# Quantifying the Effects of Fentanyl Exposure Misinformation on Opioid Mortality

Eli Kochersperger – West Virginia University

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**Abstract**

I identify the impact of fentanyl exposure misinformation- namely, the erroneous belief that momentary, passive contact with the potent opioid fentanyl can be seriously harmful- on first responder behavior during overdose events, and on overall opioid-related mortality. I examine changes in opioid-related mortality following one particularly well-covered episode involving an Ohio police officer in 2017, wherein the officer appeared to experience an acute opioid overdose after touching what was believed to be fentanyl. Employing a synthetic differences-in-differences identification strategy, I find areas with greater media exposure to this misinformation exhibit marked increases in opioid overdose deaths; as well as preliminary evidence to suggest that overdose interventions performed by first responders were less effective and argue that this represents an increased hesitancy to render aid due to the potential presence of fentanyl. These results point to the existence of a heretofore unrecognized driving factor behind the current opioid epidemic, as well as to the need for policy intervention to counteract further dissemination of such adverse misinformation.

**Keywords**: Health Behavior; Illegal Behavior and the Enforcement of Law; Belief; Unawareness

**JEL Classification**: I120, K420, D830

*By causing fear and panic among such key partners in responding to the overdose crisis, we're putting people's lives at further risk and adding to the stigma around drug use.*

— Keith Brown, Katal Center for Health, Equity and Justice director, quoted in the *Times-Union* in 2018.

# 1 Introduction

In 2021, life expectancy at birth in the United States declined by 1.16%, from 77.0 to 76.1 years (Arias et al., 2022). These stark changes are overwhelmingly the result of excess COVID-19 deaths, but deviations from long-term trends can be traced back, at least in part, to shifts that first presented in the years preceding the pandemic. In the years 2015 and 2017, on the heels of a worsening opioid epidemic, life expectancy reversed a 25-year trend of year-over-year gains to decline by 0.2 and 0.1 years (Devitt, 2018), respectively; and for as much attention as the COVID-19 pandemic has duly received, it is worth noting the sobering statistic that it was in 2021 wherein annual drug-involved overdose deaths exceeded 100,000 persons for the first time. For perspective, of the 0.9 year decline in life expectancies in that year, approximately 16% (compared to COVID-19's 50%) can be directly attributed to changes in unintentional injury deaths, of which accidental drug poisonings largely constitute. Within just the 18-45 age group these effects are even more pronounced, where accidental overdoses involving synthetic narcotics (an estimated 90% of which are associated with the opioid fentanyl or its analogs) were the leading single cause of death, exceeding even suicide, car accidents, heart disease and cancer (Jones, 2023).

It is unsurprising in light of these statistics that many advocacy groups and policymakers have taken to treating fentanyl as a singularly unique threat to public health. From a pharmacological perspective this appears warranted: Fentanyl can be 100 times more potent than morphine, and 50 times more potent than heroin (Ramos-Matos et al., 2022). Considering the relative ease of access and low cost of the drug in illicit markets, fentanyl is also commonly used as an adulterant which has further exacerbated its lethality through the consumption by unwitting- and often opioid naive- users. Only trace, bordering on imperceptible quantities (2 mg) of the raw substance are needed to trigger fatal respiratory failure when used intravenously, which has prompted some advocacy groups to claim that "if you can see it, it can kill you."[1] However, like other synthetically produced opioids tramadol and carfentanil, fentanyl poses no significant acute health risk to individuals when exposed to the substance incidentally. Dermal contact and inhalation both require

[1]Quote pulled from Jackson County, Missouri's Community Backed Anti-Crime Tax (COMBAT) program website: https://www.jacksoncountycombat.com/818/Get-The-Fentanyl-Facts.

extremely prolonged exposure intervals to receive even clinical dosages (Moss et al., 2018), which all but eliminates the possibility of overdose from momentary contact. Nonetheless, sensational news stories detailing the supposedly-instantaneous lethality of the opioid, perhaps due in part to the embellished exposure risks promulgated by the DEA and other law enforcement agencies, have flourished (Beletsky et al., 2020). So persistent a media phenomenon has this become in fact, that public perceptions of fentanyl exposure hazards no longer align with the reality described by the clinical toxicology literature; and mere speculation on the presence of the narcotic within communities has elicited such outsized alarm as to be described as a form of moral panic (Ciccarone MD, 2020).

The question this study addresses is how these erroneous beliefs on fentanyl exposure hazards, in their near-ubiquity, have factored into the broader opioid epidemic and related overdose mortality. Previous research has succeeded in establishing the direct effect that fentanyl's introduction to illicit drug markets has had on mortality within the context of use behavior, but has largely neglected the potential influence of bystander and first responder perceptions. This is relevant because opioid overdoses are unique among accidental drug poisonings for their relative treatability with prompt medical intervention. The opioid antagonist naloxone (also known by the brand name Narcan), can safely resuscitate unresponsive victims, requires no specialty medical training to ensure its correct administration, and is widely available without prescription at low or zero costs. Critical however, is that these naloxone interventions require close proximity between overdose victims and those rendering aid. If a first responder or bystander had the means to save an overdose victim, but also incorrectly believed that they would be at personal risk of injury in doing so, any ensuing hesitancy could easily translate to death.

Drawing on restricted-use mortality data from the National Vital Statistics System (NVSS) for the years 2014-2019, this study examines the influence of misinformation shocks on opioid-related mortality through the most common dissemination medium: Media reports of claimed "near-death" experiences suffered by first responders involving fentanyl. In particular, I focus on a 2017 incident involving an undercover narcotics officer in East Liverpool, Ohio, who was hospitalized following brief exposure to what was believed to be fentanyl powder. Prior media analyses (Beletsky et al., 2020) have suggested that this was the seminal event in pressing the fentanyl exposure myth into the public imagination. As such, I exploit the unexpected proliferation and random spatial variation in media coverage of this event to estimate the association between misinformation dissemination and county-quarter opioid-related mortality rates. I find that within the media market local to East Liverpool, opioid related mortality increased significantly following the 2017 event when compared against bordering counties, and that these variations in mortality cannot be attributed to other

contemporaneous factors. Similar, though slightly attenuated effects are observed within other media markets across the country that featured reporting on the East Liverpool event. Moreover, I find evidence that regions with greater media coverage of this event demonstrate marked shifts in recorded death locations- away from hospitals, and towards other third locations- among opioid overdose victims, and argue that this is the result of a reluctance to render aid based on fentanyl hazards perceptions.

The principal contribution of this paper is in providing the first credibly-causal estimates for the economic consequences of the fentanyl hazards myth, as well as more generally advancing the literature on the role of misinformation in public health policy efficacy. Focusing on mortality specifically here is essential because it directly reveals the life-threatening impact of the epidemic and guides effective interventions to save lives. Although there is a rich body of qualitative work examining the potential influence of this misinformation on first responder behavior (Attaway et al., 2021; Beletsky et al., 2020; Del Pozo et al., 2021; Herman et al., 2020), with the exception of this study's companion article (Kochersperger, 2023), no research to date has examined the direct outcomes of such beliefs on public health outcomes. Understanding the broader influence of misinformation- especially that spread through social media- on public health outcomes has been of particular attention of late on the heels of a growing anti-vaccination movement (Chou et al., 2018; Wang et al., 2019). Within just the economics literature, for instance, Carrieri et al. (2019) employ a research design similar to that used here involving one, significant media-driven misinformation shock to derive causal estimates for its effect on vaccination rates. This study therefore bridges these two literatures and bolsters the descriptive results already generated on fentanyl hazards myth phenomenon with the application of causal inference methods.

The remainder of the paper is organized as follows. In the next section I offer some context on fentanyl hazards misinformation phenomena, including a summary of common first responder overdose protocols and origins of the myth to get at possible mechanisms. In Section 3 I describe my data and empirical strategy, and report my results in Section 4. I conclude with a discussion of these results and policy ramifications in Section 6.

# 2 Background

## 2.1 Fentanyl, naloxone, and first responder overdose protocols

Fentanyl is a synthetic piperidine-based opioid drug, meaning that unlike natural or semi-synthetic opioids such as morphine, heroin, or oxycodone, it is not derived from poppies. It was first developed by Paul

Janssen in 1959 as an effort to create what was then the most potent analgesic, believing that to do so would improve safety (Stanley, 1992). The drug first received US medical approval in 1968 and- in line with its considerable potency- has maintained a somewhat niche prescribing status when compared to other opioids. Primarily used for managing major pain, most fentanyl is prescribed to patients following surgery or during late-stage cancer. Among these patients it is particularly common to prescribe transdermal patches, which are adhesive strips that cling to a person's skin and are specially formulated to allow fentanyl to enter the bloodstream over prolonged periods.

Beyond these intended therapeutic uses, fentanyl has developed a complicated and deadly legacy. The fentanyl analogs carfentanil and remifentanil were implicated in the direct deaths of 125 hostages during the 2002 Nord-Ost siege, when Russian special forces piped aerosolized forms of the opioids into the Dubrovka Theater in an attempt to subdue Chechen resistance fighters (Riches et al., 2012). Domestically, dozens of poisoning deaths among children have been credited to transdermal patches for either their mistakened application (when believed to be a band-aid), or accidental ingestion through chewing (Stoecker et al., 2016). The most significant aspect to this legacy by far however, has been fentanyl's role in the illicit opioid epidemic. Between 2013 and 2020, the number of opioid-related deaths attributed to synthetic opioids increased by a factor of 18, advancing to the point of accounting for 82% of all opioid-related deaths in 2020 (Hedegaard et al., 2021). While short-comings in cause-of-death reporting keep the precise number of deaths resulting from specifically fentanyl use difficult to determine, drug seizure data from the National Forensic Laboratory Information System (NFLIS) suggest that 59% of all analgesics, and as much as 91% of non-Buprenorphine synthetic opioids seized by law enforcement contain fentanyl, an analog, or a chemical precursor used for its production (DEA, 2021). The reasons for this extraordinary change is multi-fold, but from the supply-side it's been largely driven by economic factors: Fentanyl is cheap to produce and its high potency allows for both easier cross-border movement, and cutting with other substances (Greenwood & Fashola, 2021).

Accompanying fentanyl's growing prevalence within illicit drug markets has been an increased interest among toxicologists in understanding the precise hazards the opioid poses through passive exposure. In their review of the extant clinical literature, Moss et al. (2018) find little evidence to corroborate the idea that momentary contact poses any significant health risk: The required duration of continued exposure to powderized fentanyl to achieve a therapeutic- let alone, toxic- dosage through inhalation is on the order of *hours*, not seconds. Moreover, dermal contact alone does not appear to be capable of permitting the absorption of fentanyl to the bloodstream.[2] In one recent noteworthy event, a first responder was exposed

[2]Moss et al. (2018) provide the following scenario to illustrate just how unlikely immediate reaction is:

If bilateral palmar surfaces were covered with fentanyl patches, it would take ∼14 min to receive 100mcg of fentanyl

to a large quantity of analytically-verified liquid fentanyl when it was splashed over their skin, but exhibited no clinical effects of opioid absorption (Feldman & Weston, 2022).

Numerous harm reduction policies have been advanced in an effort to combat the worsening opioid epidemic, but few have received as much attention as increasing the availability of naloxone. As an opioid antagonist, naloxone is capable of reversing respiratory depression from acute opioid intoxication within minutes of administration. Because of its life-saving capacity, it has been recognized as an 'essential medicine' by the World Health Organization. Since auto-injector and intranasal naloxone devices received medical approval for emergency use in 2014 and 2015, respectively, their use has expanded significantly and are now widely issued to emergency medical services, law enforcement, fire departments, and community health clinics.

Despite naloxone's demonstrated life-saving capabilities, questions remain on its broader efficacy in reducing opioid mortality. Empirical efforts at understanding the influence of increased accessibility of naloxone as a policy response to the opioid epidemic have mostly focused on changes to Naloxone Accessibility Laws (NAL). Rees et al. (2019) look at changes to both NALs and Good Samaritan Laws (GSL) and find that NAL adoption yields significantly negative effects on opioid mortality, but that these estimates are almost entirely driven by early-adopters, suggesting these treatments were probably endogenous responses. Conversely, Doleac and Mukherjee (2022) employ a similar research design and find evidence that naloxone access had no significant effect on opioid mortality. Erfanian et al. (2019) attempt to account for spillover effects across borders in regards to both opioid mortality and NALs by estimating a spatial Durbin model. They find NALs have very mixed results, but generally do not appear to significantly decrease mortality directly (though certain NALs yield positive and negative effects when examined in the aggregate with spillovers to neighbors). This lack of consistent or clear estimates for these potential effects highlights a common theme: Meta-analysis (Smart et al., 2021) of multiple literatures find that NALs have mixed, if only slightly-positive impacts on opioid mortality. The ambiguity here is often attributed, like other similar harm reduction policies (Packham, 2019), to offsetting moral hazard behavior (Doleac & Mukherjee, 2022), but my results here hint to the possibility of another attenuating factor. Simply making naloxone more available may be insufficient as a lifesaving measure if people are reluctant to use it.

To illustrate the role that naloxone plays in overdose situations, I will describe a typical scenario and the

> ...This extreme example illustrates that even a high dose of fentanyl prepared for transdermal administration cannot rapidly deliver a high dose.

