# Essays in Health Economics

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Daniel Robert Guth

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Daniel Robert Guth
ORCID: 0000-0003-0937-5282

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# ABSTRACT

This thesis consists of three health economics papers, two studying the effectiveness of policy interventions on the opioid epidemic, and one on the effects of air pollution on school absences. The first two chapters were coauthored with Shiyu Zhang, a former Caltech graduate student.

The first chapter examines the market for prescription opioids following the OxyContin Reformulation, an event that made OxyContin harder to misuse. Using detailed prescription opioid sales data from 2006 to 2014, we show that event did not reduce overdose deaths but led individuals to switch to generic oxycodone as a substitute for OxyContin.

The second chapter examines geographic spillover effects from state prescription drug monitoring programs (PDMPs). We show that these policies reduce prescription opioid sales and opioid overdose deaths in the state they are enacted in. However, because they only track opioids sold locally, these programs induce individuals to drive across state lines to purchase opioids and avoid these regulations.

The final chapter examines the effects of air pollution on NYC school absences using daily changes in wind direction. I show that PM2.5 and Ozone concentrations are strongly influenced by wind patterns, and exposure to these two pollutants causes detectable increases in absences over the following two days. Reductions in PM2.5 pollution over time have prevented approximately 381,000 absences annually in NYC which increases school funding by $19 million.

# PUBLISHED CONTENT AND CONTRIBUTIONS

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Zhang, Shiyu and Daniel Guth (2021). "The OxyContin Reformulation Revisited: New Evidence From Improved Definitions of Markets and Substitutes". In: *arXiv preprint arXiv:2101.01128*. Daniel contributed to the conception of the project and in the writing of the manuscript. URL: https://arxiv.org/pdf/2101.01128.pdf.

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# NOMENCLATURE

**ARCOS.** The Automated Reports and Consolidated Ordering System (ARCOS) is the DEA's system for tracking opioid distribution and sales. A federal judge ordered the release of all ARCOS data from 2006 to 2014.

**Generic Oxycodone.** Oxycodone is the active ingredient in most high-dosage opioid pills including OxyContin, and generic oxycodone refers to generic opioid pills manufactured after OxyContin lost the patent in 2004..

**IV Regression.** An Instrumental Variables (IV) Regression uses random variation in one variable to get causal estimates of the effects on the outcome variable.

**OxyContin.** A brand of prescription opioid produced by Purdue Pharma that was marketed for severe pain and led to misuse.

**OxyContin Reformulation.** In 2010, Purdue Pharma added a chemical to OxyContin that made it harder to crush or inject for an immediate high.

**PDMP.** A Prescription Drug Monitoring Program (PDMP) is a state regulation that tracks individuals purchasing prescription opioids in that state. In Chapter 2 we focus on electronic PDMPs, that allow doctors and pharmacists to immediately view a patient's opioid history.

# INTRODUCTION

Broadly speaking, this dissertation uses econometric methods to identify relationships between public policies and health outcomes. This thesis consists of three papers, two studying the effects of policy interventions and market forces on the opioid crisis, and one on the effects of air pollution on school absences.

The first two chapters study how prescription opioid markets affected drug overdose deaths and are coauthored with Shiyu Zhang, a former Caltech graduate student. When I arrived at Caltech in 2012, there were approximately 22,000 annual opioid overdose deaths in the United States and prescription opioids were the most common opioid contributing to death. By the time I started my PhD in 2017, fatalities had more than doubled to approximately 47,000 overdose deaths, with a switch from prescription opioids, to heroin, and then fentanyl. In the most recent year with finalized data, 2021, opioid overdose deaths nearly doubled again to 80,000, with the increase almost entirely due to fentanyl.[1]

Chapter 1 examines the chemical reformulation for OxyContin in 2010 that made it more difficult to misuse. Using novel prescription opioid sales data released due to a federal court case, we show that generic oxycodone was more widely misused and predictive of future overdose deaths than OxyContin. In contrast to previous work, we show the decline in OxyContin sales first led to substitution to generic oxycodone, and then later to heroin. These findings highlight the important role generic oxycodone played in the opioid epidemic and the limited effectiveness of a partial supply-side intervention. Since writing that paper, many of the largest opioid manufacturers and distributors have settled for billions of dollars to resolve lawsuits related to their role in the opioid crisis.[2]

Chapter 2 analyzes state-level policies known as Prescription Drug Monitoring Programs (PDMPs) and their effect on the prescription opioid markets. These PDMPs document every time an individual purchases opioids at a pharmacy in that state, and we focus on electronic versions that allow doctors and pharmacists

[1] 2012 data from this link, 2017 number from this link, and most recent data from here.

[2] Figure A.1 shows the market share for opioids by manufacturer. SpecGx is owned by Mallinckrodt, which settled for $1.6 billion dollars, and Actavis and Par were bought by Teva, which settled for $4.25 billion dollars. The manufacturer of OxyContin, Purdue Pharma and its owners, settled for $6 billion dollars, and the three largest distributors of opioids settled for a combined $21 billion.

to instantly review a patient's opioid history. We show that these programs are effective at reducing sales and overdose deaths, but also that individuals cross state lines and purchase opioids in neighboring states to avoid these regulations. This chapter highlights a cost to having state-level policies when people can freely travel between states and purchase opioids while obscuring their prescription history.

Chapter 3 changes focus to air pollution and the effects on school absences as a proxy for children's health. Previous work has shown that children are especially susceptible to air pollution, and that pollution was associated with increased absences due to respiratory illness. Using all school absences in New York City for 14 years and wind-carried pollution, I estimate a casual relationship for how PM2.5 and Ozone increase absences at the daily level. My results suggest that the decrease in average daily PM2.5 pollution of 5 $\mu g/m^3$ from 2006 to 2019 led to at least 381,000 fewer absences across NYC schools and increased education spending by $19 million every year. This work shows that air pollution, even when below the federal limits, still has detectable negative effects on children.

*Chapter 1*

# THE OXYCONTIN REFORMULATION REVISITED: NEW EVIDENCE FROM IMPROVED DEFINITIONS OF MARKETS AND SUBSTITUTES

SHIYU ZHANG AND DANIEL GUTH

The opioid epidemic began with prescription pain relievers. In 2010 Purdue Pharma reformulated OxyContin to make it more difficult to abuse. OxyContin misuse fell dramatically, and concurrently heroin deaths began to rise. Previous research overlooked generic oxycodone and argued that the reformulation induced OxyContin users to switch directly to heroin. Using a novel and fine-grained source of all oxycodone sales from 2006 to 2014, we show that the reformulation led users to substitute from OxyContin to generic oxycodone, and the reformulation had no overall impact on opioid or heroin mortality. In fact, generic oxycodone, instead of OxyContin, was the driving factor in the transition to heroin. Finally, we show that by omitting generic oxycodone we recover the results of the literature. These findings highlight the important role generic oxycodone played in the opioid epidemic and the limited effectiveness of a partial supply-side intervention.

## 1.1 Introduction

Since 1999, the opioid epidemic has claimed more than 415,000 American lives (National Center for Health Statistics, Centers for Disease Control and Prevention, 2020). What started with fewer than 6,000 opioid-related deaths in 1999 grew steadily every year until fatalities reached 47,573 deaths in 2017. Following a small decline in fatal drug overdoses in 2018, deaths continue to rise. Over the past two decades, millions of Americans have misused prescription opioids or progressed to more potent opioids, first heroin and later fentanyl. Many social scientists have tried to understand how this crisis has grown over two decades despite significant public health efforts to the contrary.

Doctors and health economists have long argued that the drug most responsible for prescription opioid overdose deaths, and the key to understanding the transition from prescription opioids to heroin starting in 2010, was OxyContin. Previous

research (Van Zee, 2009), court proceedings (Meier, 2007), and books (Meier, 2003, Macy, 2018) have documented how Purdue Pharma's marketing campaign for OxyContin downplayed the risk of addiction starting in 1996. Since then, according to the National Survey on Drug Use and Health (NSDUH), millions of Americans have misused it previously. A key question in this area is whether or not making prescription opioids, especially OxyContin, more difficult to abuse will reduce overdose deaths.

In this paper, we show that restricting access to OxyContin led many users to switch to generic oxycodone but had no immediate impact on opioid or heroin mortality. Earlier analyses attributing opioid overdose deaths in the late 2000s and the subsequent rise in heroin deaths to OxyContin are incomplete because they omit generic oxycodone. Our analysis shows that the misuse of generic oxycodone was prevalent before the reformulation that restricted OxyContin access, and was even more so afterward. We also show that heroin overdose deaths increased in areas with high generic oxycodone exposure, not high OxyContin exposure, two years after the OxyContin reformulation. In addition, we show that omitting generic oxycodone in our regressions recovers the results of the literature.

