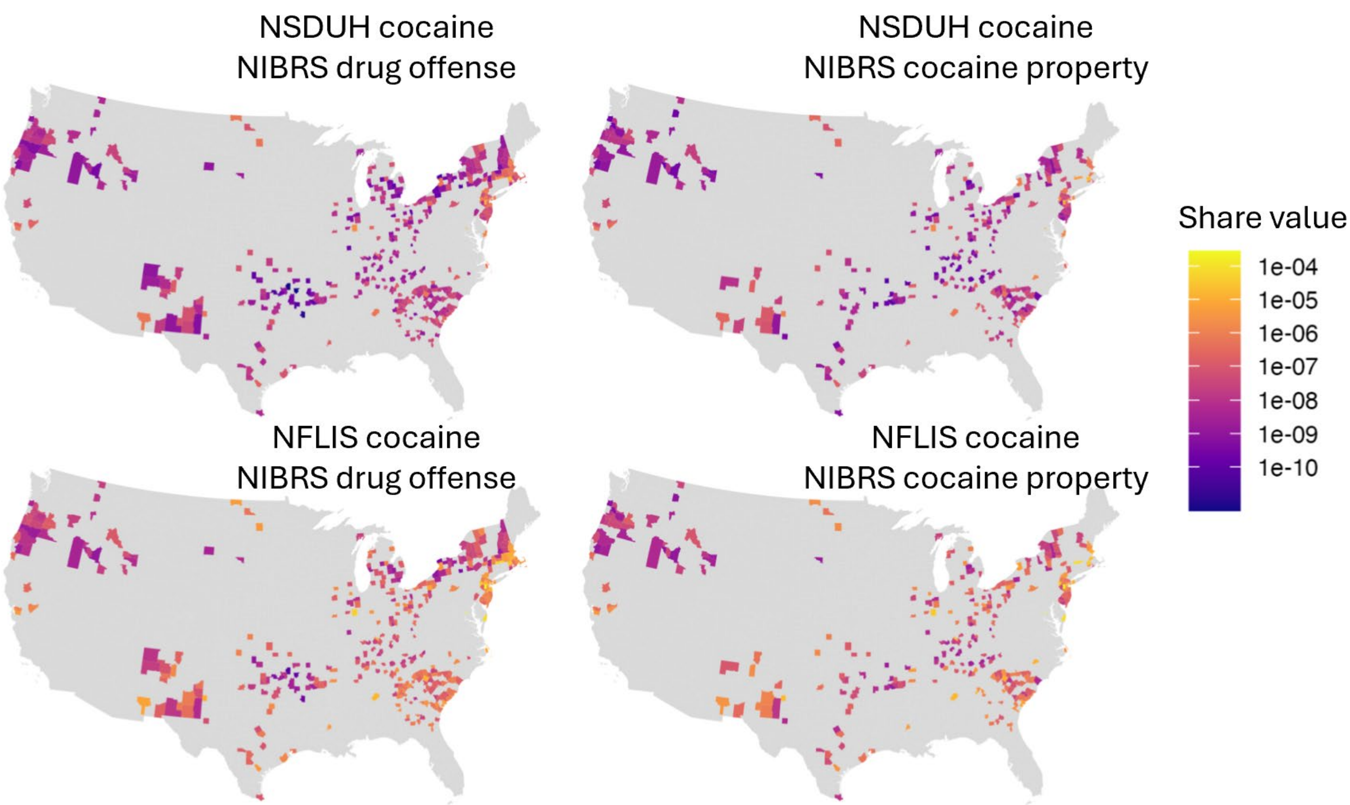
Table A1. Sensitivity to the gravity-decay bandwidth D.

	Mortality rate (#deaths per 100,000)			
D = 200 km				
$Coca_t \times Exposure_i$ (std)	0.507***	0.483***	0.715***	0.668***
	(0.081)	(0.058)	(0.107)	(0.099)
D = 500 km (baseline)				
$Coca_t \times Exposure_i$ (std)	0.546***	0.530***	0.644***	0.619***
	(0.067)	(0.051)	(0.086)	(0.087)
D = 1000 km				
$Coca_t \times Exposure_i$ (std)	0.559***	0.545***	0.627***	0.609***
	(0.058)	(0.055)	(0.097)	(0.097)
Observations	59,096	59,096	59,096	59,096
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: Each block re-estimates the Panel C specification of Appendix Table 6 under a different gravity-decay bandwidth D. A smaller D concentrates the exposure index on counties geographically close to the high-fraction customs districts (Miami, Philadelphia); a larger D spreads the exposure more evenly inland. All other features of the regression are unchanged. Standard errors (in parentheses) are clustered at the county level. ***, **, * denote significance at the 1%, 5%, and 10% levels.