That is, even when the entire surfaces of both palms are covered with patches, it still takes more than 10 minutes to receive a therapeutic dose. They note that these figures are unrealistic however, as they are "based on fentanyl patch data, which overestimates the potential exposure from drug in tablet or powder form in several ways."

standard protocols employed by those rendering aid. Firstly, note that the first responders to an overdose scene are often not emergency medical services (EMS), but law enforcement officers (LEO). Officer surveys and analysis of bodycam footage suggest that in the majority of cases LEO are first to the scene (Smiley-McDonald et al., 2022; White et al., 2022), sometimes beating EMS by several minutes. While there is some slight heterogeneity with this tendency in regards to urbanicity (Smiley-McDonald et al., 2022) (officers in rural regions report arriving around the same time as EMS), even in areas where not commonly first to the scene, LEOs are still more likely to administer naloxone than other responders when they are first (Macmadu et al., 2022). Because opioid-induced respiratory failure can cause death by brain hypoxia within a matter of minutes and responders are already operating off a time delay when arriving to a scene, LEOs often immediately administer naloxone then attempt CPR, so as to "buy time" before EMS arrives (Smiley-McDonald et al., 2022). Depending on victim response, first responders may administer multiple doses of naloxone; and if stabilized, they are typically either arrested, escorted to a hospital, or released at the scene.

Because of the likelihood of being in close proximity to narcotics, it has been recommended that first responders to suspected fentanyl overdoses dawn nitrile gloves and- when believed to be airborne- facemasks (Moss et al., 2018). While these recommendations do not differ materially from those made for any other drug overdose,[3] it has not stopped private industry from marketing specialty fentanyl personal protective equipment (PPE). These fentanyl-proof gloves, testing equipment, and hazmat suits have been adopted by some police departments (Herman et al., 2020), but have also been panned by toxicology experts as unnecessary (Lynch et al., 2018). Considering that the margin of time needed for an overdose to become lethal could be on the order of seconds, delaying needed aid to a victim to put on superfluous PPE has prompted calls to reconsider these practices and to relax even the standard recommendation for use of N95 respirators (Lynch et al., 2018; Winograd, Phillips, et al., 2020; Herman et al., 2020; Attaway et al., 2021).

## 2.2 The fentanyl panic and its origins

The principle vector through which fentanyl misinformation appears to be disseminated is media reporting on supposed exposure events, most typically those involving law enforcement officers. So prevalent have these media pieces become in communities hardest hit by the opioid epidemic that reported-on scenarios often follow a standard formula: Following an attempted drug possession arrest, an officer comes into contact with a powderized narcotic; through either the admission of the offender or just supposition, said officer comes to believe that this substance is fentanyl; after a period of several minutes the officer reports feelings

[3]The NIOSH recommendations are intended for when any illicit drugs are at an emergency medical scene and offer no additional considerations for fentanyl specifically. See: https://www.cdc.gov/niosh/topics/fentanyl/risk.html

of dizziness, shortness of breath, and may even faint; in an attempt to resuscitate the exposure victim, other officers or first responders may administer naloxone or escort them to a hospital to receive treatment. Affected first responders, their peers, and accompanying media portrayals may attest these reactions to acute opioid toxicity, but even if one were to disregard the extreme unlikelihood of passive fentanyl exposure eliciting such medical responses, there is virtually no evidence to corroborate the veracity of these claims (Lynch et al., 2018; Herman et al., 2020; White et al., 2022). Herman et al. (2020) combed through more than one-thousand media reports involving supposed first responder fentanyl exposure events between 2014-2018 and could not find a single instance where either the affected parties reported a plausible poisoning scenario or laboratory testing confirmed poisoning. Instead, they find that the most commonly reported symptoms are consistent with stress-induced panic, and that these reactions are probably psychosomatic in origin.

Although their underlying accuracy is disputed, the media presence first responder fentanyl exposure events maintain is far from trivial. Estimates on the upper-bounds of cumulative facebook user-views that these media reports have received between 2015 and 2019 is approximately 70 million, while only 6.6% of these shares correspond to articles that correctly refute the incidental exposure hazards (Beletsky et al., 2020). Accordingly, surveys suggest that knowledge of these erroneous exposure hazards have permeated aggressively through the first responder community, with as many as 80% of queried law enforcement and emergency medical services members agreeing that momentary contact with fentanyl can be deadly (Persaud & Jennings, 2020; Del Pozo et al., 2021; Attaway et al., 2021; Berardi et al., 2021; Bucerius et al., 2022). Of those law enforcement officers who echoed these sentiments, many note that they had learned of the phenomenon second-hand and not through formal police channels, suggesting that media coverage may be a contributing factor for first responder perceptions specifically (Attaway et al., 2021). Moreover, these beliefs also appear to translate directly to first responder behavior, with some law enforcement admitting to an unwillingness to render first aid to those they suspect of suffering from fentanyl poisoning (Berardi et al., 2021; Bucerius et al., 2022).

The valid hazardous concerns of fentanyl as an accidental poisoning agent can be traced to historical events with relative ease, yet the origins of the fentanyl exposure hazards myth is somewhat more opaque: Urban legends of malefactors clandestinely dosing unsuspecting highway patrolmen date back to at least the 1970's[4]; and parallels have been noted to earlier, similarly specious medical panics regarding first responder exposure misinformation during the HIV/AIDS epidemic[5] and early waves of clandestine methamphetamine

[4]See "LSD Given to Police Officer" at Snopes here: https://www.snopes.com/fact-check/jar-jar-drinks/

[5]One ambulance service director in New York State, who had worked as an EMT during the HIV/AIDs crisis made this

lab raids the 1980's and 90's[6] (Bucerius et al., 2022). What is known for certain is that beginning in 2016, medical toxicologists began receiving inquiries concerning the veracity claims made in the media that fentanyl could harm first responders on touch (Herman et al., 2020). In that same year, the US Drug Enforcement Administration, published a press release describing one exposure event involving law enforcement in New Jersey, the details of which were shared further by the National Police Foundation (Del Pozo et al., 2021).

Beyond the influence of these agency press releases, social and news media analysis performed by Beletsky et al. (2020) pinpoint one heavily-reported event as being the primary culprit in cementing these ideas within the public consciousness. While attempting to make an arrest in May 2017, an East Liverpool, OH police officer was exposed to what was believed to be fentanyl. Within minutes he became lightheaded and naloxone was administered several times, but his symptoms were severe enough to eventually require hospitalization. Media coverage of the event was swift, with early news reports receiving tens of thousands of facebook shares (Beletsky et al., 2020). As illustrated in Figure 1, average search prevalence among Google queries for terms related to fentanyl exposure increased by a factor of five immediately following this particular event. Similarly, in Figure 2 I compare search prevalence for all queries involving the term "fentanyl" across time within the Youngstown, OH Designated Market Area (DMA) (where East Liverpool is located) against neighboring DMAs, as well as all other DMAs in the surrounding states of Pennsylvania, Ohio, and West Virginia. One can observe that in the media market local to East Liverpool, general interest in fentanyl peaks immediately following the 2017 event, before returning to similar search frequencies of the neighboring areas. This is consistent with Beletsky et al. (2020)'s observation that media coverage of this event- despite its relative popularity- varied substantially across space. Even as more events such as these would unfold involving LEOs from all over the country, the East Liverpool event remains unique in terms of both its timing, and ultimate breadth of coverage.

In 2018, the American College of Medical Toxicology (ACMT) and American Academy of Clinical Toxicology (AACT) released a joint statement to counter the sensationalist claims which had been made in the preceding two years (Moss et al., 2018), but this effort appears to have largely fallen on deaf ears. Since then, hundreds more articles have been published detailing claimed exposure events (Beletsky et al., 2020),

comparison more overt, saying (Bump, 2018):

> It was a lot of hype ... We didn't understand it, we didn't know how it was transmitted, and I think we're seeing the same thing here. But the reality is, the initial scares about exposure to this drug just have not panned out.

[6] In a similar fashion to specialized fentanyl PPE, beliefs about long term complications related to meth lab exposure among retired law enforcement officers prompted the Utah state government to finance a controversial therapy regimen in 2007. The sauna-based therapy (which was developed by L. Ron Hubbard and delivered through a Church of Scientology-associated organization (Scientology Critical Information Directory, 2009)) claimed to "sweat out" toxins, though this was criticized for having little to no scientific basis (Bonisteel, 2015).

with the phenomenon being reported as recently as Summer 2023.[7]

# 3 Empirical Approach

## 3.1 Data

To observe the effects of fentanyl misinformation on opioid mortality, I utilize Nielsen's Designated Media Markets (DMA) to identify treated counties within the same media market as East Liverpool, OH. As argued above, this May, 2017 event appears to be the pivotal event in establishing public misconceptions on the hazards of passive fentanyl exposure, so distinguishing between regions based on their exposure to misinformation through the media would permit one to identify treatment effects. DMAs are very similar to the Federal Communications Commission's (FCC) Television Market Areas (TMA), the legally-defined borders that determine broadcast rights and channel availability for all over-air, satellite, and cable television. TMAs borders are usually larger than actual media coverage areas (particularly in mountainous regions like Appalachia) however, so Nielsen DMAs are adjusted to only include counties with significant metered-viewership. I hand-code these DMAs based on a publicly available map[8]. Because there still remains the potential for some bordering counties to be treated by broadcast misinformation, I additionally employ FCC significant viewership data to include any counties that could be plausibly subject to spillovers[9].

To describe mortality effects, I utilize the CDC's restricted-use multiple cause of death file. These data include the entire universe of deaths within the United States over the period 2014-2019 and are recorded at the individual-level. Following the procedures outlined by Svetla et al. (2015), I identify all overdose deaths as those with ICD-10 underlying cause of death codes X40-X44, X60-X64, X85, Y10-Y14; then identify opioid overdose deaths from these as those with with mortality-associated conditions codes T40.0, T40.1, T40.2, T40.3, T40.4, or T40.6[10] I aggregate these opioid overdoses and compute the per 100,000 population death rate by the county-quarter and initially drop any counties which did not record a single opioid overdose over the six year sample. To account for potential undercounting of opioid overdoses, I also use aggregated counts of overdose deaths which include the mortality-associated conditions code T50.9 for poisoning by unspecified drugs, medicaments and biological substances (Buchanich et al., 2018). As an additional robustness check,

[7] See news article, *Florida sheriff: Deputy exposed to fentanyl, saved by Narcan*, here: https://www.newsnationnow.com/crime/fentanyl/florida-sheriff-deputy-exposed-to-fentanyl-saved-by-narcan/

[8] Available here: https://web.archive.org/web/20230315182138/https://thevab.com/storage/app/media/Toolkit/DMA_Map_2019.pdf

[9] The FCC is legally obligated to conduct periodic viewership surveys to determine which specific channels receive significant viewership outside of their designated TMAs. I use the 2017 survey, which is available from here: https://transition.fcc.gov/mb/significantviewedstations061817.pdf

[10] These T-codes correspond, respectively, to: opium, heroin, natural and semisynthetic opioids, methadone, synthetic opioids, and other or unspecified opioids.

I perform placebo tests employing similar mortality rates for motor vehicle accidents, heart attacks, and assault excluding the use of drugs or medicants[11]

Additional control covariates include county-quarter demographic and economic measures, as well as opioid-use proxies such as the annual opioid dispensing rate and heroin arrest rates, and policy indicators for naloxone access, good samaritan, and opioid prescription control laws. One likely confounder which may prevent the interpretation of any naive regression results as causal is the significant county-level variation in fentanyl and heroin prevalence over the observed time frame. Broader historical analyses of the opioid epidemic have emphasized two consumption innovations– the transition from prescription painkillers to heroin, and eventually from heroin to fentanyl– as epochal in defining the associated mortality. There is particular concern over the market transition towards primarily-synthetic opioid consumption: Because the introduction of fentanyl was so swift, disparate, and ultimately lethal, the potential for spuriously conflating those associated outcomes with media misinformation treatment effects seems a valid concern. To control for these confounding effects then, I also utilize law enforcement drug seizure data collected from Harm Reduction Ohio, which includes the entire universe of Ohio's Bureau of Criminal Investigation's (BCI) crime lab results for the years 2014-2019. A complete breakdown of controls employed, their sources, and spatio-temporal coverage is available in Table 1.

Unlike user surveys, which are largely dependent on the word of dealers in establishing the provenance and composition of traded goods, the precise chemical makeup of seized samples are determined through a gas chromatography process, and as such much less likely to omit or mistake the presence of specific opioids. The BCI laboratory is also by far the largest crime lab within the state– of Ohio's 88 counties, only two are absent for the years observed. These data contain individual offense-level observations (including seizure data, arresting authority, and county of seizure location) and the corresponding chemical makeup of any drugs seized, which represents a significant improvement over opioid possession or intent-to-distribute arrest records. I extract from this dataset the total county-quarter counts of seizures which tested positive for fentanyl, or the closely related carfentanil, and do the same for heroin[12].

Summary statistics for key variables of interest and covariates are listed in Table 2. To align with my primary identification approach, I separate between columns 1 and 2 the statistics corresponding to the treated counties (those within the Youngstown DMA), and control counties (those within a DMA that shares

[11]For motor vehicle accident deaths, I use all underlying cause of death codes corresponding to unintentional motor vehicle deaths: [V02-V04](.1,.9), V09.2, [V12-V14](.3-.9), V19(.4-.6), [V20-V28](.3-.9), [V29-V79](.4-.9), V80(.3-.5), V81.1, V82.1, [V83-V86](.0-.3), V87(.0-.8), V89.2. For assault-related deaths, I use all codes contained under X86-99 and Y00-Y05, which includes all forms of assault, excluding assault by drugs, medicaments and biological substances. For heart attack-related deaths I use all codes contained under I10-15, hypertensive diseases; I20-25, ischemic heart diseases; I46, cardiac arrest; and I50, heart failure.

[12]Because fentanyl is almost universally used as an adulterant of heroin or psychostimulants, it is worth noting that these fentanyl and heroin counts are not exclusive.

a border with the Youngstown DMA). One can observe that average mortality within the treated counties is greater than their neighbors. To expand on this, in Figure 3 panel *a* I plot a time series of average county-month opioid overdoses for both the treated counties and their direct neighbors (that is, only the *counties* that share a border with the treated), and all other counties in Ohio, Pennsylvania, and West Virginia. Because of the relative ruralness of these treated counties, direct neighbors are probably more appropriate baselines for comparison here. The parallel trends in mortality prior to treatment, and divergence afterwards appear to lend credence to the media exposure hypothesis, although the general stabilization or decline in mortality is somewhat unexpected. In panel *b* I plot the time series for the county-month average percentages of drug seizures containing heroin and fentanyl for the entire state of Ohio. These plots demonstrate the importance of including opioid type prevalence measures, as fentanyl overtook heroin in ubiquity at almost precisely the same time as the East Liverpool event. Omission of such controls could spuriously inflate derived estimates if fentanyl was more lethal, and differentially distributed among treated and control areas.