This analysis was not possible until several years ago when the *Washington Post* won a court order and published the complete Automation of Reports and Consolidated Orders System (ARCOS). The ARCOS tracks the manufacturer, the distributor, and the pharmacy of every pain pill sold in the United States. The newly released data allow us to analyze what happened to sales of generic oxycodone and OxyContin when OxyContin suddenly became more difficult to abuse. The previous literature focused on analyzing OxyContin because of Purdue's notorious role in the opioid crisis. However, the new data shows that the sales of OxyContin was only a small part of the sales of all prescription opioids: in terms of the number of pills, OxyContin was 3% of all oxycodone pills sold from 2006 to 2012; in terms of morphine milligram equivalents (MME), OxyContin has closer to 20% market share over this period. The new transaction-level ARCOS data allows us to track the sales of generic oxycodone and fill in the narrative gaps of how the opioid crisis progressed in the United States.

Following Alpert, Powell, and Pacula (2018), W. N. Evans, Lieber, and Power (2019), and T. Cicero and Ellis (2015), we treat the introduction of an abuse-deterrent formulation (ADF) of OxyContin as an exogenous shock that should only

affect people who seek to bypass the extended-release mechanism for a more immediate high. We construct measures of exposure by combining ARCOS sales and the NSDUH data on drug misuse. The NSDUH is the best survey of people who use drugs at the state level, and by combining it with local sales we can capture variation in drug use within the state. We leverage this variation in OxyContin and generic oxycodone exposure to examine how the reformulation affected OxyContin sales, generic oxycodone sales, opioid mortality, and heroin mortality. Our first contribution is that we fix the omitted-variable problem by differentiating between OxyContin and generic oxycodone, and we show that this leads to different conclusions than what previous literature suggests. Our second contribution is disaggregating the data to metropolitan statistical area (MSA), which allows us to address endogeneity at the state level.

To preview our results, we find strong evidence of substitution from OxyContin to generic oxycodone immediately after the reformulation. This substitution was larger in places that had more OxyContin misuse pre-reform, which is consistent with our hypothesis that users would switch between oxycodones rather than move on to heroin. Because this substitution should be concentrated among people misusing OxyContin, the results imply large changes in consumption at the individual level. Back-of-the-envelope calculation suggests 68% of the decline in OxyContin sales was substituted to oxycodone in MSAs with high OxyContin misuse. The findings are consonant with surveys like Havens et al. (2014), Coplan et al. (2013), and Cassidy et al. (2014) who all document substitution to generic oxycodone after the reformulation by people seeking to bypass the ADF. We also find suggestive evidence of substitution from generic oxycodone to OxyContin after the reformulation in places where generic oxycodone misuse was high, a channel that has been unexplored in previous research.

Our event study approach also shows that generic oxycodone exposure is predictive of future heroin overdose deaths whereas OxyContin exposure is not. The results are not contingent on methodology or our construction of exposure measures. Crucially, if we run the same exact regressions at the state or MSA level and omit generic oxycodone, we recover the results of the literature where OxyContin misuse appears to be significantly predictive of future heroin overdose deaths. We find that every standard deviation increase in generic oxycodone exposure pre-reformulation is associated with a 40.8% increase in heroin mortality in 2012 from the 2009 baseline level. As further evidence against the argument that there was immediate

substitution from OxyContin to heroin after the reformulation, we note that in all of our regressions the increase in heroin deaths wasn't statistically significant until 2012. As suggested in O'Donnell, Gladden, and Seth (2017), the rise in heroin deaths can be attributed in part to an increase in the supply of heroin as well as the introduction of fentanyl into heroin doses.

Our findings highlight the pitfalls of omitting important substitutes to OxyContin in analyzing the prescription opioid crisis. Purdue Pharma has received well-deserved attention over the years for its role in igniting the crisis. The company has been involved in many lawsuits over the years, but perhaps the most damaging were lesser-known cases that involved losing its patent in 2004[1] which cleared the way for a rapid increase in generic oxycodone sales in the early 2000s. While Purdue Pharma was being sued and scrutinized, several manufacturers took the opportunity to fill in the gaps of OxyContin. By 2006, generic oxycodone outsold OxyContin by more than 3-to-1 after accounting for pill dosage differences. This paper sheds lights on the role generic oxycodone played and continues to play in the opioid crisis and helps policy makers update their picture of the opioid use disorder (OUD) landscape.

The paper also calls attention to the limited effectiveness of a partial supply-side intervention to curb OUD. Purdue Pharma was once a dominant player in the opioid market, but by the time of the reformulation, that dominance had vanished and it was only one of the many manufacturers whose drugs were actively misused by Americans. Purdue was the first company to include abuse-deterrent formulation (ADF) in their opioids, but it is not until recent years that other brands started adding anti-deterrent compounds to their products (Pergolizzi et al., 2018). When substitutions to other abusable opioids are easy, cutting supplies of one kind is less effective.

The rest of the paper runs as follows. Section 2 gives more background on the opioid crisis and explains how previous research has characterized the OxyContin reformulation. In Section 3 we describe the new ARCOS sales database, the NSDUH misuse data, the NVSS mortality data, as well as our constructed misuse measure and descriptive statistics. Section 4 describes our empirical strategy for testing our hypotheses. Section 5 discusses our results and what it means for our understanding of the transition between illicit drugs, and Section 6 concludes.

[1] Federal ruling, risk management plan proposals for generic oxycodone.

## 1.2 Background and Literature Review

This section proceeds in chronological order. First, we provide a history of oxycodone and its most important formulation, OxyContin. We then describe the OxyContin reformulation in 2010 and what it meant for prescription opioid misuse, as well as how the previous literature analyzed the reformulation. Next, we present the nascent research on substitution between different opioids and how our contribution fits in this strain of work. We conclude with a summary of the literature on heroin mortality in the early 2010s and its link with the prescription opioid crisis.

Oxycodone was first marketed in the United States as Percodan by DuPont Pharmaceuticals in 1950. It was quickly found to be as addictive as morphine (Bloomquist, 1963), and in 1965 California placed it on the triplicate prescription form (Quinn, 1965).[2] Before the 1990s, doctors were hesitant to prescribe oxycodone to non-terminally ill patients due to its high abuse potential (DeWeerdt, 2019). The sales of oxycodone-based pain relievers did not take off until the mass marketing of OxyContin, Purdue's patented oxycodone-based painkiller. OxyContin was first approved by the FDA in 1995. The drug's innovation was an "extended-release" formula, which allowed the company to pack a higher concentration of oxycodone into each OxyContin pill and the patients to take the pills every 12 hours instead of 8 hours. OxyContin's original label, approved by the FDA, stated that the "delayed absorption, as provided by OxyContin tablets, is believed to reduce the abuse liability of a drug." In 2001, the FDA changed OxyContin's label to include stronger warnings about the potential for abuse and Purdue agreed to implement a Risk Management Program to try and reduce OxyContin misuse.[3]

OxyContin was one of the first opioids marketed specifically for non-cancer pain. In the early 1990s, pain started to enter the medical discussion as the "fifth vital sign" and something to be managed. As described in Meier (2003), Van Zee (2009), and elsewhere, Purdue's sales representatives pushed OxyContin and were told to downplay the risk of addiction. Quinones (2015) describes how Purdue cited a 1980 short letter published in the *New England Journal of Medicine* describing extremely low rates of opioid addiction among hospital patients undergoing hospital stays, but the company repeatedly implied this result extended to the general population or to individuals who left the hospital with take-home prescriptions of OxyContin. The

[2] Triplicate programs required pharmacists to send a copy to the government, and Alpert, Evans, et al., 2019 show that these had a persisting effect on reducing the number of opioid prescriptions.

[3] From the FDA Opioid Timeline.

short letter was uncritically or incorrectly cited 409 times as evidence that addiction was rare with long-term opioid therapy (Leung et al., 2017). As a result of Purdue's aggressive marketing and downplaying of the drug's abuse potential, OxyContin was a huge financial success and effectively catalyzed the prescription opioid crisis.