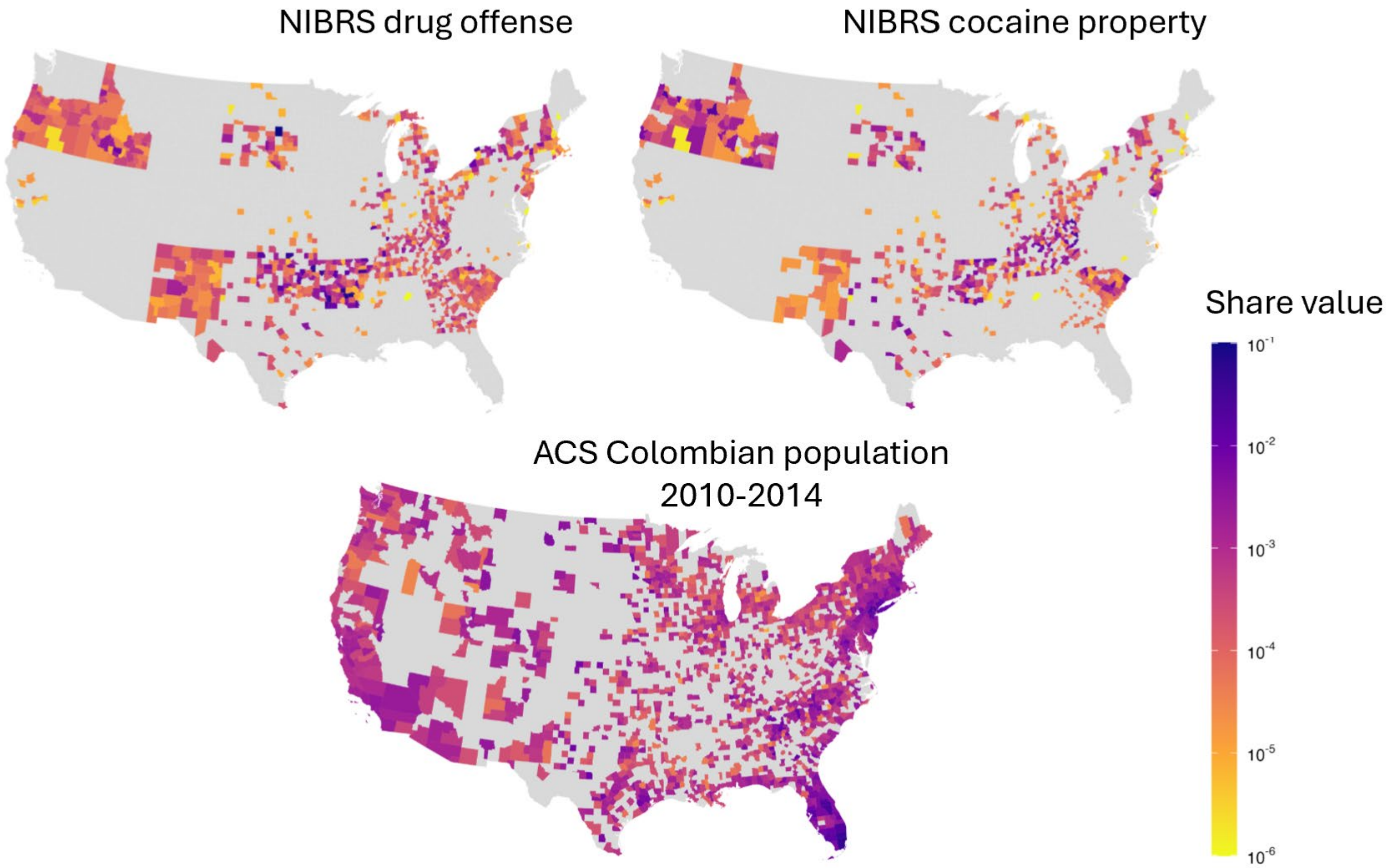
Appendix Figures and Tables

Appendix Figure 1. Geographic Distribution of County-Level Cocaine Exposure Measures



Notes: This figure maps four variants of the county-level cocaine exposure measure (Exposure_i), each constructed as the product of a state-level cocaine-use proxy and a county-level cocaine-concentration proxy. The top row uses state-level cocaine-use prevalence from the National Survey on Drug Use and Health (NSDUH, 2010–2014); the bottom row uses state-level cocaine drug reports from the DEA's National Forensic Laboratory Information System (NFLIS). The left column apportions the state-level measure to counties using the county's share of NIBRS drug-offense incidents; the right column uses the county's share of NIBRS cocaine-specific property-crime incidents. Colors indicate the relative intensity of cocaine exposure within a state (higher values in yellow, lower in purple). Darker shades represent counties more historically involved in cocaine markets. Grey areas indicate counties with missing or zero exposure observations.