## 3.2 Identification Strategy

My primary identification approach is to look at opioid mortality within the Youngstown Ohio DMA and compare this against the opioid mortality in some combination of bordering counties before and after the 2017 East Liverpool event. Because East Liverpool lies squarely within the Youngstown media market, and because both the timing and location of the event are seemingly random, the identifying assumption is that any difference in overdose mortality trends between counties within and outside this market can be attributed to behavioral changes among first responders due to the difference in exposure to the corresponding media coverage. In examining this particular exogenous media shock, I exploit random changes in public perceptions of the fentanyl exposure hazards to identify the causal effects on first responder behavior. The counterfactual here would be that, absent some media intervention, individuals would not erroneously believe fentanyl to be so hazardous as to stymie or delay emergency response to overdoses. This is essentially the canonical differences-in-differences (DiD) research design, but I use additional data-driven methods to create a better match on pre-treatment observables,

To describe this procedure in more detail, consider a conventional approach at deriving these misinformation treatment effects using the following two-way fixed effects (TWFE) model:

$$y_{ct} = \mu + \alpha_c + \beta_t + \tau \cdot \mathrm{MI}_{ct} \tag{1}$$

where $y_{ct}$ is the opioid-related mortality rate in county $c$ in quarter $t$; $\alpha_c$ and $\beta_t$ are county and quarter

fixed effects, respectively; and $\text{MI}_{ct}$ is an indicator equal to one representing misinformation exposure for all counties within the Youngstown DMA following the May 2017 treatment, and zero otherwise. Because this model specification holds the composition of intervention and comparison groups stable, and assuming that treatment assignment is not itself endogenous with opioid-mortality, then $\tau$ here can be interpreted as a causal average treatment effect on the treated as long as the parallel trends assumption is satisfied. A common critique with this standard DiD approach however, is that the validity of the parallel trends assumption cannot be formally tested, meaning that *ad hoc* control definitions could be yielding spurious results. Considering the relatively small size of the treated sample examined here, this is particularly threatening to the causal interpretation as the control group could be misspecified.

With this in mind, I instead estimate a synthetic differences-in-differences (SDiD) model that minimizes the following error:

$$\underset{\alpha,\beta,\mu,\tau}{\arg\min} \left\{ \sum_{c=1}^{C} \sum_{t=1}^{T} \left(y_{ct} - \mu - \alpha_c - \beta_t - \tau \cdot \text{MI}_{ct}\right)^2 \hat{\omega}_c^{\text{sdid}}\, \hat{\lambda}_t^{\text{sdid}} \right\} \tag{2}$$

where $\hat{\omega}_c^{\text{sdid}}$ is a vector of statistically-derived county control weights, and $\hat{\lambda}_t^{\text{sdid}}$ is a vector of time weights computed according to Arkhangelsky et al. (2021). The SDiD control generated by these weights minimizes the error in pre-treatment trends when compared with the treated, so that it represents a more realistic counterfactual than any *ad hoc* specification. Arkhangelsky et al. (2021) describe this as a "generalized" differences-in-differences model, because- unlike the canonical DiD which assigns uniform weight to each pre-period control- SDiD weights those control observations which best construct parallel pre-trends. Because of this strength, SDiD method has been demonstrated to generally outperform both conventional TWFE and SDiD control estimation approaches (Arkhangelsky et al., 2021). In addition to these approaches, I estimate a staggered-SDiD that considers all counties within DMAs that had at least one news article published making reference to the East Liverpool event as treated, and their bordering DMAs as controls. This is a similar identification approach to the primary method, but permits DMAs other than just Youngstown to be treated and uses variation in timing of media coverage.

A growing concern among applied researchers with interpreting either conventional synthetic control or SDiD estimates derived from observations of the dependent variable alone as causal is that the asymptotic irrelevance of auxiliary covariates may not necessarily hold over finite sample spaces. While the recommendation is often overlooked, Abadie et al. (2010) and others (Kaul et al., 2022) propose the inclusion of potential confounders whenever researchers find them relevant. Recent simulation results indicate that omitting these confounders in model specifications could not only introduce significant bias to estimates but also render the precise direction of this bias virtually unknowable from the outset (Pickett et al., 2022). As such, when

possible I duplicate all estimation procedures with and without the inclusion of plausible confounders, and display both sets of results.

To utilize contemporaneous observations of relevant covariates within the synthetic differences-in-differences estimation, Arkhangelsky et al. (2021) propose first regressing the dependent variable on the covariates, then running the SDiD procedure on the obtained residuals. Kranz (2022) demonstrate however, that in instances where covariates have time-varying influence on the dependent variable, this residuals approach often fails at constructing a SDiD control which satisfies the parallel trends condition. They instead suggest a correction approach that utilizes fitted values for the dependent variable derived from a two-way fixed effects regression including the covariates. Because several important control variables within my model- in particular, those pertaining to opioid demand- likely differ in their influence on opioid-related mortality across time,[13] I opt to utilize this 'projected' covariate approach when running my regressions.

Lastly, it is important to acknowledge that- while initially untreated- the control counties are susceptible to contamination over time as information spillovers occur through social and national media coverage of later, similar misinformation-triggering events. It is unlikely that my model is capable of entirely controlling for spillovers. Instead, I opt to place particular emphasis on examining dynamic trends in mortality for the bulk of my analysis: If delineating between treated and untreated counties becomes more difficult as time passes, I should still be able to observe any eventual convergence between the treated and controls.

# 4 Results

## 4.1 Primary Results

The primary results of my regressions are outlined in Table 3. As detailed already, I estimate SDiD models which assume all counties within the Youngstown DMA are treated through exposure to misinformation following the coverage of the East Liverpool event, while donors to the SDiD control are drawn from the immediately bordering counties to this treated DMA. In the upper panel, I estimate SDiD models both with and without auxiliary covariates using several measurements for accidental drug poisoning. These estimates comprise of all drug-related poisonings; opioid-related poisonings; possibly-opioid-related poisonings which include all opioid-related deaths, as well as those coded as related to unspecified drugs; illicit opioid-related poisonings (those associated with either heroin or a synthetic opioid, like fentanyl); and synthetic opioid-

[13]For instance, I employ possession and distribution arrests data as a proxy for illicit opioid demand, but these data do not distinguish between opioid types. Since the observed timeframe also coincides with the transition from predominantly heroin to fentanyl use, parametrizing this demand effect as constant while there are unobserved changes in illicit opioid lethality could bias pre-trends.

related poisonings. In the lower two panels I re-estimate my opioid-related SDiD model, but stratify by decedent demographics and age groups.

Summarizing these results, I observe large and statistically-significant increases to overdose mortality rates within the Youngstown DMA following the East Liverpool event. Across specifications both with and without the inclusion of control variables, I observe overall accidental drug poisoning and opioid-related poisonings are increasing by between 2-4 additional deaths per 100-thousand population. My preferred outcome measure for this analysis, opioid-related mortality, increased by approximately 2.84 deaths per 100-thousand population. This jump in mortality represents a 56.6% increase to the average quarterly mortality for the treated counties relative to pre-treatment period. The results for possibly-opioid related, illicit opioid-related, and synthetic opioid-related overdose deaths highlight data quality concerns that have been voiced by other researchers. It appears likely that- consistent with earlier findings (Buchanich et al., 2018)- many illicit opioid-related overdose deaths are being coded under this general "unspecified drugs" category. A lack of adequate toxicology screening, particularly early on when fentanyl first entered illicit drug markets, may have failed to correctly identify synthetic opioids when they were in fact present. While I cannot be certain that all of these deaths represent fentanyl, or even opioid-related deaths, concern for categorical undercounting here seems valid. Because of this, caution is warranted in interpreting these more drug-specific mortality coefficient estimates, as they are almost certainly biased downward. More reassuringly however, the stratified SDiD results illustrate sensible heterogeneity in changes to opioid-related mortality. Consistent with other research on the opioid epidemic, the bulk of these effects are being driven by white males between the ages of 25-34.

To better illustrate the dynamic trends of this phenomenon, I plot time series of the observed treated opioid-related mortality against the computed counterfactuals for the estimated opioid-related mortality SDiD model in Figure 5. Interestingly, opioid mortality appears to decline immediately following treatment for all counties, but critically, the treated counties rebound much more quickly. This could be representing an incubation peroid for the misinformation to disseminate and take hold.

One potential confounder missing from these model specifications is the variation in the ubiquity of particularly potent illicit opioids, fentanyl and heroin, over the observed period. Prior research examining Ohio over this same timeframe (Peterson et al., 2016; Zibbell et al., 2022) has identified the regional prevalence of fentanyl as a significant driver of opioid mortality, so the inclusion of some measure of this within the estimated model appears justified. By employing drug seizure data from Ohio's Bureau of Criminal Investigation's crime lab (BCI), I use county-quarter counts of total seizures that tested positive for these

compounds to proxy for their prevalence within local drug markets. A problematic factor with using these direct seizure counts is that they could conceivably be endogenous with opioid deaths.[14] As an alternative then- and since I am only interested in the *relative* prevalence of these opioids- I divide these drug-specific counts by the total count of all drug seizures conducted within that county-quarter. To test whether these proportional estimates are endogenous, I estimate a series of simple two-way fixed-effects models where I regress total drug seizures, fentanyl as percent of seizures, and heroin as percent of seizures on the one-year lag of opioid deaths. The results for these models are listed in the upper panel of Table 4, but to summarize: As anticipated, deaths do appear to be significantly increasing the number of seizures performed in the subsequent years, while the relative proportions of these seizures being either fentanyl or heroin do not seem to affected.

With these prior results in mind, I attempt to control for opioid prevalence variation by including the proportional measures of fentanyl and heroin ubiquity as additional covariates and re-estimate my primary SDiD model specification on the subsample of Ohio counties. While not a direct threat to the validity of my reduced form estimates, a concern with interpreting these results could be a misidentification of the underlying mechanisms. For instance, it may be the case that these misinformation shocks are increasing opioid-related mortality, but are doing so by increasing consumer demand for fentanyl by users. To test this, I replicate my Ohio SDiD results twice more, but with fentanyl and heroin prevalence on the left-hand side.

The results for the three Ohio models are listed in the lower panel of Table 4. In column 1, I note that even with the inclusion of fentanyl and heroin drug seizure proportions, the primary specification SDiD results within Ohio do not significantly change. In columns 2 and 3, I see that when treating fentanyl and heroin prevalence as the dependent variable, there is no significant change following the East Liverpool misinformation shock. This highlights that changes to opioid overdose death are likely not arising from an increase in demand following media reports on fentanyl's potency. Taken together, these results strengthen the central argument that these observed changes to opioid-related mortality are being driven by some external factor other than fentanyl, or even heroin prevalence.

## 4.2 Robustness Checks

An initial concern with my estimates is that cluster bootstrap-derived standard errors are less dependable for small treated sample sizes. Because I have only four treated counties, I re-estimate my primary SDiD spec-

[14]This could arise, for instance, when a year of unexpectedly high opioid-related deaths within a county prompts local policymakers to invest more heavily in drug enforcement, and consequently sees an increase in seizures performed in the subsequent years.

ifications, but instead employ the placebo protocol outlined by Arkhangelsky et al. (2021). This approach is similar to permutation tests performed in randomization inference used for conventional DiD estimators (Conley & Taber, 2011): To directly estimate the noise level of the control units, a number of controls are randomly assigned as treated and the SDiD model is re-estimated on the donor set alone. Assuming homoscedasticity across units, this variance estimator would provide more accurate- if also more conservative- confidence bounds for the causal treatment effect. I perform this placebo procedure using 500 random placebos and plot the empirical distribution for their derived SDiD coefficients in Figure 6. Across specifications both with and without the inclusion of covariates, I find my initial SDiD estimates for opioid-related mortality retain their 99% significance level .

I re-estimate my opioid-related mortality model with considerations for a spate of other potential threats to validity and list the results in Table 5. These variations include an alternative treatment specification meant to control for information spillovers that includes any counties the FCC has listed as having significant viewership of any stations within the Youngstown DMA; alternative dependent variables of opioid-related death counts in levels and logs derived from the inverse hyperbolic sine transformation, rather than mortality rates; and a conventional DiD model. In the top panel I estimate these across all observations for the period 2014-2019, while in the bottom panel I re-estimate the opioid-related mortality model on the subset of observations occurring after the October 2015 adoption of the ICD-10 coding system to account for any potential data inconsistencies. For each of these, I experiment with several different donor-set specifications to derive my SDiD controls. Under my preferred specification in column 1, donors to the SDiD control are drawn from the immediately bordering counties to the treated DMA; under the specification in column 2, I expand this donor set to include all counties of bordering DMAs; and in column 3 I include all counties in bordering DMAs but exclude immediately bordering counties so as to control for spillovers. In Table 6 I replicate these results, but use local commuter zone delineations from Fowler and Jensen (2020) instead of DMAs. Under the primary donor set specification, the magnitude of the coefficients listed in column 1 are consistent with the preferred SDiD estimates, and are broadly significant. DiD estimates are qualitatively similar to SDiD, but are insignificant, which highlights the potential advantages that this more generalized estimation approach affords. Coefficients generally maintain their magnitudes across the wider donor set definitions in columns 2 and 3, but are noisier.