In May 2007, Purdue signed a guilty plea for misleading the public about the risk of OxyContin and paid more than $600 million in fines. Less than six months later, the company applied to the FDA for approval of a new reformulated version of OxyContin that included a chemical to make it more difficult to crush and misuse (Rappaport, 2009). Although not completely effective in reducing misuse, it was approved by the FDA and after August 2010 accounted for all OxyContin sales in the United States. Until 2016, with Mallinrockdt's Xtampza ER, Purdue was the only prescription opioid manufacturer to make abuse-deterrent oxycodone pills. The majority of all oxycodone sold over this time was generic oxycodone that remained abusable.[4]

Most research shows that OxyContin misuse fell following the reformulation. As described in T. Cicero and Ellis (2015), although some users were able to circumvent the abuse-deterrent formulation (ADF) to inject or ingest, the reformulation did reduce misuse. W. N. Evans, Lieber, and Power (2019) finds that the reformulation coincided almost exactly with a structural break in aggregate oxycodone sales, which had previously been increasing. Shortly after the OxyContin reformulation was implemented, researchers began to notice illicit drug use moving towards other drugs such as heroin or generic oxycodone (T. Cicero, Ellis, and Surratt, 2012, Coplan et al., 2013, Alpert, Powell, and Pacula, 2017, W. N. Evans, Lieber, and Power, 2019, Havens et al., 2014, Cassidy et al., 2014). Our paper extends the analysis of the impact of reformulation on opioid use by separately identifying the shifts in OxyContin and generic oxycodone misuse.

We build upon a rich literature that studies opioid misuse through surveys or analysis of the aggregated ARCOS reports. Surveys mostly polled either informants or users themselves (for details see Inciardi et al., 2009). The best surveys have been of users in smaller samples at individual treatment facilities, like in Hays (2004) and Sproule et al. (2009). However, selection bias is a problem for surveying treatment facilities, as that is a specific subset of patients whose habits may be different from

[4]Many other companies attempted to make abuse-deterrent opioid pills at the same time, as shown in Webster (2009), but Purdue was the first to market. Adler and Mallick-Searle (2018) and Pergolizzi et al. (2018) list other opioids with an ADF.

the overall drug-using population (particularly because they are seeking treatment). Some researchers have also used the quarterly ARCOS reports to study national trends in consumption, like in Alpert, Powell, and Pacula (2018), Mallatt (2018), and Atluri, Sundarshan, and Manchikanti (2014). The quaterly ARCOS reports have no information on the market share of each brand of prescription opioid, thereby restricting any analysis to the aggregate level only. Our work is closely connected to the second set of papers, but we are able to leverage ARCOS's transaction level data to distinguish sales of OxyContin from generic oxycodone.

This newly released ARCOS data allows us to make two methodological improvements. First, the literature treats the OxyContin reformulation as an exogenous shock at the state level. This assumption is problematic because each state's dependency on OxyContin as well as exposure to the reformulation is the result of the state's regulatory environment (Alpert, Evans, et al., 2019). These regulatory factors could have an impact on how people react to the reformulation, and thus create a hidden link between OxyContin exposure and the reformulation outcomes. Using the new ARCOS data, we can disaggregate to Metropolitan Statistical Areas (MSAs), which allows our model to identify drug substitutions using within-state variations in opioid sales and mortality while controlling for across-state variations in policies and drug enforcement.

The second benefit of the new ARCOS data set is that it allows us to disaggregate different kinds of prescription opioid sales on a national scale. Previous national studies were unable to distinguish between these drugs due to limitations in existing data. The NSDUH survey, the primary data source for drug misuse at the national level, only documented past year use of OxyContin. Death certificates do not distinguish between OxyContin and generic oxycodone. The aggregate ARCOS data lumps all oxycodone sales into one group. Because of OxyContin's unique role in fomenting the opioid epidemic, it has received most of the attention of researchers. The literature assumes that the study of OxyContin was equivalent to the study of all oxycodone. As a result, although non-OxyContin oxycodone misuse is significant in size, it has been understudied. One notable exception is Paulozzi and Ryan (2006), which notes that non-OxyContin oxycodone was a better predictor of state opioid deaths than OxyContin.

The previous literature also attempts to link the misuse of prescription opioids to the rise in heroin misuse. Siegal et al. (2003) are the first to suggest the pathway from

prescription opioids to heroin, and they further note a reverse in trend where heroin users switched to prescription opioids when heroin was unavailable. Compton, Jones, and Baldwin (2016) describe how by the 21st-century people who initiated heroin use were very likely to have started by using prescription opioids non-medically. The most recent works on OxyContin reformulation suggest that the reformulation played an important part in reigniting the heroin epidemic since 2010. T. Cicero and Ellis (2015) and Mars et al. (2014), who rely on smaller surveys, find the predominant drug people switched to after reformulation was heroin. W. N. Evans, Lieber, and Power (2019) identify a structural break in heroin deaths in August 2010 that was accompanied by higher growth in heroin deaths in areas with greater pre-reformulation access to heroin and opioids. Similarly, Alpert, Powell, and Pacula (2018) shows that the rise in heroin deaths was larger in places with higher OxyContin misuse pre-reformulation. However, the evidence linking the reformulation to the rise in heroin death is not conclusive: other researchers suggest the sharp rise in heroin use may have predated the OxyContin reformulation by a few years (Dasgupta et al., 2014, Cassidy et al., 2014). With the new ARCOS data, we are able to examine the claim that the OxyContin reformulation caused the subsequent heroin epidemic in more detail. In particular, we separate the impact of the reformulation on heroin use from the gradual shifts in oxycodone misuse that are independent of the reformulation.

### 1.3 Data and Descriptive Statistics

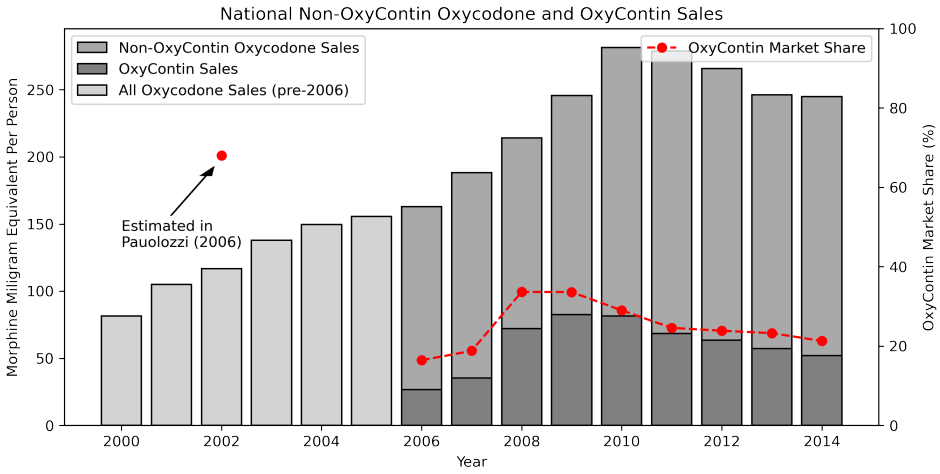
To estimate the impact of the OxyContin reformulation on opioid use and mortality, we combine several data sources including sales of OxyContin and non-OxyContin alternatives from ARCOS, opioid and heroin mortality from the NVSS, and self-reported OxyContin and Percocet misuse from the NSDUH. Our main regression leverages variations in pre-reform exposure to OxyContin and generic oxycodone to identify the impact of the reformulation on opioid sales and mortality. We define a new measure of exposure by interacting the state-level self-reported opioid misuse and MSA-level opioid sales. In this section, we describe the three sources of data, the market definition, the construction of the OxyContin and generic oxycodone exposure measure, and present summary statistics of our data.

## Data

### ARCOS and the Sales of Prescription Opioid

As part of the Controlled Substances Act, distributors and manufacturers of controlled substances are required to report all transactions to the DEA. This Automation of Reports and Consolidated Orders System (ARCOS) database contains the record of every pain pill sold in the United States. The complete database from 2006 to 2014 was recently released by a federal judge as a result of an ongoing trial in Ohio against opioid manufacturers.[5]

The ARCOS database has been used previously to study opioids, but only using the publicly available quarterly aggregate weight of drugs sold (Atluri, Sundarshan, and Manchikanti, 2014) or via special request to the DEA (Modarai et al., 2013). The newly released full database reports the manufacturer and the distributor for every pharmacy order. These data allow us to track different brands of prescription opioids separately, and calculate what fraction of oxycodone sold is OxyContin at any level of geographic aggregation. We can thus construct what we believe is the first public time-series of OxyContin and generic oxycodone sales from 2006 to 2014.




*Note:* We supplemented the 2006 to 2014 data with publicly available aggregate data from 2000 to 2005. The publicly available aggregate data does not break down the oxycodone sales by manufacturer.

Figure 1.1: Growth of oxycodone and OxyContin sales.

As we can see from Figure 1.1, total oxycodone sales increased substantially from

[5]Link to the ARCOS Data published by the *Washington Post*.

2000 to 2010, with per-person sales nearly quadrupling in the ten years period. From 2010 to 2015, sales of oxycodone declined as a result of aggressive measures taken by the states and the federal government to counter opioid addiction (Kennedy-Hendricks et al., 2016).