Appendix Figure 1, continued. Geographic Distribution of County-Level Cocaine Exposure Measures



Notes: This figure maps the three raw data components that underlie the county-level cocaine exposure measure Exposure_i (see Equation 1). Panel 1 shows the county's share of NIBRS drug-offense incidents (all drug-related police incidents, 2010–2014). Panel 2 shows the county's share of NIBRS cocaine-specific property-crime incidents over the same period. Panel 3 shows the county's share of Colombian-born population from the 2010–2014 American Community Survey (ACS). Colors indicate the relative intensity within a state (higher values in yellow, lower in purple). Darker shades represent counties more historically involved in cocaine markets or with larger Colombian communities. Grey areas indicate counties with missing or zero observations.

Appendix Table 1. Post-2015 Change in Cocaine Price, Purity, and Volume

	Mean Price ($)	Mean Purity (%)	SD Purity (%)
Post-2015	29.7	15.2***	-1.5
	(31.6)	(2.5)	(1.0)
Observations	439	213	202
Y-mean	997.0	67.6	12.6
State fixed effects	Yes	Yes	Yes

Notes: Unit of observation is state-year (DEA LIMS, 2010–2022). Price and purity are computed from midpoints of categorical bins in the LIMS Public Use File. All regressions include state fixed effects with clustering at the state level. ***, **, * denote significance at the 1%, 5%, and 10% levels, respectively.

Appendix Table 2. Effects on Non-Cocaine-Related "Deaths of Despair"

A. Cultivation area as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	-0.101	-0.103	-0.016	-0.018
	(0.077)	(0.066)	(0.082)	(0.081)
Observations	59096	59096	59096	59096
Y-mean	24.821	24.821	24.821	24.821
Y-sd	11.986	11.986	11.986	11.986
B. Production as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	-0.134	-0.139*	-0.038	-0.041
	(0.094)	(0.070)	(0.106)	(0.104)
Observations	29561	29561	29561	29561
Y-mean	29.063	29.063	29.063	29.063
Y-sd	12.683	12.683	12.683	12.683
C. Post 2015 as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	-0.072**	-0.066**	-0.054	-0.049
	(0.035)	(0.028)	(0.043)	(0.034)
Observations	59096	59096	59096	59096
Y-mean	24.821	24.821	24.821	24.821
Y-sd	11.986	11.986	11.986	11.986
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: Each panel reports estimates from county-by-year regressions of non-cocaine-related "deaths of despair" mortality on exposure to Colombia's coca expansion. The key variable, $Coca_t \times Exposure_i$, is the interaction between Colombia's coca shock (measured variously as cultivation area, potential production, or a post-2015 indicator) and each county's pre-2015 exposure to Colombian cocaine. Exposure is proxied using pre-shock measures from the National Survey on Drug Use and Health (NSDUH) and the DEA's National Forensic Laboratory Information System (NFLIS), based on drug-offense or cocaine-specific property crime shares. All models include county and year fixed effects; standard errors (in parentheses) are clustered at the county level. ***, **, * denote statistical significance at the 1, 5, and 10 percent levels, respectively.

Appendix Table 3. Robustness: Unweighted Regressions

A. Cultivation area as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.523***	0.462***	0.569***	0.483**
	(0.142)	(0.146)	(0.153)	(0.208)
Observations	59096	59096	59096	59096
Y-mean	4.688	4.688	4.688	4.688
Y-sd	6.778	6.778	6.778	6.778
B. Production as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.550***	0.487***	0.599***	0.510**
	(0.150)	(0.166)	(0.174)	(0.238)
Observations	29561	29561	29561	29561
Y-mean	5.824	5.824	5.824	5.824
Y-sd	7.838	7.838	7.838	7.838
C. Post 2015 as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.215***	0.210***	0.250***	0.213**
	(0.062)	(0.065)	(0.068)	(0.093)
Observations	59096	59096	59096	59096
Y-mean	4.688	4.688	4.688	4.688
Y-sd	6.778	6.778	6.778	6.778
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: This table replicates the main specification from Table 1 but estimates all regressions without population weighting. The dependent variable is the cocaine-related overdose death rate per 100,000 population. The key regressor, $Coca_t \times Exposure_i$, interacts Colombia's coca cultivation or production (or a post-2015 indicator) with each county's pre-2015 cocaine exposure share. Exposure is proxied using the National Survey on Drug Use and Health (NSDUH) and the DEA's National Forensic Laboratory Information System (NFLIS), based on drug-offense or cocaine-property crime measures. All models include county and year fixed effects, and standard errors (in parentheses) are clustered at the county level. ***, **, * denote significance at the 1%, 5%, and 10% levels, respectively.