To better understand these results, I plot the three SDiD controls based on the different donor set specifications from row 1 of Table 5 in Figure 7. One can observe that the generated SDiD controls are nearly identical across these specifications, but that the alternative donor set definitions including more

counties from the bordering DMAs are weighting earlier observations from the pre-period more heavily (represented by the shaded regions in the bottom-left). It is primarily because of these differing time weights- *not* the composition of the SDiD control- that the alternative donor set specifications are yielding smaller, noisier treatment effect estimates. SDiD time weights are assigned by minimizing the error between pre- and post-treatment observations of the dependent variable on all controls (that is, it affords more weight to pre-treatment periods which are better predictors of post-treatment control outcomes). If the donor set contains controls which are wholly inappropriate for construction of the SDiD control, the unit weighting algorithm would assign them low weights when estimating treatment effects; but because the time weighting algorithm is applied across *all* members of the donor set, then these invalid controls are receiving the same weight as any other. Therefore, when appropriate and invalid controls follow different time trends, the vector of generated time weights is probably biased away from being the best predictor of relevant post-treatment outcomes.

Put differently, the inclusion of control counties which are qualitatively different from those within the treated DMA could be excessively weighting early-period observations if these inappropriate control counties report flat or declining opioid mortality rates, rather than the wider increasing trend. One way of possibly identifying this biasing effect is to examine covariate balance on the SDiD-weighted controls against the treated counties. The reasoning behind this is that a donor set with better balance in terms of observables linked to opioid mortality should result in a SDiD control that more accurately mirrors an ideal counterfactual. I perform a series of covariate balancing tests for each of these three donor set specifications and list the results in Table 7. I find that for all of the examined covariates except subprime credit score percentages, opioid dispensing rates, and the opioid prescription restriction policy indicator, my primary specification demonstrates a greater balance than the alternative donor sets. This is intuitive when considering Youngstown's locale: As the radius of counties included within the donor set is expanded outward, it begins to encroach on the denser, more urban Cleveland, Akron and Pittsburgh metropolitan areas. Nevertheless, the magnitude of the alternative SDiD estimates do not vary substantially, so taken together with these other considerations I retain the initial, adjacent counties specification as my preference.

Because my principal identification strategy considers only one relatively small media market as treated, it is possible that these results could be driven by some unobserved change to the underlying first responder mechanism other than the misinformation effect that I describe. For instance, it could be that counties within the Youngstown DMA experience similar changes in law enforcement or EMS staffing and response policies that incidentally coincide with the East Liverpool event. However unlikely, in such an instance my

estimates would be sizably biased upwards. To descriptively analyze this possibility, I collect municipal- and county-level expenditures data on police protection, fire prevention, and health services spending from the Census' Annual Survey of State and Local Government Finances[15]. I plot time series for per-capita spending in Figure 8 and compare the expenditures made within Youngstown OH DMA against those made by governments elsewhere in Ohio, Pennsylvania, and West Virginia. I observe no substantial relative trend changes within the Youngstown OH DMA following the East Liverpool event. Because of the inconsistency in spatial coverage for these data however (not all local and county governments surveyed in every year), I would emphasize that these figures are only suggestive.

To address this issue then, I estimate three additional model sets identical to my preferred specification that instead use motor vehicle accidents, heart attack-related, and assault-related death rates as the dependent variable. The rationale here is that if there is some alternative factor influencing first responder behavior, one would be able to observe similar changes in other common forms of death where mortality is subject to these agents' behavior (that is, other causes of death where lives can be saved with timely intervention by LEO and EMS). If not however, these regressions would act as falsification tests and return null results. The results of these falsification tests are outlined in Table 8, and as expected, motor vehicle, heart attack and deaths show no significant variation within the Youngstown DMA compared to control areas.

## 4.3 Mechanisms Analysis

### 4.3.1 Identifying media's direct role in misinformation shocks

Though there is evidence to corroborate the claim that the East Liverpool event influenced opioid mortality, it is still unclear how precisely this occurred. I have assumed up to this point that media coverage is the primary driver, but social media and word-of-mouth are equally-plausible vectors for misinformation. To test this, I employ a slightly different treatment definition which utilizes fentanyl misinformation media coverage data collected by Beletsky et al. (2020) to identify the associated effects of mass media reporting on mortality. These data collected from the Mediacloud system cover archived news articles for the period 2015-2019 which contain various combinations of phrases indicating the presence of erroneous fentanyl exposure hazards information. [16]

[15]Depending on the specifics of local and county government program structure, outlays for first responder services could appear in any one of these categories. Generally, since ambulance services are largely operated as private entities in rural areas, most government expenditures for emergency medical training and equipment will appear as either police protection or fire prevention expenses

[16]Beletsky et al. (2020) manually confirmed the content of each entry as misinformation and code for each observation the date of publication, location of publisher, associated event (for instance, whether the article makes explicit reference to the East

I estimate these media-exposure models employing the staggered SDiD method outlined by Arkhangelsky et al. (2021) in their appendix. This approach separates treated groups by their treatment date, estimates an SDiD model for each treated group on the pooled control units (while excluding the other treated units), then generates the average treatment effect on the treated (ATT) as a weighted average of each sub-group's ATT according to their relative proportion of post-treatment observations. The treatment here is defined as whether a given county's DMA has originated some media coverage prior to the observed date, while controls apply the methodology of my primary estimates and are drawn from a donor set of all directly adjacent counties to treated DMAs. Because these models are being identified on regional variation in reporting alone and include time fixed effects, these estimates would correspond to only the influence of local, rather than national reporting by media outlets on opioid mortality. Following (Packham, 2019), I increase this restrictiveness when performing this national-scale analysis to include only counties which recorded at least one opioid overdose for each year in the sample. A map of the misinformation-originating counties, their DMAs and neighboring DMAs based on the Beletsky et al. data is depicted in panel (b) of Figure 4.

The results of my media exposure staggered-SDiD are listed in Table 9. In column 1, I use the complete set of all treated DMAs, while in column 2 I exclude the Youngstown OH DMA to account for potentially-biasing local misinformation vectors (e.g. word-of-mouth deriving from those involved or otherwise familiar with the East Liverpool event, absent any media coverage). Though attenuated downward relative to estimates which consider only the Youngstown OH DMA as treated, opioid-related mortality rates demonstrate significant increases within media markets following reporting on the 2017 East Liverpool event. Under the preferred staggered-SDiD specification which excludes the Youngstown OH DMA and includes auxiliary covariates, I observe an increase of 0.448 opioid-related deaths per 100 thousand people, or an increase of approximately 12%. Even when restricting the treatment definition to regional media reporting, these specific misinformation shocks are consistently increasing opioid-related mortality at a substantial level.

#### 4.3.2 Identifying changes to first responder behavior

Understanding how these fentanyl misinformation shocks actually translate to changes in mortality remains an open question. If the mechanism pathway that I have already proposed is valid, than I should be able to examine direct changes to first responder behavior- in particular naloxone administration rates- within the treated regions. Unfortunately, naloxone administration data within Ohio for much of the observed time

Liverpool or some other first responder incident). I extract from these data all coverage relating to the East Liverpool event nationally and geocode each observation to their corresponding DMA.

frame is incomplete. Most notably, naloxone administrations performed by law enforcement are conspicuously absent from the extant data. Additionally, changes to community programs that train and distribute naloxone kits could be affecting their use more than even misinformation.

Though only descriptive, I collect from the Ohio Department of Health data for naloxone trainings, distributed kits, and self-reported naloxone resusitations performed by laymen and plot their time series in Figure 9. These figures are suggestive only as there is no way to know whether the trained individuals or distributed kits actually remained within the recorded counties. Similarly, resusciations performed by laymen are almost certainly undercounted. That being said, naloxone distribution and trainings do not appear to significantly differ between the Youngstown OH DMA and the remainder of Ohio counties. Naloxone administrations performed by laymen may be affected by misinformation shocks by staying persistantly low relative to the rest of the state, but there is essentially no data for the pre-period with which to make this claim credibly. I additionally collect and plot data for naloxone administrations performed by EMS from the Ohio Department of Public Safety, but similarly find no significant changes between treated and untreated regions. Following the observations from Kochersperger (2023), it seems likely that if naloxone use is changing substantially among first responders, this is probably most pronounced among unobserved law enforcement officers.

As an alternative approach at concretely describe the underlying behavioral mechanisms that are driving this change in mortality, I examine changes in death locations. If perceptions of the hazards of fentanyl exposure are discouraging the timely administration of aid, then the number of opioid overdose deaths recorded within hospitals would decline in treated relative to untreated areas. I estimate this directly with the following linear probability model (LPM):

$$\begin{aligned} Y_{c,i,t} =& \beta_{11,i}\text{OD}_i + \beta_{12,i}\text{Post}_t \times \text{OD}_i + \beta_{13,i}\text{Post}_t \times \text{OD}_i \times \text{Treat}_c \\ &+ \delta X_i + \gamma_{c,t} + \varepsilon_{c,i,t} \end{aligned}$$

where $Y_{c,i,t}$ is a dichotomous outcome variable representing whether or not individual $i$, residing in county $c$ that died in month $t$, has their listed place of death as being in one of five places[17]: inpatient hospital setting, outpatient or emergency room hospital setting, dead on arrival to hospital, home or residence, and other. $\text{OD}_i$ indicates whether the cause of death is attributed to an opioid overdose, which when interacted

[17]Although I only include five here, the CDC MCOD file includes eight possible values that this location of death variable can take: Hospital, clinic or medical center - inpatient; hospital, clinic or medical center - outpatient or admitted to emergency room; hospital, clinic or medical center - dead on arrival; decedent's home; hospice facility; nursing home/long term care; other; and place unknown. The omitted locations are excluded due to low counts.

with the $\text{Post}_t$ and $\text{Treat}_c$ dummies imply a triple-differences identification approach. $\text{Post}_t$ and $\text{Treat}_c$ follow an identical definition to those employed in the staggered-SDiD model based on the Beletsky et al. (2020) data, where the interaction of the two implies a county's DMA has originated media coverage that makes reference to the East Liverpool event. $X_i$ is a vector of individual-level demographic controls, while $\gamma_{c,t}$ are county-month fixed effects. I employ individual death certificate data from the CDC multiple cause of death file and restrict my sample to all deaths attributed to an external injury or poisoning (S00-T88) for the years 2015-2019 within a treated DMA or their adjacent county neighbors. Because opioid-related deaths are relatively uncommon events outside of Appalachia, using alternative external injury deaths as a baseline to compare against would afford a more complete picture first responder practices. As well, this triple-differences approach allows me to observe if there are any structural changes in death locations, beyond just those related to opioid overdoses.

The results of these LPM models are depicted in Table 10. To summarize: Within counties that have been exposed to misinformation pertaining to the East Liverpool event, and relative to other causes of death, the likelihood of an opioid overdose death being recorded in an inpatient or outpatient/emergency room setting decreases by approximately 2% and 1.9%, respectively; and the likelihood of those same deaths occurring someplace other than a medical setting or residence increases by approximately 3%. The interpretation of the results within Table 10 is that hesitancy in administering aid has yielded fewer attempts at resuscitation, and as such *moved* the location of death from medical to non-medical settings. Considering the urgency of opioid poisoning and the general preventability of death with timely administration of aid, this latter point is troubling.

# 5 Conclusions

In this paper, I have identified the significant impact of fentanyl exposure misinformation on first responder behavior during overdose events and overall opioid-related mortality. In particular, I have examined first responder responses to the widespread dissemination of inaccurate information regarding the supposedly-lethal hazards of passive fentanyl exposure. By analyzing changes in opioid-related mortality following a well-covered episode involving an Ohio police officer in 2017, the study reveals that areas with higher media exposure to this misinformation experience significant increases in opioid overdose deaths. This study underscores the importance of accurate information dissemination and highlights the potentially deadly consequences of misinformation on public health outcomes.

The primary takeaway from my results is that opioid-related mortality appears to be increasing by

approximately 2.84 deaths per 100-thousand population, per county-quarter; national-scale results place this figure at 0.448 additional deaths per 100-thousand population, per county-quarter. For my preferred specification, this jump in mortality represents 56.4% of the average quarterly mortality for the treated counties over the period observed. Back-of-the-envelope calculations identify 199 avoidable overdose deaths, or 72 per year within the Youngstown DMA, according to the SDiD model; and 5,479, or 1,992 per year nationally, according to the preferred staggered-SDiD model. For perspective on these magnitudes in the context of other opioid pandemic policies, Rees et al. (2019) find that the adoption of naloxone access laws by states corresponded to a net decrease of 62-69 opioid-related deaths per year, nationally. Using the Florence et al. (2021) estimate of $11.548 million in total economic costs per opioid overdose, this would put total costs at $2.298 and $48.222 billion within the Youngstown DMA and nationally, respectively.

There are several noteworthy policy implications of my findings. First, it would appear that some corrective effort on the part of criminal justice authorities is needed to combat further dissemination of misinformation. Recent efforts at retraining first responders to correct for fentanyl hazard misperceptions do appear effective (Winograd, Phillips, et al., 2020; Del Pozo et al., 2021), but there are limitations to the generalizability of these results. An obvious next-step then would be to pursue a randomized control trial experiment to observe the causal influence of first responder retraining on overdose response behavior and mortality.

Enhanced first responder training is an obvious remedy, but there are a number of reasons for policy makers to take pause when considering this particular approach. Namely, narrative correction does not appear to enjoy the same degree of social media play or lurid virality of the initial fentanyl exposure events, so efforts at retraining first responders could be costly if it were required to be conducted at a scale that compensates for this lack of information spillovers; and while there is promise that such retraining can influence first responder beliefs, it is unclear how universally this improved knowledge translates to actual behavior. Analysis of more general overdose education and naloxone distribution training revealed more complicated effects on law enforcement beliefs Winograd, Stringfellow, et al. (2020), with 31% of participants reporting *more* negative attitudes towards overdose victims following training. Similarly, the companion paper to this research (Kochersperger, 2023) observes differing responses to the fentanyl misinformation shock in naloxone administration rates across first responder types, with by far the largest declines being observed among law enforcement. This pronounced susceptibility to misinformation points to a broader issue among law enforcement that may be rooted in something more fundamental than a limited knowledge of toxicology. As outlined earlier in the research background, the present fentanyl panic appears to be just

the current iteration of a long-present myth-spinning phenomenon; so even if fentanyl hazards impressions are completely reversed, this may do little to limit future panics.