The newly available ARCOS data suggests that the commonly held belief about OxyContin's dominance in the prescription opioid market at the time of reformulation is incorrect. The last time OxyContin's market was estimated was in 2002 by Paulozzi and Ryan (2006), who acquired from the DEA a year's worth of ARCOS data aggregated at the state level. In that year, OxyContin was 68% of all oxycodone sales by active ingredient weight and scholars have assumed that Purdue's market share stayed high until the OxyContin reformulation. However, as Figure 1.1 shows, by 2006 when our data starts, OxyContin sales only accounted for 18% of all oxycodone sold by weight and never got above 35% during this period. The share is even smaller if we count the number of pills sold, since the average OxyContin active ingredient weight is five to ten times higher than that of oxycodone from other brands. The share of OxyContin decreased dramatically from 2002 to 2006 because Purdue lost the patent rights in 2004. As a result, non-OxyContin oxycodone sales grew much faster in the early 2000s than OxyContin sales. Figure A.1 in Appendix presents the market share for all oxycodone manufacturers by dosage strength, and Purdue Pharma is only dominant at higher dosages ($\geq$ 40mg). The overestimation of OxyContin's importance in the pre-reform period explains why the previous literature overlooked the role generic oxycodone played in the opioid epidemic.

The ARCOS sales data are the primary variables in our main regressions. We aggregate sales by MSA, year, and brand. To focus on the impact of the reformulation on OxyContin and non-OxyContin alternatives, we group all alternative oxycodone products into one measure, and we will refer to it as generic oxycodone for the rest of the analysis.[6]

**NVSS Mortality Data**

The second outcome of interest in our main regression is opioid mortality. We use the restricted-use multiple-cause mortality data from the National Vital Statistics System (NVSS) to track opioid and heroin overdose. The dataset covers all deaths

[6]We acknowledge some non-OxyContin alternatives are branded and non-generic (i.e., Percocet and Percodan or later Roxicodone), but the majority of them are generic products. Generic oxycodone in this paper should be interpreted as all non-OxyContin oxycodone products.

in the United States from 2006 to 2014. We follow the literature's two-step procedure to identify opioid-related deaths. First, we code deaths with ICD-10 external cause of injury codes: X40–X44 (accidental poisoning), X60–64 (intentional self-poisoning), X85 (assault by drugs), and Y10–Y14 (poisoning) as overdose deaths. Second, we use the drug identification codes, which provide information about the substances found in the body at death, to restrict non-synthetic opioid fatalities to those with ICD-10 code T40.2, and heroin deaths to those with code T40.1. Figure 2 shows the trend over our period of study for the two series.

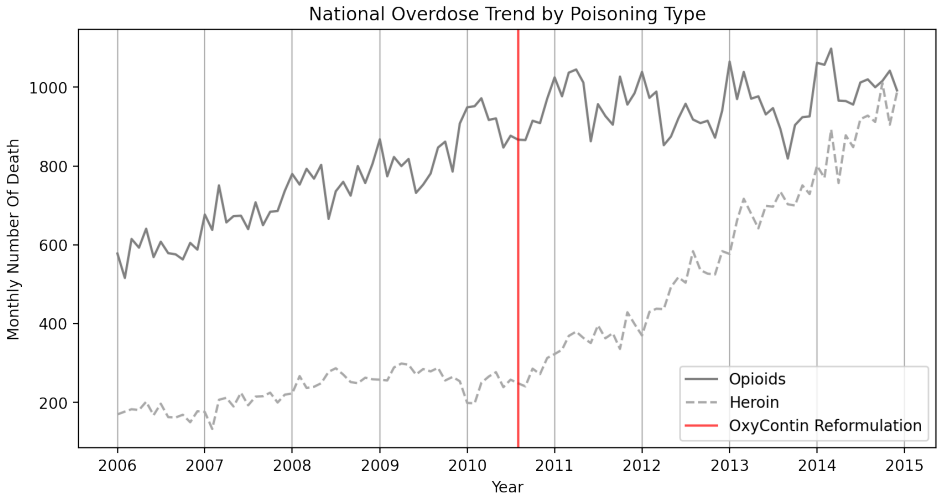



Figure 1.2: Mortality trends for opioids and heroin overdose deaths.

The number of opioid fatalities grew in our sample period, from on average 600 deaths per month to 1000 per month. The number of heroin deaths was stable from 2006 to 2009 at about 200 deaths per month, and then it rose sharply from 2011 to 2015. As we've stated in the literature review section, the cause of the increase in heroin mortality is unclear. While some papers blame the OxyContin reformulation, there is evidence indicating the availability of heroin increased substantially after 2010 (O'Donnell, Gladden, and Seth, 2017).

Since the number of drug overdose deaths with no drug specified accounts for between one-fifth and one-quarter of the overdose cases (Ruhm, 2017), our measures of opioid and heroin deaths likely underestimate the true number of deaths.[7] However, the underestimation would not pose a problem for our regressions. There are

[7]Specifically, we omit ICD-10 code T50.9 (unspecified poisioning) from our analysis, and some fraction of these deaths are due to opioids or heroin but were not diagnosed or recorded as such.

variations in how coroners attribute the cause of death across states, but such variation would be captured by the state fixed effects. In addition, we do not anticipate systematic changes to each state's practices due to the reformulation.

**NSDUH and Measuring Misuse**

We use state-level data from the National Survey on Drug Use and Health (NSDUH) to measure nonmedical use of opioids. The NSDUH publishes an annual measure of OxyContin misuse, asking the respondents whether they have ever used OxyContin "only for the experience or feeling they caused" (NSDUH Codebook). As first described in Alpert, Powell, and Pacula (2018), the advantage of the NSDUH misuse measure is that it seperates out misuse from medical use. However, only OxyContin is reported in the NSDUH and there is no equivalent measure for generic oxycodone.

Fortunately, the NSDUH reports PERCTYL2, which asks whether individuals ever misused Percocet, Percodan, or Tylox.[8] These drugs are oxycodone hydrochloride with acetaminophen and have a maximum dosage of 10mg of oxycodone per pill. The three drugs were popular among users in the pre-OxyContin era (Meier, 2003). In the present day, the PERCTYL2 variable captures misuse of not only the three branded drugs but also other generic oxycodone products that are popular on the street.

The most direct evidence supporting this claim is the fact that generic oxycodone pills have often been referred to as "Percs" colloquially in the last decade. Many news report indicated that generic oxycodone has the street name "Perc 30" but is in fact not Percocet. The *Patriot Ledger* reported in a 2011 article[9] that "Perc 30s" were the newest drug of choice in South Shore of Massachusetts, saying:

> Perc 30s are not Percocet — the brand name for oxycodone mixed with acetaminophen, the main ingredient in Tylenol — but a generic variety of quick-release oxycodone made by a variety of manufacturers. They are sometimes referred to as "roxys" after Roxane Laboratories, the first company to make the drug, or "blueberries," because of their color.

[8] Percocet Drug Information. Tylox was discontinued in 2012 following the FDA regulations limiting acetaminophen.

[9] *Patriot Ledger* Link. Other references to generic non-OxyContin oxycodone as Perc 30s: *Phoenix House*, *Washington State Patrol*, *Boston Globe*, *Salem News*, Massachusetts Court Filing, *Cape Cod Times*, *Pocono Record*, *Bangor Daily News*, *Patch*, *CNN* Op-Ed

Since many generic oxycodone users wouldn't know the name of the drug they use other than by its street name, but could distinguish between immediate release oxycodone and extended release OxyContin, it is likely that they answer affirmatively to misusing Percocet when they are, in fact, using generic oxycodone.[10]

There are also several empirical observations that support this claim. The first is that we continue to see increases in the lifetime misuse of Percocet, Percodan, and Tylox even after they were replaced by OxyContin as the preferred prescription opioid to misuse. The misuse rate of Percocet, Percodan, and Tylox increased 30% from 4.1% to 5.6% from 2002 to 2009 (see Figure A.2 in Appendix), which would not have been possible if these drugs, or what people believed were "Percs", were not actively misused by new users post-introduction of OxyContin.

The second observation is that, based on the average sales data from 2006 to 2014, a disproportionate number of people have reported misusing Percocet, Percodan, or Tylox as compared to the actual sales of the three drugs. The sales of Endo Pharma, the manufacturer of Percocet and Percodan[11], are orders of magnitude less than the sales of Purdue while more than twice as many people reported misusing the three drugs as compared to OxyContin (see Figure A.3 in Appendix). A back-of-the-envelope calculation shows that if PERCTYL2 misuse captures only the misuse of Percocet and Percodan, then the proportion of pills misused out of all pills sold is 29 times higher for Percocet and Percodan than than the same proportion for OxyContin[12], a very unlikely situation given the popularity of OxyContin on the street.