Appendix Table 4. Robustness: Counties with Positive Exposure Only

A. Cultivation area as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t$ × $Exposure_i$ (std)	0.760**	0.818**	0.990***	1.081**
	(0.339)	(0.402)	(0.290)	(0.534)
Observations	7960	5940	7960	5940
Y-mean	7.538	7.196	7.538	7.196
Y-sd	6.883	6.673	6.883	6.673
B. Production as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t$ × $Exposure_i$ (std)	0.842**	0.909*	1.096***	1.204*
	(0.402)	(0.475)	(0.358)	(0.644)
Observations	3930	2970	3930	2970
Y-mean	9.893	9.562	9.893	9.562
Y-sd	8.253	8.048	8.253	8.048
C. Post 2015 as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t$ × $Exposure_i$ (std)	0.325***	0.355***	0.420***	0.464*
	(0.146)	(0.175)	(0.134)	(0.240)
Observations	7960	5940	7960	5940
Y-mean	7.538	7.196	7.538	7.196
Y-sd	6.883	6.673	6.883	6.673
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: This table reports robustness checks restricting the sample to U.S. counties with strictly positive pre-2015 cocaine exposure ($Exposure_i > 0$). The dependent variable is the cocaine-related overdose death rate per 100,000 population. The key regressor, $Coca_t \times Exposure_i$, interacts Colombia's coca cultivation or production (or a post-2015 indicator) with each county's pre-2015 exposure to Colombian cocaine. Exposure measures are based on pre-shock proxies from the National Survey on Drug Use and Health (NSDUH) and the DEA's National Forensic Laboratory Information System (NFLIS), using drug-offense or cocaine-property crime shares. All models include county and year fixed effects; standard errors (in parentheses) are clustered at the county level. ***, **, * denote significance at the 1%, 5%, and 10% levels, respectively.

Appendix Table 5. Robustness: NIBRS-Reporting Counties, All Exposure Measures

Cultivation area as $Coca_t$				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.280**	0.253**	0.365***	0.336**
	(0.125)	(0.127)	(0.107)	(0.168)
Observations	11,340	11,340	11,340	11,340
Y-mean	7.538	7.538	7.538	7.538
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: This table restricts the sample to counties with positive NIBRS-derived cocaine exposure (i.e., counties that reported drug offenses in the NIBRS system during 2010–2014). The dependent variable is the cocaine-related overdose death rate per 100,000 population. The key regressor, $Coca_t \times Exposure_i$, interacts Colombia's coca cultivation with each county's pre-2015 cocaine exposure share. All four composite share measures produce significant results. All regressions include county and year fixed effects, population weights, and standard errors (in parentheses) clustered at the county level. ***, **, * denote significance at the 1%, 5%, and 10% levels, respectively.

Appendix Table 6. Robustness: Functional Form and Alternative Proxy for the Exposure Index

A. Multiplicative Index				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.387***	0.351**	0.472***	0.435**
	(0.131)	(0.136)	(0.104)	(0.177)
Observations	59,096	59,096	59,096	59,096
Y-mean	6.320	6.320	6.320	6.320
Y-sd	6.441	6.441	6.441	6.441
B. Additive Index				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.472***	0.466***	0.919***	0.918***
	(0.176)	(0.176)	(0.268)	(0.271)
Observations	59,096	59,096	59,096	59,096
Y-mean	6.320	6.320	6.320	6.320
Y-sd	6.441	6.441	6.441	6.441
C. Multiplicative Index with Perishables Proxy				
	Mortality rate (#deaths per 100,000)			
$Coca_t \times Exposure_i$ (std)	0.546***	0.530***	0.644***	0.619***
	(0.067)	(0.051)	(0.086)	(0.087)
Observations	59,096	59,096	59,096	59,096
Y-mean	6.320	6.320	6.320	6.320
Y-sd	6.441	6.441	6.441	6.441
County, Year FEs	Y	Y	Y	Y
Drug use measure	NSDUH	NSDUH	NFLIS	NFLIS
Crime measure	Drug offense	Cocaine property	Drug offense	Cocaine property

Notes: Each panel reports estimates using a different functional form for the exposure index. Panel A uses the multiplicative baseline (product of three standardized share components); Panel B sums the three components instead (additive index). Columns vary the drug-use data source (NSDUH vs. NFLIS) and the crime component (drug-offense share vs. cocaine property crime share), following the same column ordering as Table 1. Panels A and B include the Colombian population share; Panel C replaces it with the perishables proxy. All regressions include county and year fixed effects, population weights, and standard errors (in parentheses) clustered at the county level. ***, **, * denote significance at the 1%, 5%, and 10% levels, respectively. Panel C replaces the Colombian population share in the multiplicative index with a county-level Colombian-perishables import fraction, computed via gravity decay (bandwidth 500 km) from each customs district's headquarters county.