Another policy consideration is that media coverage of fentanyl hazards need not necessarily promote false information, and can even be a useful means of correcting misperceptions. A cursory survey of recent media coverage of supposed fentanyl exposure incidents does reveal the pronounced use of more skeptical language, and even statements outright dismissing the likelihood of events as described by law enforcement. Still, it is unclear whether this reactive fact-checking approach is a sufficient means of undoing the damage already wrought by the initial misinformation shocks.

During the early stages of the AIDS epidemic when misbeliefs of the virus' transmission vectors and contagiousness dominated public perceptions, concerted efforts on the part of a select few journalists to correct these narratives were consistently undermined by the broader media environment (Beharrell, 2003). Ultimately, this narrative correction depended on not only the dogged efforts of media insiders, but also celebrity intervention. Noteworthy events such as Princess Diana's visits to the opening of London's Middlesex Hospital AIDS ward in 1987, and Harlem Hospital's AIDS unit in 1989 were seminal in advancing the idea that AIDS victims were not passively contagious after she was photographed shaking hands and hugging them without gloves. Similarly, Earvin "Magic" Johnson's much-publicized HIV-positivity disclosure and subsequent sudden retirement from the NBA has been demonstrated to have reduced stigma surrounding HIV testing, and increased diagnoses among heterosexual men (Cardazzi et al., n.d.). While none of this is to suggest that celebrity endorsements represent a realistic policy response, it does highlight the corrective capacity that media and media consumption can command. Herman et al. (2020) note six months elapsed between the time of the East Liverpool event and the release of the ACMT-AACT joint statement that debunked many of the sensational fentanyl exposure claims; but that over this same time the scientific community was quiet and permitted the unchecked dissemination of misinformation. At a minimum, policymakers should expand on the medical misinformation correcting initiatives pioneered during the COVID-19 pandemic and prioritize swift fact-checking in the future. Effective policy to counter these misinformation narratives must both correct misperceptions among first responders and disincentive the continued dissemination of misinformation by the media.

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# Tables

Table 1: Data description, coverage and sources

| Data employed | Level of measure | Geographic Coverage | Temporal Coverage | Data Source |
|---|---|---|---|---|
| Multiple cause-of-death file | Individual deaths, aggregated to county-quarter | All US counties | 2014-2019 | National Vital Statistics System, Centers for Disease Control and Prevention |
| Percentage of the population with a credit score below 660 | County-quarter | All US counties | 2014-2019 | Equifax Subprime Credit Population, Equifax and Federal Reserve Bank of New York |
| Arrests per 100k for possession or distribution of heroin and similar drugs | Month-agency counts, aggregated to county-quarter | All US counties | 2014-2019 | Uniform Crime Reporting, Summary Reporting System, Federal Bureau of Investigation |
| Unemployment rate | County-quarter | All US counties | 2014-2019 | Local Area Unemployment Statistics, Bureau of Labor Statistics |
| Percent of laborforce employed in construction | County-quarter | All US counties | 2014-2019 | Local Area Unemployment Statistics, Bureau of Labor Statistics |
| County-level demographic estimates (percent hispanic, black) | Year-quarter | All US counties | 2014-2019 | County Population Totals, U.S. Census Bureau |
| Poverty rate | Year-quarter | All US counties | 2014-2019 | Small Area Income and Poverty Estimates, U.S. Census Bureau |
| Policy indicator for whether state has a naloxone access law | State-quarter | All US counties | 2014-2019 | Prescription Drug Abuse Policy System |
| Policy indicator for whether state has a law restricting prescriptions for opioid analgesics | State-quarter | All US counties | 2014-2019 | Prescription Drug Abuse Policy System |
| Policy indicator for whether state has a drug overdose Good Samaritan Law | State-quarter | All US counties | 2014-2019 | Prescription Drug Abuse Policy System |
| Policy indicator for whether state requires the PDMP to be queried under any circumstance | State-quarter | All US counties | 2014-2019 | Prescription Drug Abuse Policy System |
| Opioid dispensing rate per 100 people | County-year | Most US counties (n=2975) | 2014-2019 | Centers for Disease Control and Prevention, National Center for Injury Prevention and Control |
| Crime lab analysis of seized drugs (percent of seizures containing heroin or fentanyl) | Individual seizures, aggregated to county-quarter | 86 Ohio counties | 2014-2019 | Ohio Bureau of Criminal Investigation |

Table 2: County-quarter summary statistics table.

| | Control Counties | | Treated Counties | |
|---|---|---|---|---|
| Statistic | N | Mean | N | Mean |
| Overdose Count | 288 | 5,951 | 96 | 11.406 |
| Overdoses per 100k Pop. | 288 | 4,740 | 96 | 5.910 |
| Annual Population | 288 | 119,087 | 96 | 161,827 |
| % of Pop. Hispanic | 288 | 1.714 | 96 | 2.724 |
| % of Pop. Black | 288 | 4.461 | 96 | 9.438 |
| Unemployment Rate | 288 | 5.624 | 96 | 6.072 |
| % of Laborforce Employed in Construction | 288 | 4.445 | 96 | 3.734 |
| Poverty Rate | 288 | 13.221 | 96 | 16.058 |
| % of Pop. with Subprime Credit | 288 | 24.656 | 96 | 26.918 |
| Prescription Opioid Dispensing Rate | 288 | 82.011 | 96 | 96.646 |
| Heroin or Related Drug Arrests per 100k Pop. | 96 | 15.144 | 96 | 14.662 |
| Naloxone Access Laws | 288 | 0.931 | 96 | 0.969 |
| Good Samaritan Laws | 288 | 0.722 | 96 | 0.656 |
| Opioid Prescription Restriction Laws | 288 | 0.441 | 96 | 0.438 |
| Mandatory PDMP Laws | 288 | 0.913 | 96 | 0.948 |
| Fentanyl % of Seizures | 144 | 12.234 | 72 | 14.469 |
| Heroin % of Seizures | 144 | 20.507 | 72 | 24.228 |

(a) All data described above represents average observed values by county-quarter for the years 2014-2019. Treated counties are defined as those within the Youngstown, OH DMA (Columbiana, Mahoning, Trumbull, OH; Mercer, PA); control counties are those that are directly adjacent and share a common border. Fentanyl and heroin seizure figures are available within Ohio alone.

Table 3: SDiD coefficient estimates for mortality by drug type, stratified by demographics and age.

| | Pre-treatment Mean | SDiD estimates | SDiD estimates w/ covariates |
|---|---|---|---|
| Drug-related | 7.890 | 3.347*** | 3.953*** |
| | | (0.914) | (1.073) |
| Opioid-related | 5.012 | 2.353*** | 2.838*** |
| | | (0.811) | (1.099) |
| Possibly opioid-related | 7.251 | 3.129*** | 4.051*** |
| | | (0.823) | (1.091) |
| Illicit opioid-related | 3.811 | 1.520 | 1.876 |
| | | (1.009) | (1.201) |
| Synthetic opioid-related | 2.221 | 2.322* | 2.115* |
| | | (1.222) | (1.200) |
| Opioid related, Male | 5.224 | 1.858* | 3.285** |
| | | (1.109) | (1.552) |
| Opioid related, Female | 5.200 | 1.456 | 1.720 |
| | | (1.135) | (1.356) |
| Opioid related, White | 3.213 | 2.040** | 2.849** |
| | | (0.903) | (1.262) |
| Opioid related, Black | 7.018 | −4.034 | −1.447 |
| | | (4.178) | (5.876) |
| Opioid related, Hispanic | 3.090 | 4.324 | 1.121 |
| | | (2.639) | (4.016) |
| Opioid related, Age <25 | 1.398 | −0.515 | 0.172 |
| | | (1.024) | (1.042) |
| Opioid related, Age 25-34 | 13.395 | 6.659 | 11.445** |
| | | (4.945) | (4.556) |
| Opioid related, Age 35-44 | 11.095 | 7.970* | 6.284 |
| | | (4.611) | (4.438) |
| Opioid related, Age 45-54 | 8.129 | −0.425 | 0.953 |
| | | (1.881) | (2.052) |
| Opioid related, Age >54 | 2.258 | 0.767 | 0.800 |
| | | (0.811) | (0.878) |
| Observations | | 384 | 384 |

*Note:* *p<0.1; **p<0.05; ***p<0.01

(a) Results depicted here are derived by estimating an SDiD model which considers counties within the Youngstown OH DMA after the 2017 East Liverpool event as treated, and their immediately bordering counties as the SDiD control donor set. Dependent variables include per 100 thousand mortality rates for: all drug-related poisonings; opioid-related poisonings; possibly-opioid-related poisonings which include all opioid-related deaths, as well as those coded as related to unspecified drugs; illicit opioid-related poisonings (those associated with either heroin or a synthetic opioid, like fentanyl); and synthetic opioid-related poisonings. Results in column 3 are estimated by employing the time-variant covariate correction from Kranz (2022) and include the following auxiliary covariates: Percent of county population hispanic, black, or with a subprime credit score; percent of county laborforce employed in construction; unemployment and poverty rates; annual prescription opioid dispensing rate; arrests for heroin or related drugs per 100 thousand population; and policy indicators for whether the county's state had enacted naloxone access, good samaritan, mandatory PDMP, or opioid prescription restriction laws. Figures enclosed in parentheses are cluster bootstrap standard errors.

Table 4: Ohio fentanyl and heroin prevalence analysis results.

*(a) Opioid prevalence endogeneity test*

| | *Dependent variable:* | | |
|---|---|---|---|
| | Total Drug Seizures | Fentanyl Seizures, % of Total | Heroin Seizures, % of Total |
| | (1) | (2) | (3) |
| Lagged Opioid Overdose Deaths | $-0.290^{**}$ | $0.001^{*}$ | 0.00000 |
| | (0.124) | (0.0003) | (0.0004) |
| Observations | 1,740 | 1,740 | 1,740 |

*(b) SDiD results with opioid prevalence measures*

| | *Dependent variable:* | | |
|---|---|---|---|
| | Opioid-Related Overdose Deaths Per 100k | Percent of Drug Seizures Containing Fentanyl | Percent of Drug Seizures Containing Heroin |
| SDiD Estimates | $3.190^{**}$ | 0.015 | 0.101 |
| | (1.315) | (0.036) | (0.073) |
| Pre-treatment Mean | 5.821 | 0.075 | 0.311 |
| Observations | 216 | 216 | 216 |

*Note:* $^{*}$p<0.1; $^{**}$p<0.05; $^{***}$p<0.01

(a) The results in the top panel are derived by regressing each Ohio county-quarter measure of the dependent variables for the years 2015-2019 on the number of opioid-related deaths that occurred in the same county-quarter of the preceding year; as well as including county and quarter fixed-effects for all Ohio counties.

(b) The results in the bottom panel outline SDiD coefficient estimates derived from the primary model specification on the Ohio subsample, along with their bootstrapped standard errors in parentheses. Column 1 replicates the primary opioid-related results estimate from table 3 including covariates, but additionally includes measures for the percent of drug seizures conducted within those county-quarters that tested positive for heroin and fentanyl. Columns 2 and 3 follow the same controls specification, but set the fentanyl and heroin drug seizure percentages as the dependent variable.

Table 5: SDiD robustness checks, DMAs as treatment unit

| | *SDiD control donor set:* | | |
|---|---|---|---|
| | Adjacent counties as control | Bordering DMAs as control | Bordering DMAs without adjacent counties |
| *Estimates without Covariates* | | | |
| Youngstown DMA as treated | 2.353*** | 1.352 | 1.477 |
| | (0.811) | (1.042) | (1.168) |
| Significant viewership of Youngstown DMA station as treated | 2.031** | 1.643* | 1.671* |
| | (0.848) | (0.986) | (0.965) |
| Opioid-related death rate, logged | 0.417** | 0.161 | 0.222 |
| | (0.186) | (0.211) | (0.315) |
| Opioid-related deaths, levels | 3.600** | 3.772 | 4.049* |
| | (1.668) | (2.229) | (2.136) |
| DiD | 1.493 | 1.536 | 1.551* |
| | (0.980) | (0.987) | (0.883) |
| *Estimates with Covariates* | | | |
| Youngstown DMA as treated | 2.838*** | 0.891 | 0.818 |
| | (1.065) | (0.801) | (0.837) |
| Significant viewership of Youngstown DMA station as treated | 2.394* | 1.215* | 1.088 |
| | (1.334) | (0.717) | (0.765) |
| Opioid-related death rate, logged | 0.519** | 0.040 | 0.014 |
| | (0.233) | (0.145) | (0.187) |
| Opioid-related deaths, levels | 4.138*** | 2.935** | 3.176* |
| | (1.579) | (1.302) | (1.745) |
| DiD | 2.214* | 1.078* | 0.920 |
| | (1.134) | (0.582) | (0.685) |
| Observations | 384 | 1,200 | 912 |
| *Estimates without Covariates* | | | |
| Post ICD-10 adoption | 1.994** | 1.365 | 1.473 |
| | (0.926) | (0.978) | (1.055) |
| *Estimates with Covariates* | | | |
| Post ICD-10 adoption | 3.091* | 1.429 | 1.390* |
| | (1.681) | (0.898) | (0.766) |
| Observations | 272 | 850 | 646 |

*Note:* *p<0.1; **p<0.05; ***p<0.01

SDiD results for opioid-related mortality depicted here include: An alternative treatment specification that counts any counties the FCC has listed as having significant viewership of any stations within the Youngstown DMA as treated; alternative dependent variables of opioid-related death counts in levels and logs derived from the inverse hyperbolic sine transformation; and a conventional DiD model. The bottom panel re-estimates the opioid-related mortality model on the subset of observations occurring after the October 2015 adoption of the ICD-10 coding system. Column 1 defines donors to the SDiD control from the immediately bordering counties to the treated DMA; column 2, expands this donor set to include all counties of bordering DMAs; and in column 3 includes all counties in bordering DMAs but excludes immediately bordering counties. The covariate-inclusive estimates employ the correction from Kranz (2022) and includes the following time-variant controls: Percent of county population hispanic, black, or with a subprime credit score; percent of county laborforce employed in construction; unemployment and poverty rates; annual prescription opioid dispensing rate; arrests for heroin or related drugs per 100 thousand population; and policy indicators for whether the county's state had enacted naloxone access, good samaritan, mandatory PDMP, or opioid prescription restriction laws.