This deduction is further supported by misuse data reported in the NSDUH. We know that generic oxycodone is commonly misused.[13] If oxycodone has any other drug names, the popularity of that drug name in the NSDUH surveys should increase to reflect the increase in misuse in recent years. In addition to inquiring about popular brands, the NSDUH survey asks respondents to list any other prescription oxycodone that they have misused before. Dozens of pain relievers are reported, but in 2010 "oxycodone or unspecified oxycodone products" was only named by 0.10%[14] of the

[10] In the ARCOS dataset these pills are simply listed as "Oxycodone Hydrochloride 30mg"

[11] Tylox not included since it was discontinued.

[12] In terms of number of pills circulated, OxyContin is 12.1 times Percocet and Percodan from 2006 to 2014. In terms of misuse, OxyContin is 41% of Percocet and Percodan in the same period.

[13] Law enforcement and journalists have previously identified the 30mg oxycodone pill as the most commonly trafficked opioid, see DEA Link, ICE Link, and *Palm Beach Post* Link.

[14] NSDUH Codebook variables ANALEWA through ANALEWE list the other pain relievers

respondents. No other brand of oxycodone pill is reported as commonly misused. We know from the reports in press and documents in court that generic oxycodone is a popular opioid on the street, and we know that Percocet is the only other commonly misused opioid documented in the NSDUH survey. Thus, the only way to reconcile the discrepancy between these two sources is that people mistakenly perceive generic oxycodone as Percocet or respond to the NSDUH as if they do. Thus, we use lifetime OxyContin and lifetime Percocet misuse for the construction of OxyContin and generic oxycodone exposure measures in this chapter.

**Market Definition and Endogeneity Problems**

Previous studies of the OxyContin reformulation depend on state-level variation to causally identify the impact of the reformulation. Treating OxyContin reformulation as an exogenous shock at the state level is potentially problematic. Although the timing of the reformulation is exogenous, each state's exposure to it is a result of a combination of the state's regulatory environment and Purdue's initial marketing strategy (Alpert, Evans, et al., 2019). These factors have substantial impact on how people in a state respond to the reformulation, creating a hidden link between exposure to the reformulation, the identifying variation, and subsequent drug use, the outcome variable.

One can limit the impact of endogenous regulation by disaggregation, but only if there is substantial intra-state variation in exposure to the reformulation. Both the ARCOS database and the NVSS mortality data have great geographic detail. Conducting our analysis on metropolitan statistical areas (MSAs), we find large variation in both OxyContin use and opioid mortality across MSAs in the same state. At the aggregate level in 2009, the average OxyContin market share in a state is 35.6%. 65 of the 379 MSAs (17.1%) in our sample have an OxyContin market share that is 10% greater or smaller than their state average. The average opioid mortality is 0.343 deaths per 100,000 population in 2008. The variation in death is even more significant. More than 310 (83%) MSAs have a mortality rate 20% higher or lower than their state average, and more than 192 (51%) have a mortality rate 50% higher or lower than their state average. We present the full distribution of deviations of OxyContin market share and opioid mortality from state average in Figure A.4 and Figure A.5 in the Appendix.

reported. Even if we assumed all 2.49% of respondents saying they took a prescription pain reliever not listed had taken generic oxycodone, it is still less than half of the reported Percocet misuse.

Disaggregating to the MSA-level allows us to control for the state's regulatory environment and hence eliminate the most problematic source of endogeneity. We use intra-state variation in exposure to the reformulation for identification. Intra-state heterogeneity in opioid use is associated with past economic conditions (Carpenter, McClellan, and Rees, 2017), location of hospitals and treatment centers (Swensen, 2015), preferences of local physicians (Schnell, 2017), and local policy, some of which could still be correlated with the locality's response to the reformulation. Analysis at the MSA level clearly allows us to make a much stronger claim than analysis at the state level.

In addition, as we will show in the next sections, the disaggregation increases the statistical power of our regressions beyond the impact of the tripled sample size. Our results indicate that defining the market at the MSA level better captures the interaction between drug use and mortality than the state level. The important variations in drug use, for example between Los Angeles-Long Beach-Santa Ana at 4.4% of nonmedical use of pain relievers and San Francisco-Oakland-Fremont at 5.6%, disappears when they're aggregated to the state level (*2005-2010 NSDUH MSA Detailed Tables* 2012).

**OxyContin and Non-OxyContin Oxycodone Exposure**

Since the OxyContin reformulation was a national event independent of local conditions, we can estimate its impact by comparing the outcomes in areas of high prior exposure to opioids with outcomes in areas of low exposure. Ideally, we want to quantify exposure using the volume of OxyContin misused in each region pre-reform while controlling for the volume of generic oxycodone misused. In practice, we do not observe these quantities. The best proxy in the literature is the self-reported misuse rate from the NSDUH.

Based on the NSDUH misuse, we create a new measure of OxyContin and non-OxyContin oxycodone exposure by combining the NSDUH state-level misuse rate with ARCOS MSA-level sales. Specifically, for each drug, we calculate:

$$\text{Exposure}_m^{\text{pre-reform}} = \text{Lifetime Misuse}_s^{2004-2009} \times \text{Sales}_m^{2009} \tag{1.1}$$

Our measure is the interaction term of sales of OxyContin/generic oxycodone in an MSA and the lifetime misuse rate of that drug in the corresponding state. This new measure has two advantages over the conventional misuse rate from NSDUH: it captures intra-state variation in misuse and it more accurately reflects the current

misuse of both OxyContin and generic oxycodone.

The NSDUH surveys approximately 70,000 respondents every year and uses demographic reweighting techniques to get accurate state level estimates. Once we get to the MSA level, the number of people surveyed as well as the number of positive responses to questions on opioid misuse are extremely small. As a result, most of the outcomes at the MSA level are censored by the NSDUH to protect individual privacy. Using only the survey data means that we would use same state misuse value for all MSAs and therefore forgo any intra-state variation in drug use. In comparison, our proposed measure relies on deviations from normal sales patterns to generate variations in exposure rates for the MSAs. Our definition assumes that the percentage of people reported misusing a particular drug in a state is equivalent to the proportion of sales that are being misused. In a state where all the MSAs have identical sales, all the MSAs will have identical exposure rates by definition. However, if one MSA has higher sales of OxyContin compared with the rest of the state, our OxyContin exposure measure in that MSA will be higher than the rest of the state. This construction of exposure mirrors our intuitive understanding that the misuse of a drug in a locality is a function of the overall misuse and the availability of that particular drug in the area.

The NSDUH survey[15] reports past-year misuse of OxyContin but only lifetime misuse of generic oxycodone. Previous studies did not focus on generic oxycodone misuse, so these studies rely on past-year OxyContin misuse rate. In our case, to disentangle substitution among prescription opioids, we have to make the comparison between OxyContin and generic oxycodone equal. Resorting to lifetime misuse rates for both series sacrifices the timely nature of the NSDUH misuse rates. By combining the lifetime misuse rates with sales in the year before reformulation, we capture recent changes in use of both drugs. To make our results comparable with previous studies, in the Appendix section, we repeat our entire analysis with OxyContin last-year misuse and generic oxycodone lifetime misuse. Most of our conclusions stand despite giving OxyContin a more favorable treatment.

To construct our measure, we follow the precedent set in the literature by using a six-years-average state-level lifetime misuse rate pre-reform (2004 to 2009) and sales in 2009. The goal of the time average is to reduce the variance of the state-level misuse rates. We check the validity of our measure by regressing opioid death on it

[15] In all surveys prior to 2014.