Table 6: SDiD robustness checks, commuting zones as treatment unit

| | *SDiD control donor set:* | | |
|---|---|---|---|
| | Adjacent counties as control | Bordering commuter zones as control | Bordering commuter zones without adjacent counties |
| *Estimates without Covariates* | | | |
| Youngstown commuter zone as treated | 2.031** | 1.809** | 1.680 |
| | (0.813) | (0.893) | (1.076) |
| Opioid-related death rate, logged | 0.290 | 0.219 | 0.249 |
| | (0.185) | (0.191) | (0.250) |
| Opioid-related deaths, levels | 2.979* | 3.463* | 3.721* |
| | (1.557) | (1.874) | (1.945) |
| DiD | 1.562* | 1.884** | 2.032*** |
| | (0.868) | (0.752) | (0.782) |
| *Estimates with Covariates* | | | |
| Youngstown commuter zone as treated | 2.394*** | 1.534* | 1.187 |
| | (1.190) | (0.884) | (0.874) |
| Opioid-related death rate, logged | 0.457* | 0.122 | 0.006 |
| | (0.269) | (0.186) | (0.235) |
| Opioid-related deaths, levels | 3.329*** | 3.113* | 3.676 |
| | (1.547) | (1.718) | (2.250) |
| DiD | 1.933 | 1.636** | 1.693** |
| | (1.199) | (0.717) | (0.778) |
| Observations | 384 | 960 | 696 |

*Note:* *p<0.1; **p<0.05; ***p<0.01

SDiD results for opioid-related mortality depicted here substitute local commuter zone delineations from Fowler and Jensen (2020) for DMAs and include the following variations: Alternative dependent variables of opioid-related death counts in levels and logs derived from the inverse hyperbolic sine transformation; and a conventional DiD model. Column 1 defines donors to the SDiD control from the immediately bordering counties to the treated commuter zone; column 2, expands this donor set to include all counties of bordering commuter zone; and in column 3 includes all counties in bordering commuter zone but excludes immediately bordering counties. The covariate-inclusive estimates employ the correction from Kranz (2022) and includes the following time-variant controls: Percent of county population hispanic, black, or with a subprime credit score; percent of county laborforce employed in construction; unemployment and poverty rates; annual prescription opioid dispensing rate; arrests for heroin or related drugs per 100 thousand population; and policy indicators for whether the county's state had enacted naloxone access, good samaritan, mandatory PDMP, or opioid prescription restriction laws.

Table 7: Covariate balance tables for SDiD donor set specifications.

| | *SDiD control donor set:* | | |
|---|---|---|---|
| Covariate | Adjacent counties as control | Bordering DMAs as control | Bordering DMAs without adjacent counties |
| Overdoses per 100k Pop. | 2.455 | 3.243 | 3.079 |
| Annual Population | 602.214 | 1,186.986 | 845.837 |
| % of Pop. Hispanic | 41.647 | 99.505 | 76.825 |
| % of Pop. Black | 139.241 | 311.535 | 227.105 |
| Unemployment Rate | 3.790 | 8.271 | 8.118 |
| % of Laborforce Employed in Construction | 8.118 | 11.409 | 10.479 |
| Poverty Rate | 11.566 | 15.863 | 14.689 |
| % of Pop. with Subprime Credit | 19.887 | 19.043 | 16.435 |
| Prescription Opioid Dispensing Rate | 21.501 | 25.714 | 19.436 |
| Heroin or Related Drug Arrests per 100k Pop. | 3.721 | 5.894 | 5.832 |
| Naloxone Access Laws | 0.006 | 0.547 | 0.566 |
| Good Samaritan Laws | 0.654 | 0.733 | 0.752 |
| Opioid Prescription Restriction Laws | 0.150 | 0.115 | 0.100 |
| Mandatory PDMP Laws | 0.006 | 1 | 1 |

Figures in the table above represent the maximum standardized mean differences on observables when comparing covariate values for the treated counties against the weighted controls drawn from the SDiD control donor set. Columns 1, 2, and 3 represent the donor set specifications for all immediately adjacent counties to the Youngstown OH DMA, all counties in within bordering DMAs, and all counties in within bordering DMAs excluding immediately adjacent counties to the Youngstown OH DMA, respectively. Weights assigned to controls are derived from the opioid-related mortality SDiD model estimated without covariates.

Table 8: Falsification test SDiD results.

| | *Dependent variable:* | | |
|---|---|---|---|
| | Motor Vehicle Accident Deaths Per 100k | Heart Attack-Related Deaths Per 100k | Assault-Related Deaths Per 100k |
| Without Covariates | −0.484 | −0.144 | 0.076 |
| | (0.557) | (4.695) | (0.260) |
| With Covariates | −0.703 | 3.938 | 0.026 |
| | (0.648) | (6.321) | (0.293) |
| Pre-treatment Mean | 2.605 | 66.37 | 1.152 |
| Observations | 384 | 384 | 384 |

*Note:* $^{*}$p<0.1; $^{**}$p<0.05; $^{***}$p<0.01

The results above are derived by following the same specifications as the opioid-related mortality models in table 3, row 1, but using mortality rates for motor vehicle accident, heath attack, and assault-related deaths. Auxiliary covariates include: Percent of county population hispanic, black, or with a subprime credit score; percent of county laborforce employed in construction; unemployment and poverty rates; annual prescription opioid dispensing rate; arrests for heroin or related drugs per 100 thousand population; and policy indicators for whether the county's state had enacted naloxone access, good samaritan, mandatory PDMP, or opioid prescription restriction laws. Figures enclosed in parentheses are cluster bootstrap standard errors.

Table 9: Staggered SDiD estimates for all US counties treated with media coverage of East Liverpool event.

| | *SDiD control treatment specification:* | |
|---|---|---|
| | All treated DMAs and adjacent counties as controls | All treated DMAs except Youngstown, OH and adjacent counties as controls |
| Without Covariates | 0.571*** | 0.565*** |
| | (0.180) | (0.197) |
| With Covariates | 0.464** | 0.448** |
| | (0.202) | (0.214) |
| Pre-treatment Mean | 3.903 | 3.892 |
| Observations | 13,752 | 13,656 |

*Note:* *p<0.1; **p<0.05; ***p<0.01

Treatment variables used here is an indicator for whether a county's DMA has originated media coverage that makes reference to the East Liverpool event. The results for column 1 use a sample for all counties in treated DMAs and their adjacent neighbors, while column 2 excludes counties within the Youngstown OH DMA; both samples drop counties which did not record at least one opioid death per year over the observed period. Covariates employed by the models in row 2 include: Percent of county population hispanic, black, or with a subprime credit score; percent of county laborforce employed in construction; unemployment and poverty rates; annual prescription opioid dispensing rate; arrests for heroin or related drugs per 100 thousand population; and policy indicators for whether the county's state had enacted naloxone access, good samaritan, mandatory PDMP, or opioid prescription restriction laws. Estimates enclosed in brackets are staggered-adoption cluster bootstrap standard errors derived from the method described by Clarke et al. (2023).

Table 10: Place-of-death linear probability results.

| Death Location: | Inpatient | Outpatient & Emergency Room | DOA | Home | Other |
|---|---|---|---|---|---|
| *Variables* | | | | | |
| Media-Treated | 0.0007 | 0.0006 | $-2.15 \times 10^{-6}$ | $-4.54 \times 10^{-5}$ | -0.0010 |
| | (0.0018) | (0.0016) | (0.0006) | (0.0001) | (0.0009) |
| Is Opioid Overdose | -0.1203*** | -0.0118*** | 0.0008 | 0.2367*** | -0.0782*** |
| | (0.0018) | (0.0016) | (0.0006) | (0.0023) | (0.0023) |
| Media-Treated× Is Opioid Overdose | -0.0197*** | -0.0186*** | $-6.46 \times 10^{-5}$ | 0.0014 | 0.0298*** |
| | (0.0031) | (0.0025) | (0.0010) | (0.0035) | (0.0037) |
| Pre-treatment Means | 0.2609 | 0.1307 | 0.0125 | 0.2720 | 0.2233 |
| Percent Effect | −0.0755 | −0.1423 | −0.0051 | 0.0051 | 0.1335 |
| *Fit statistics* | | | | | |
| Observations | 809,703 | 809,703 | 809,703 | 809,703 | 809,703 |
| $R^2$ | 0.20116 | 0.11889 | 0.11737 | 0.19103 | 0.21648 |
| Within $R^2$ | 0.01169 | 0.00050 | $5.05 \times 10^{-5}$ | 0.03469 | 0.00363 |

*Note:* *p<0.1; **p<0.05; ***p<0.01
*Clustered (County×Month) standard-errors in parentheses*

Linear probability model estimates for the marginal likelihood of one of five specific death locations being listed on certificate. Treatment defined as the interaction between an indicator for whether an individual death observation's DMA has originated media coverage that makes reference to the East Liverpool event, and an indicator for whether the observed death is attributed to an opioid overdose. Sample is drawn from the complete set of individual deaths recorded in a treated DMA or neighboring county based on Beletsky et al. (2020), which attribute the underlying cause to an external injury or poisoning (corresponding to ICD-10 codes S00-T88). Covariates include reported age, sex, race, ethnicity, highest level of educational attainment, as well as county-month fixed effects.

# Figure Captions

Figure 1. Time series of Google search interest in the hazards of fentanyl exposure.

Figure 2. Regional time series of Google search interest in fentanyl.

Figure 3. Time series for opioid mortality and prevalence.

Figure 4. Treatment maps for East Liverpool event.

Figure 5. Plotted primary SDiD results.

Figure 6. Distribution of placebo test results.

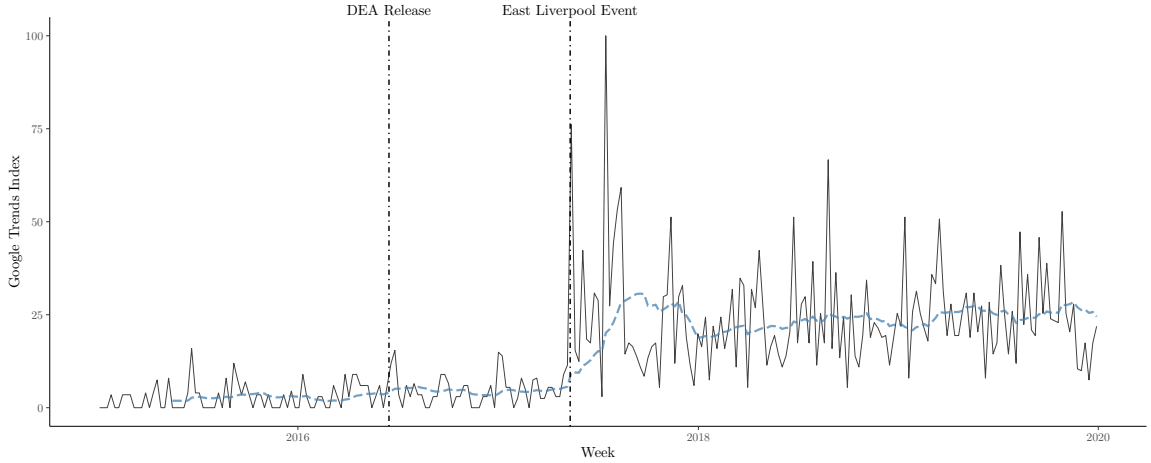
Figure 7. Comparison of SDiD controls constructed under different donor set specifications.

Figure 8. Regional time series of local and county-level government expenditures on police protection, fire prevention, and health services.

Figure 9. Regional time series of naloxone kit distribution, trainings, and administrations.

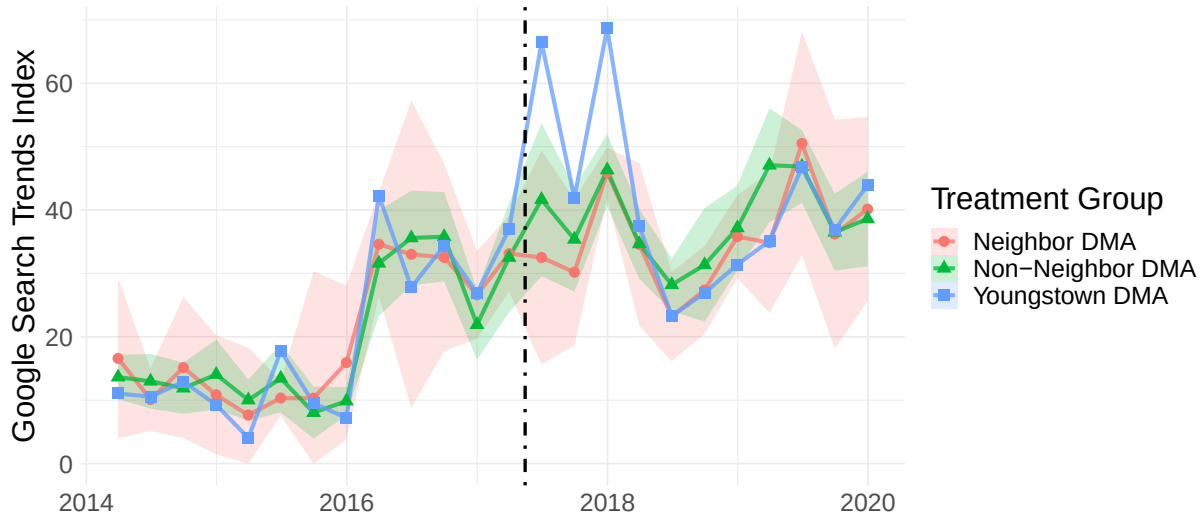
# Figures

Figure 1: Time series of Google search interest in the hazards of fentanyl exposure.



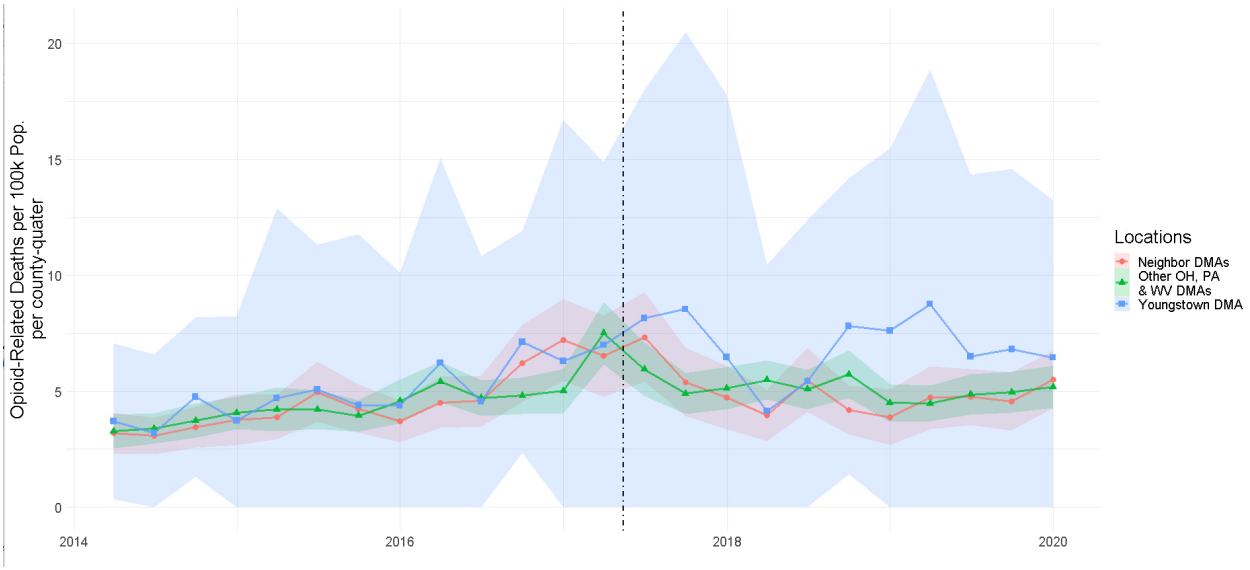

Google search data was collected from Google Trends and represents the relative popularity of search terms over the specified time frame; the time series was derived from querying the Google trends for "fentanyl AND (touch* OR contact* OR absor* OR inhal* OR expos*)". Solid black line is the weekly average for the Google Trends Index, while the dashed blue line is the rolling average of the 20 preceding weeks. The dashed vertical lines demonstrate the dates of the DEA press release and the East Liverpool event.

Figure 2: Regional time series of Google search interest in fentanyl.

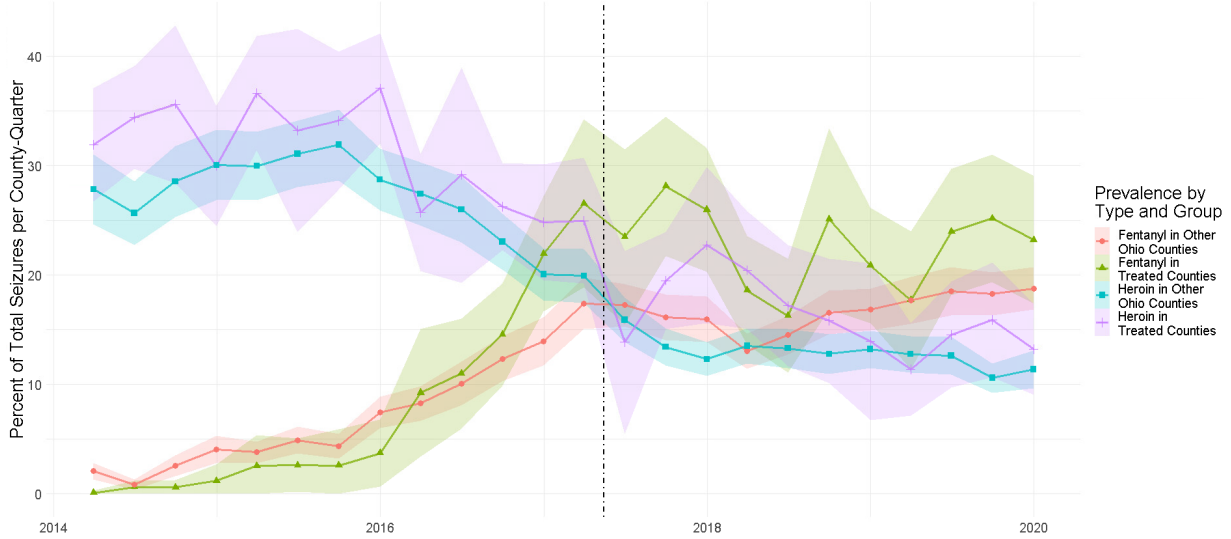



Google search data was collected from Google Trends and represents the relative popularity of search terms over the specified time frame. Time series were derived from querying the Google trends for "fentanyl" for each Designated Market Area (DMA)-quarter over 2014-2019. The Youngstown DMA contains East Liverpool, OH; while neighbor DMA includes all counties in media markets that share a border with the Youngstown DMA; and non-neighbor DMAs include all other counties in Ohio, Pennsylvania and West Virginia for which search trends data is recorded. Shaded regions represent the 95% confidence intervals and the dashed vertical line demonstrates the date of the East Liverpool event.

Figure 3: Time series for opioid mortality and prevalence.

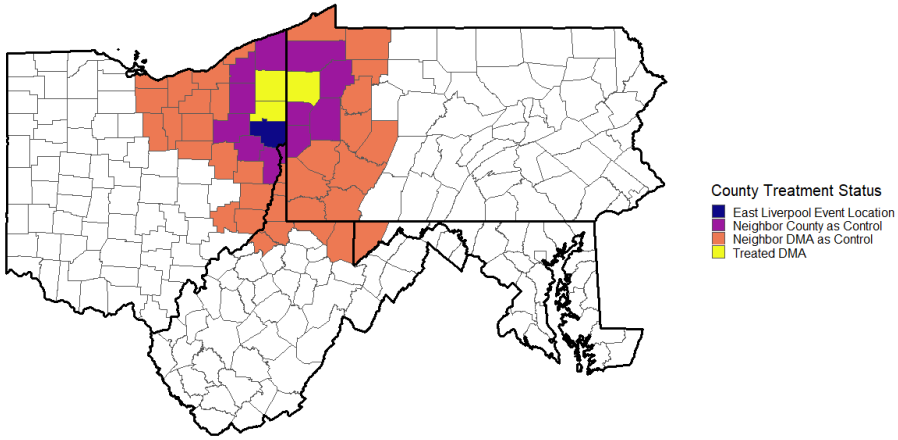



(a) Opioid-related deaths per 100 thousand population by county-quarter. Shaded regions represent the 95% confidence intervals and the dashed vertical line demonstrates the date of the East Liverpool event.

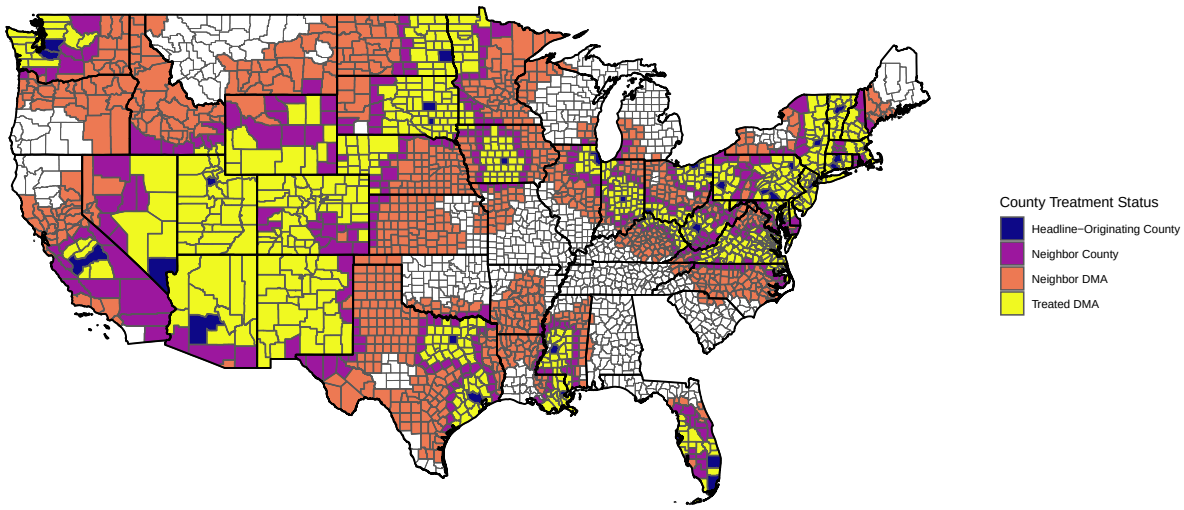



(b) Opioid prevalence as percent of drug seizures that tested positive for fentanyl and heroin based on the BCI data, by county-quarter. Shaded regions represent the 95% confidence intervals and the dashed vertical line demonstrates the date of the East Liverpool event.

Figure 4: Treatment maps for East Liverpool event.

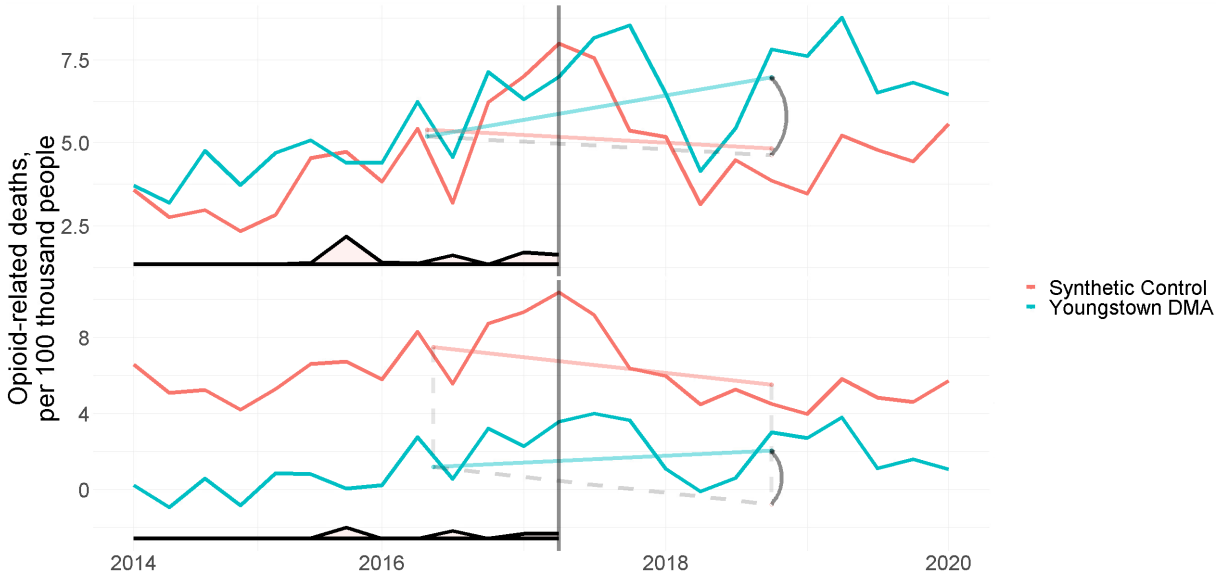



(a) A map of counties considered as treated under my primary identification strategy. Blue-shaded region is Columbiana county, where East Liverpool is located; yellow-shaded regions are other counties within the same DMA; purple-shaded counties are bordering counties within neighbor DMAs; and the orange-shaded region are all other counties within the neighboring DMAs.



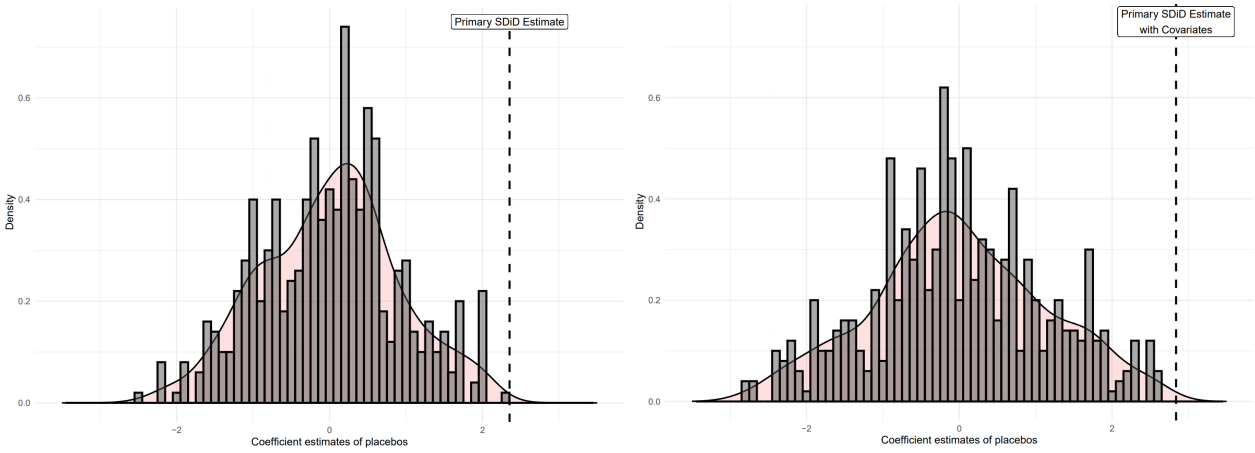

(b) A map of national media coverage of East Liverpool event, as based on the data from Beletsky et al. (2020). Blue-shaded regions correspond to counties where a news article which made reference to the East Liverpool event originated; yellow-shaded regions are other counties within the same DMAs as those which originated coverage of the East Liverpool event; purple-shaded counties are bordering counties within neighbor DMAs; and the orange-shaded region are all other counties within the neighboring DMAs.