Table 1.1: Summary Statistics

| | All MSAs | MSAs with low OxyContin exposure | MSAs with high OxyContin exposure | MSAs with low oxycodone exposure | MSAs with high oxycodone exposure |
|---|---|---|---|---|---|
| *NSDUH lifetime misuse rates (2004-2009)* | | | | | |
| OxyContin misuse rate (%) | 2.22 | 1.88 | 2.56 | 1.87 | 2.56 |
| Oxycodone misuse rate (%) | 5.19 | 4.22 | 6.17 | 3.75 | 6.64 |
| *Annual ARCOS sales (all sample period)* | | | | | |
| Oxycontin sales per person | 65.71 | 43.47 | 88.06 | 50.70 | 80.79 |
| Oxycodone sales per person | 181.84 | 112.50 | 251.55 | 99.24 | 264.88 |
| *Annual death per 100,000 (all sample period)* | | | | | |
| Opioid | 0.32 | 0.23 | 0.41 | 0.23 | 0.42 |
| Heroin | 0.13 | 0.09 | 0.16 | 0.10 | 0.16 |
| *Census Demographics (2009)* | | | | | |
| Number of MSAs | 379 | 190 | 189 | 190 | 189 |
| Population | 679878 | 745327 | 614082 | 663740 | 696101 |
| Age | 36.13 | 34.68 | 37.59 | 34.84 | 37.43 |
| Male (%) | 49.24 | 49.35 | 49.13 | 49.40 | 49.08 |
| Separated (%) | 18.83 | 18.24 | 19.42 | 18.32 | 19.34 |
| High school and above (%) | 84.20 | 82.79 | 85.61 | 83.68 | 84.72 |
| Bachelor and above (%) | 25.36 | 24.77 | 25.96 | 24.85 | 25.87 |
| Mean income | 64213 | 63414 | 65016 | 63058 | 65374 |
| Low income (%) | 35.38 | 35.79 | 34.98 | 35.90 | 34.86 |
| White (%) | 82.17 | 79.99 | 84.36 | 81.22 | 83.12 |
| Black (%) | 11.20 | 13.09 | 9.30 | 11.80 | 10.60 |
| Asian (%) | 3.03 | 3.47 | 2.60 | 3.52 | 2.54 |
| Native American (%) | 0.18 | 0.20 | 0.17 | 0.20 | 0.17 |

*Note:* Simple average, not weighted by population.

and compare the results with the same regressions on either only ARCOS sales or only NSDUH misuse. Results are summarized in Table A.1 in Appendix. The fit of the generic oxycodone regression is much improved with the interacted variable ($R^2 = 0.187$) relative to using only one with NSDUH misuse ($R^2 = 0.062$) or sales ($R^2 = 0.176$). The improvement is even larger for the OxyContin regression ($R^2 = 0.128$) relative to using only one with NSDUH ($R^2 = 0.084$) or with sales ($R^2 = 0.086$).

**Descriptive Statistics**

Table 1.1 reports summary statistics for five groups of MSAs: all MSAs, MSAs with high OxyContin exposure, MSAs with low OxyContin exposure, MSAs with high generic oxycodone exposure, and MSAs with low generic oxycodone exposure.

MSAs with high OxyContin exposure and MSAs with high generic oxycodone exposure have similar demographic summary statistics. These two groups of MSAs also are not different statistically in their heroin mortality. Disentangling the impact of various opioids on the rise in heroin mortality is impossible with nationally aggregated or state-level data due to the high correlation in misuse between the two prescription opioids. The high correlation also implies that regressing heroin death on OxyContin without controlling for generic oxycodone use will likely lead to an overestimation of OxyContin's impact.

MSAs with high misuse differ from MSAs with low misuse. High misuse states have higher sales of both types of prescription opioids (twice as much for both types of opioids), higher mortality rate (twice as much for both opioid and heroin overdose), smaller population, higher average age, higher median income, higher percentage of white population, and lower percentage of black population. The differences in racial composition repeat well-established findings in the literature: prescription opioid misuse was originally concentrated among white users, and by 2010 new heroin users were almost entirely white (T. J. Cicero et al., 2014). These differences in demographic variables motivate the inclusion of control variables in our main regressions.

### 1.4 Empirical Strategies

Our goal is to investigate two questions. First, what was the reformulation's immediate impact on OxyContin and generic oxycodone use? Second, what was the reformulation's long-run effect on opioid mortality, heroin mortality, and on the progression of opioid addiction?

We follow the event study framework from Alpert, Powell, and Pacula (2018) to estimate the causal impact of the OxyContin reformulation on OxyContin and generic oxycodone sales and opioid and heroin mortality. We exploit variation in MSAs' exposure to the reformulation due to the differences in their pre-reform OxyContin use while controlling for pre-reform generic oxycodone use. Our approach is similar to Finkelstein (2007), where the OxyContin reformulation has more "bite," or more of an effect, in areas where OxyContin misuse was higher than in places where generic oxycodone was the preferred drug. The approach allows us to measure whether MSAs with higher exposure to OxyContin experienced larger declines in OxyContin sales, larger increases in alternative oxycodone, or larger increases in opioid and heroin mortality. The empirical framework is:

$$
\begin{aligned}
Y_{mt} = \alpha_s + \delta_t + \sum_{i=2006}^{2014} \mathbb{1}\{i = t\}\beta_i^1 \times \text{OxyContin Exp}_m^{Pre} \\
+ \sum_{i=2006}^{2014} \mathbb{1}\{i = t\}\beta_i^2 \times \text{Oxycodone Exp}_m^{Pre} + X'_{mt}\gamma + \epsilon_{mt}
\end{aligned}
\tag{1.2}
$$

where $Y_{mt}$ are the outcome variables of interest in MSA $m$ at year $t$; $\text{OxyContin Exp}_m^{Pre}$ and $\text{Oxycodone Exp}_m^{Pre}$ are time-invariant measures of OxyContin and oxycodone exposure before the reformulation (see Section 3.5 for construction), and are interacted with a set of $\beta_t^1$ and $\beta_t^2$ for each year. We include state-fixed effects to control for regulatory differences among states and year-fixed effects to control for national changes in drug use. We also include a full set of MSA-level demographic variables. We weight the regression by population and exclude Florida.[16] We show the full set of $\beta_t$ estimates graphically, normalizing by the 2009 coefficient. The $\beta_t$ identifies the differences in sales and death across MSAs due to their higher or lower pre-reform OxyContin or oxycodone exposure. Standard errors are clustered at the MSA level to account for serial correlation. In the Appendix section, we present beta estimations from variations of our base model, which include (1) using a MSA-fixed effect instead of state-fixed effect, (2) replacing OxyContin lifetime misuse rate with OxyContin last-year misuse rate, (3) regressing at the state level, and show that our conclusions are insensitive to most of these variations.

To complement our results, we also use a strict difference-in-difference framework to estimate effect of the reformulation conditioning on OxyContin and non-OxyContin oxycodone exposure levels. Our specification is:

$$
\begin{aligned}
Y_{mt} = \alpha_s + \gamma_t + \delta_1 \mathbb{1}\{t > 2010\} \\
+ \delta_2 \mathbb{1}\{m \in HighOxyContin\} + \delta_3 \mathbb{1}\{m \in HighOxycodone\} \\
+ \delta_4 \mathbb{1}\{t > 2010\} \times \mathbb{1}\{m \in HighOxyContin\} \\
+ \delta_5 \mathbb{1}\{t > 2010\} \times \mathbb{1}\{m \in HighOxycodone\} + X'_{mt}\beta + \epsilon_{mt},
\end{aligned}
\tag{1.3}
$$

where $HighOxyContin$ and $HighOxycodone$ are the set of MSAs with higher than median pre-reform exposure to OxyContin and oxycodone respectively. We restrict the regression to include only the three years prior (2008 to 2010) and the three

[16] The literature excludes Florida because it underwent massive increases in oxycodone sales over this period, some of which was trafficked to other states.

years after (2011 to 2013) the reformulation. The advantage of this specification is that it does not assume that OxyContin or oxycodone exposure affects the outcome variable linearly. Instead of having a flexible $\delta$ for each year, we have only one $\delta$ for each of the pre- or post-reform period. In this specification, we simply test whether higher exposure MSAs reacted differently to the reformulation as compared to lower exposure MSAs (if $\delta_4$ and $\delta_5$ are significant). We include state-fixed effects to control for state-level heterogeneity, year fixed effects for national trend, and a set of time-varying MSAs level covariates. Again, standard errors are clustered at the MSA level.

## 1.5 Results

We proceed in two steps. First, we provide direct evidence that the OxyContin reformulation caused OxyContin sales to decrease and generic oxycodone sales to increase, and that the changes in sales are proportional to the pre-reformulation level of OxyContin exposure. Second, we estimate the impact of the reformulation on opioid and heroin mortality. We find that high pre-reformulation levels of OxyContin exposure were not associated with high opioid deaths, but there was a strong positive effect from generic oxycodone exposure in both the pre- and post-reform period. We find that higher pre-reform OxyContin and pre-reform oxycodone exposure were both positively but not significantly associated with later heroin deaths, but the oxycodone coefficient is larger. If we run the heroin regression separately with only OxyContin exposure we recover the results of the literature, but running the heroin regression with only oxycodone exposure better fits the data.

### Reformulation's Impact on Opioid Sales

We begin by showing graphically that OxyContin sales decreased and generic oxycodone sales increased in high OxyContin misuse MSAs immediately after reformulation. Figure 1.3 and Figure 1.4 present the full set of coefficients from estimating the event study framework on OxyContin and generic oxycodone sales. Each data point in the figure is the coefficient of the interactive term of misuse and sale, which we call exposure, for OxyContin or generic oxycodone in a specific year, and it captures any additional change in sales in that year driven by high OxyContin or oxycodone exposure. In Figure 1.3, we observe a larger decrease in OxyContin sales post-reform in MSAs with higher pre-reform OxyContin exposure. As Figure 1.4 shows, higher OxyContin exposure MSAs saw greater increases in generic oxycodone sales post-reform. The effects are statistically significant at the 95%

confidence level. An one standard deviation increase in OxyContin exposure translates into an additional 21.2 MME decrease in per person OxyContin sales and 11.8 MME increase in per person oxycodone sales in 2011. These changes represents a 24% decrease in OxyContin sales and a 8.8% increase in oxycodone sales from the 2009 level. The effects are economically significant especially given that the reformulation should only affect the population abusing OxyContin, so this drop in sales is driven by a fraction of all users. The two observations combined support the hypothesis that reformulation caused substantial substitution from OxyContin to generic oxycodone.

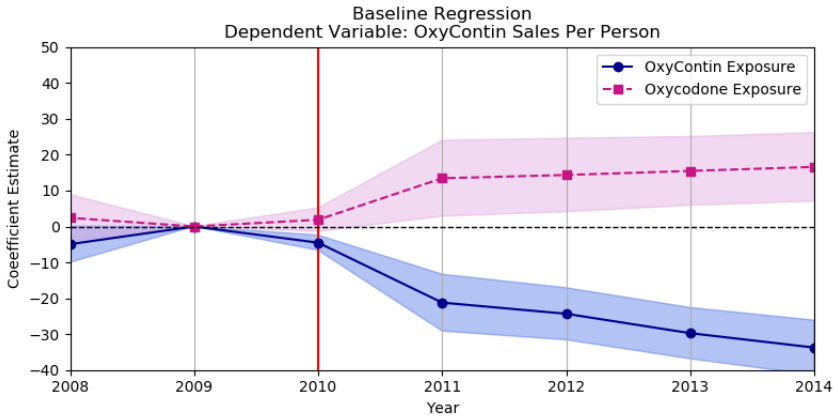



Figure 1.3: Main regression on OxyContin sales. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

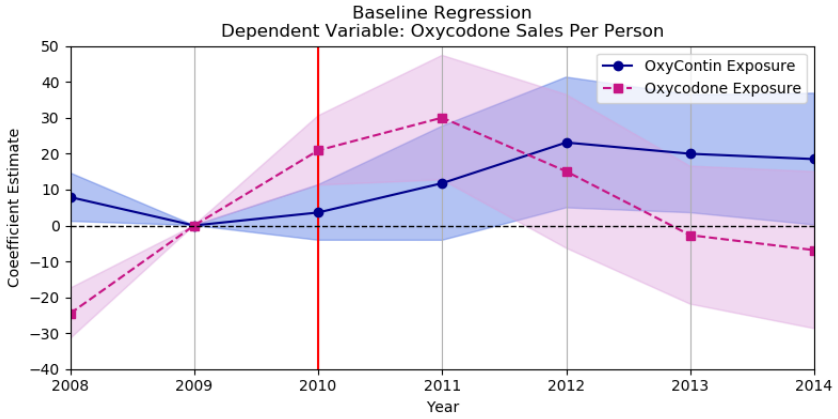



Figure 1.4: Main regression on generic oxycodone sales. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

Figure 1.3 also documents that high pre-reform oxycodone misuse MSAs saw large increases in OxyContin sales right after the reformulation. This phenomenon has

been unreported previously, but would be consistent with Schnell (2017)'s physician benevolence hypothesis where good physicians switch patients from oxycodone to reformulated OxyContin to lower the future risk of abuse. Although the switch toward OxyContin is smaller in magnitude than the switch from OxyContin, this increase is the first documented positive impact of the OxyContin reformulation in the literature. It seems both physicians and users saw the two types of drugs as substitutes. Unfortunately, there are not enough MSAs where the switch toward OxyContin is significant enough that it cancels out the switch away from OxyContin to examine the possible substitution channel in the other direction.

Because we include both OxyContin and generic oxycodone misuse in the same regression, we can separate the increases in oxycodone sales due to its own popularity from the increases due to spillover effects from the OxyContin reformulation. Figure 1.4 shows increasing growth in oxycodone sales in MSAs with higher oxycodone misuse until 2011, and the growth rate declined after. The smoothness of the oxycodone curve indicates that the OxyContin reformulation had no impact on how oxycodone misuse affected oxycodone sales. This trend corresponds well with many states tightening control over opioid prescription policies in 2011 and 2012 in response to rising sales and and increased awareness of opioid misuse.

Another way of estimating the impact of the reformulation is through difference-in-difference regressions. Column (1) of Table A.2 in Appendix shows the regression on OxyContin sales. OxyContin sales in all MSAs decreased by 8.05 MME post-reform, a 9.4% decrease with respect to the average per person sales of 85.6 MME in 2009. High OxyContin misuse MSAs had a higher level of OxyContin sales to start with, but experienced an additional 15.1 MME drop (an additional 17% decrease) post-reform. Given that only 2.46% of the population ever misused OxyContin[17] and the reformulation only affected the people misusing it, a 17% additional decrease in all OxyContin sales would translate into a very significant decrease in sales to the population that misuses it. The negative and significant $Post \times High\ OxyContin$ coefficient confirms previous findings that high OxyContin exposure MSAs saw larger decreases in OxyContin sales post-reform.

Column (2) of the same table reports the regression on generic oxycodone sales. Generic oxycodone sales per person increased 41.7 MME in the post period, a 31.2% increase with respect to the average per person alternative oxycodone sales of 133.5

[17] NSDUH, 2010.

MME in 2009. High OxyContin misuse MSAs experienced an additional 10.3 MME increase, which translates to a 68% conversion from OxyContin to generic oxycodone in those areas. Combining the findings from columns (1) and (2), we see direct substitution from OxyContin to generic oxycodone in local sales immediately after reformulation, and the substitution pattern is more pronounced in MSAs with high OxyContin exposure as expected.

To visualize the trend of OxyContin and alternative oxycodone sales, in Figure A.6 in the Appendix, we break all MSAs into three bins by the magnitude of the observed drop in OxyContin sales due to the reform. Then, we plot the per person OxyContin and generic oxycodone sales for the three group respectively. By definition, the high empirical drop group experienced the largest decreases in OxyContin sales from 2009 to 2011 (-29%) and the low drop group experienced an increase in OxyContin sales (+15%). Sales of generic oxycodone started at different levels, but shared the same growth rate until the reformulation in 2010. Since 2010, the higher the empirical drop in OxyContin, the faster the growth in generic oxycodone. The high group saw in a 72 MME increase (46% from 2009) in generic oxycodone sales, while the low group only saw an 29 MME increase (29% from 2009). The high growth rate of generic oxycodone in high drop MSAs support the substitution story. The post-reform level of OxyContin sales converges to the same level for all three groups, suggesting that the remaining sales most likely represent non-replaceable demand for medical OxyContin use.

**Reformulation and Opioid and Heroin Mortality**

Next, we estimate the impact of the reformulation on overdose mortality. In Figure 1.5, we report the full set of coefficients from estimating the event study framework on opioid mortality. Each data point in the figure is the coefficient of the interactive term of misuse and sale for OxyContin or generic oxycodone in a specific year, and it captures any additional change in opioid mortality in that year driven by high OxyContin or oxycodone exposure. The OxyContin coefficients are never significant, suggesting higher pre-reform OxyContin misuse is not predictive of either higher or lower opioid death post-reform. The lack of any trend indicates that any benefit of the OxyContin reformulation on reducing OxyContin consumption is offset by the substitution to generic oxycodone. In aggregate, the reformulation had no impact on non-heroin opioid deaths.