Figure 5: Plotted primary SDiD results.



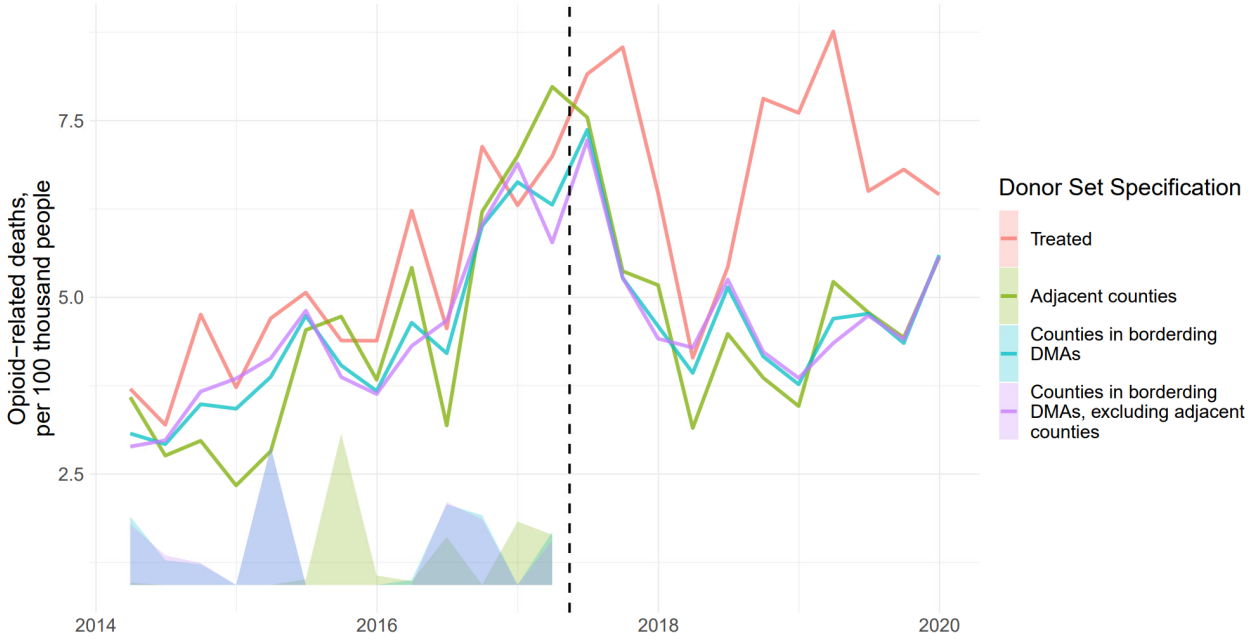

(a) SDiD results depicted here are according to the opioid-related mortality specifications from table 3. Pink-shaded regions at the bottom of plots depict time-weights, black arrowed-line represents the average treatment effect. The bottom panel is estimated employing the time-variant covariate correction from Kranz (2022) (hence the seemingly-negative values for the SDiD control the beginning of the observation period) and includes the following auxiliary covariates: Percent of county population hispanic, black, or with a subprime credit score; percent of county laborforce employed in construction; unemployment and poverty rates; annual prescription opioid dispensing rate; arrests for heroin or related drugs per 100 thousand population; and policy indicators for whether the county's state had enacted naloxone access, good samaritan, mandatory PDMP, or opioid prescription restriction laws.

Figure 6: Distribution of placebo test results.



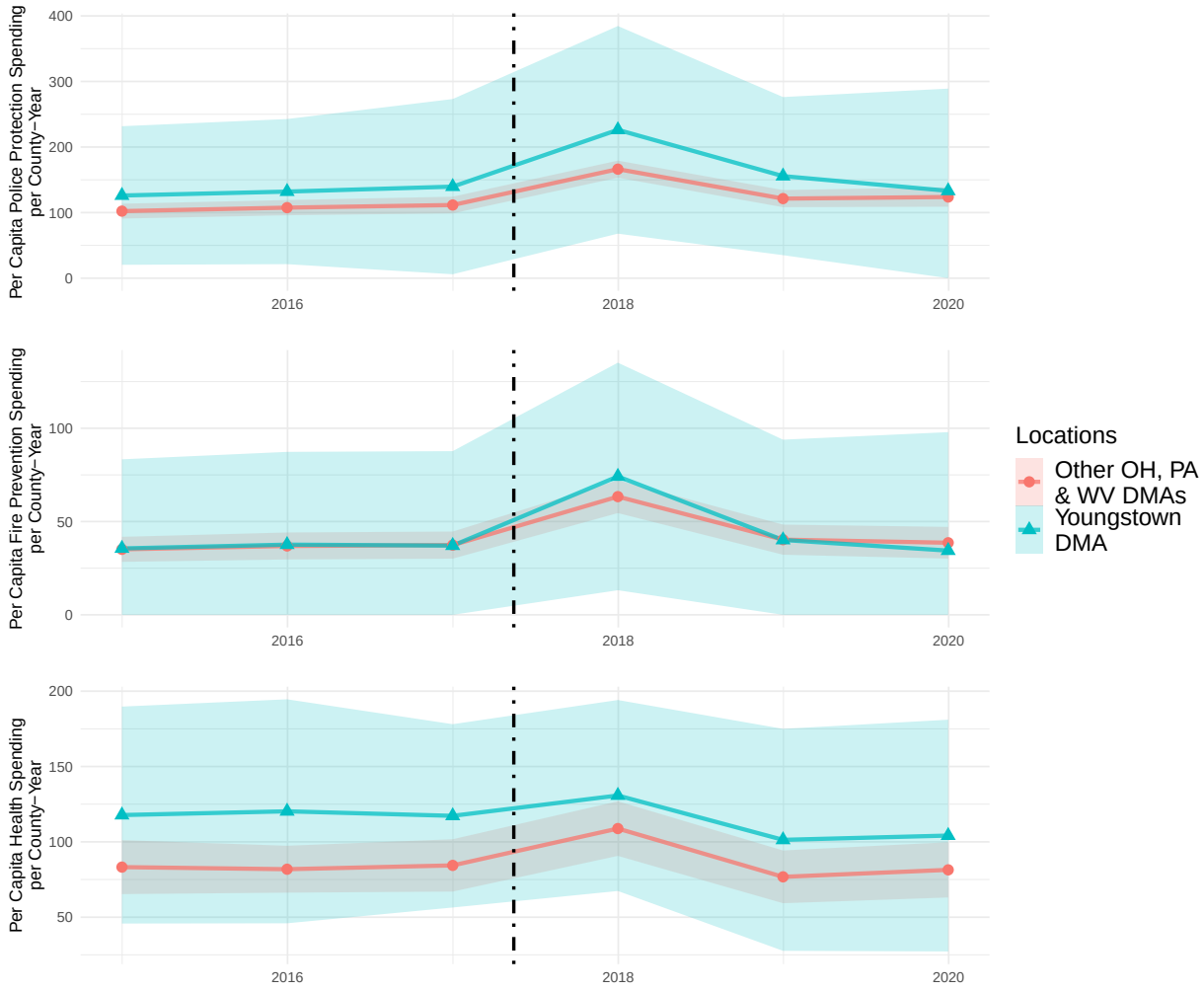

(a) Histogram and density plots above describe the empirical noise distribution for the primary opioid-related mortality SDiD model controls. Coefficient estimates here are derived according to the placebo protocol outlined by Arkhangelsky et al. (2021), which randomly assigns controls as treated and the SDiD model is re-estimated on the donor set alone. Distributions are based on 500 replications for each model specification. Dashed lines indicate the value of the estimated SDiD treatment effects.

Figure 7: Comparison of SDiD controls constructed under different donor set specifications.



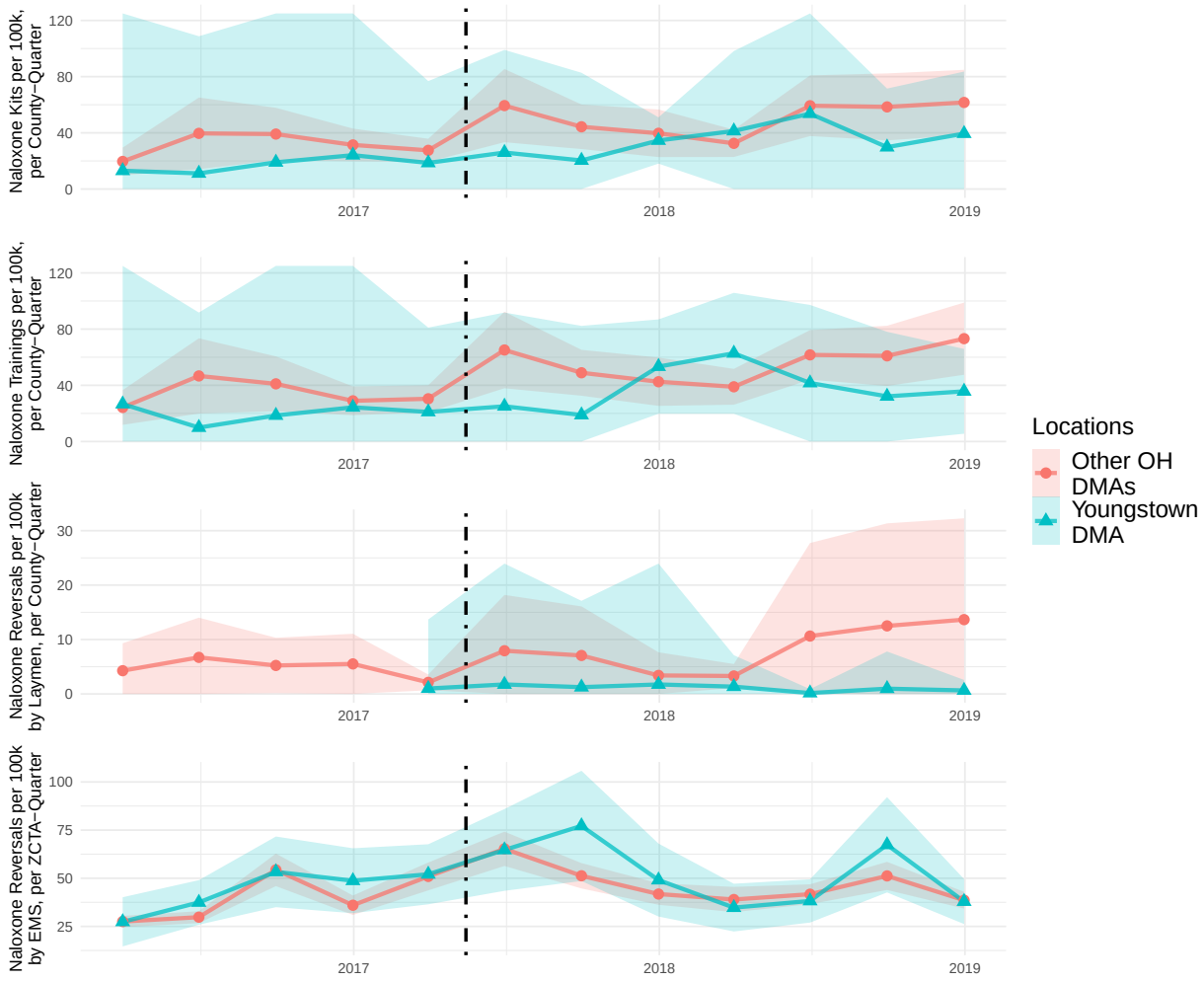

(a) SDiD results depicted here are according to the opioid-related mortality specifications from row 1 of table 5. Shaded regions at the bottom of plots depict time-weights for the corresponding donor set specification, which include: All immediately adjacent counties to the Youngstown OH DMA, all counties in within bordering DMAs, and all counties in within bordering DMAs excluding immediately adjacent counties to the Youngstown OH DMA, respectively.

Figure 8: Regional time series of local and county-level government expenditures on police protection, fire prevention, and health services.




(a) Time series figures for per capita municipal- and county-level expenditures on police protection, fire prevention, and health services spending from the Census' Annual Survey of State and Local Government Finances. Plots are averages across counties within the Youngstown OH DMA against all other counties in Ohio, Pennsylvania, and West Virginia by county-year. Shaded regions represent the 95% confidence intervals and the dashed vertical line demonstrates the date of the East Liverpool event.

Figure 9: Regional time series of naloxone kit distribution, trainings, and administrations.




(a) Time series figures for naloxone trainings, distributed kits, self-reported naloxone resusitations performed by laymen, and naloxone resusitations performed by EMS per 100k population. Plots are averages across counties/ZCTAs within the Youngstown OH DMA against all other counties in Ohio by county-quarter or ZCTA-quarter. Shaded regions represent the 95% confidence intervals and the dashed vertical line demonstrates the date of the East Liverpool event.