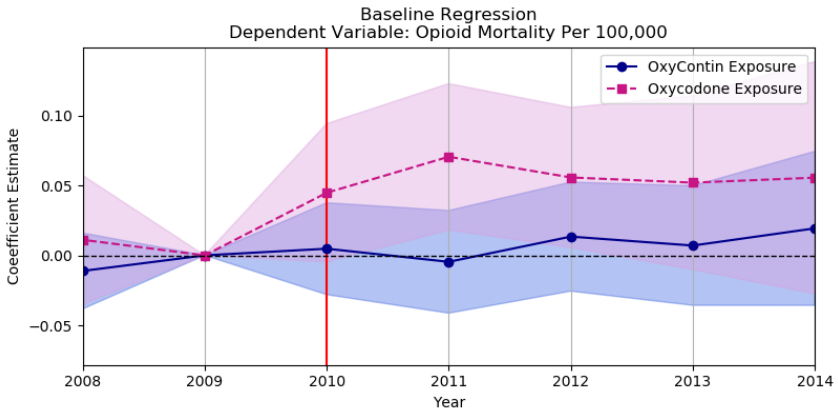



Figure 1.5: Main regression on opioid mortality. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

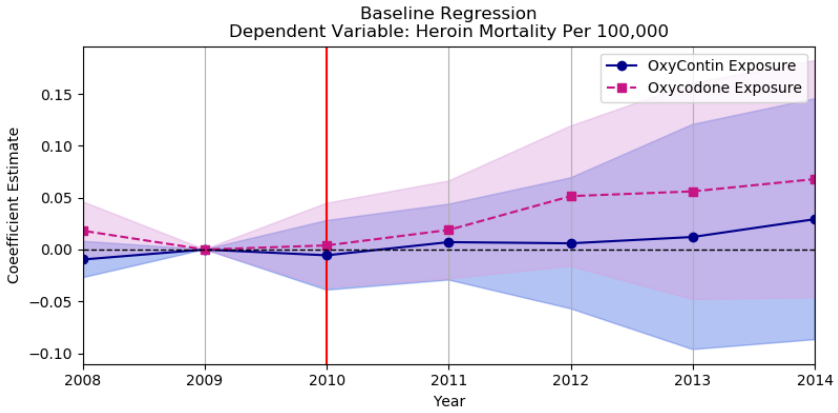



Figure 1.6: Main regression on heroin mortality. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

In Figure 1.6, we report the event study coefficients on heroin mortality. Again the OxyContin coefficients are tiny and insignificant, while the oxycodone coefficients grow over time but never reach statistical significance at conventional levels. The lack of statistical significance is due to the small number of heroin moralities in the whole sample and high correlations between OxyContin and oxycodone exposure. If we were to run the OxyContin and oxycodone regression separately (see Figure A.28 and Figure A.32 in Appendix), oxycodone exposure had a much larger and more significant impact on heroin mortality. The results provide tentative evidence that the higher the generic oxycodone exposure in an MSA, the higher the increases in heroin mortality. However, the results do not support the alternative hypothesis that the OxyContin reformulation was solely responsible for the increase in heroin mortality.

The difference-in-difference results mirror our finding from the event study framework. Column (3) of Table A.2 in Appendix suggests that opioid deaths are 0.08 higher in high oxycodone exposure MSAs, which is equivalent to 27% of the average opioid overdose of 0.29 per 10,000 people in 2009. Opioid mortality is 0.05 lower (17% of the 2009 average) in higher OxyContin exposure MSAs after controlling for oxycodone use. Higher OxyContin exposure does not lead to higher or lower opioid overdose post-reform, while higher generic oxycodone exposure is associated with 0.06 (20.6% of 2009 average) more opioid death in the post period.

Column (4) of the same table reports the difference-in-difference regression on heroin death. Heroin mortality has increased by 0.14 in the post period in all MSAs, which is equivalent to a 111% increase from the average 2009 level of 0.126 heroin death per 10,000 population. High OxyContin exposure MSAs did not experience additional jumps in heroin mortality, while high oxycodone exposure MSAs did experience an additional 0.07 (56% with respect to 2009 average) increase in death. Again, the evidence from the difference-in-difference regressions indicates that OxyContin was not responsible for the rise in heroin mortality.

In Figure A.7 in the Appendix, we show the average trend of the opioid and heroin mortality for groups with high, medium and low observed drop in Oxycontin sales. If the reformulation was responsible for the subsequent heroin epidemic, then the MSAs mostly likely to have additional jumps in heroin mortality would be the MSAs with the largest OxyContin drop. As shown in the figure, the three groups went through the same explosive growth in heroin mortality (around 38% from 2009 to 2011, and similar rate afterward), indicating the rise in heroin was independent of the decrease in OxyContin sales. This evidence conclusively rejects the hypothesis that the OxyContin reformulation is solely responsible for the subsequent heroin epidemic.

**Discussion**

*(A) The Reformulation's Impact on Opioid Mortality*

Until now, the literature has found mixed results for the effects of the OxyContin reformulation on opioid mortality. In contrast to previous work, we find no statistically significant impact of the reformulation on opioid mortality as a result of substantial substitutions from OxyContin to generic oxycodone post-reform. Increases in generic oxycodone sales compensated for 55% of the drop in OxyContin

sales in high OxyContin misuse MSAs by our event study framework, and 68% by our difference-in-difference estimation. Opioid mortality continued to increase in the post-reform period, but not was driven by high OxyContin exposure.

*(B) The Reformulation's Impact on Heroin Mortality*

Our results stand in direct contrast to the findings of the literature. Instead of being the event that precipitated the heroin epidemic, the OxyContin reformulation shifted misuse to other opioids, of which heroin was only one. We cannot refute the hypothesis that some OxyContin users switched to heroin due to the reformulation. Our analysis refutes the hypothesis that the reformulation was the sole cause of the heroin epidemic. Instead of OxyContin misuse, we identified generic oxycodone misuse as a much more powerful driver of increases in heroin mortality post-2011. What prompted the increases in heroin use is still an unresolved question. Previous research has suggested an increase in the supply of heroin (O'Donnell, Gladden, and Seth, 2017) around this time, as well as crackdowns in Florida on pill-mills reducing the supply of oxycodone (Kennedy-Hendricks et al., 2016).

*(C) Bridging the Differences between our Findings and the Literature*

One of the innovations in this paper is to shed light on a hidden source of opioid misuse: the misuse of generic oxycodone. This segment of prescription opioids was overlooked by other scholars because of OxyContin's dominance in opioid misuse in the early years as well as, we argue, the lack of identifiable brand names for the generic products. Empirical studies based on market data or interviews of opioid users noted that many people misused generic oxycodone products (Paulozzi and Ryan, 2006, Inciardi et al., 2009). Leaving out oxycodone misuse, an important driver of opioid and heroin mortality that is positively correlated with OxyContin misuse, would produce spurious regression results.

To show that the difference in findings is not driven by our constructed misuse measure, or our choice of framework, we test whether we can reproduce findings in the literature by running all of our regressions using only OxyContin (see Section A in the Appendix). Our OxyContin misuse exposure individually predicts an increase in opioid and heroin mortality post-reform as the literature claims. This finding is the basis of previous studies supporting the claim that the OxyContin reformulation is the main cause of the subsequent heroin epidemic. However, if we run the same set of regressions using only generic oxycodone (see Section A), we were able to produce

the same findings. The only way to differentiate the impact of OxyContin from that of generic oxycodone is to include both in the same regressions. Variations in local OxyContin and oxycodone exposure allow us to identify the impact of both series, if any exist. As we've shown in our main regressions, the impact of OxyContin on heroin disappears after controlling for the effect of generic oxycodone.

*(D) Market Definition*

Another innovation in this paper is a finer definition of the opioid market. It is important to consider what we gain from disaggregating to the MSA level. The specific OxyContin market share in a state is endogenous to a great many things, including advertising (Van Zee, 2009) and triplicate status (Alpert, Evans, et al., 2019). Although the OxyContin reformulation was an exogenous shock, its interpretation is made very complicated because its impact depended on each state's regulatory history and prescribing environment. We do our regressions at the MSA level, where there are unobserved local conditions that affected sales of OxyContin and generic oxycodone, while controlling for state-level laws and restrictions. By comparing two different MSAs with the same regulatory environment but different exposures to the reformulation, we can get at the marginal effects of OxyContin and generic oxycodone exposure. Contrasting the state-level regression estimates (see Section A) with our main results, our main results are larger in magnitude and more statistically significant. The MSA level estimation of the effect of exposure on mortality is more stable.

*(E) Definition of OxyContin Misuse*

The literature relies on NSDUH's OxyContin past-year misuse. To make our findings comparable with previous studies and robust to the choice of misuse measure, we repeat our entire analysis with OxyContin last-year misuse and generic oxycodone lifetime misuse (see Section A for results.) As noted in Section 1.3, using last-year OxyContin misuse gives an unfair advantage to OxyContin due to the timeliness of the measure. If our findings on oxycodone persist despite the unequal treatment of the two misuse measures, then it is a stronger indication of the essential role generic oxycodone played in the opioid and heroin epidemic.

Comparing the two sets of results, we observe the same decline in OxyContin sales and increase in generic oxycodone sales, although smaller in magnitude. Both sets of coefficients on opioid mortality become positive but insignificant. Finally,

comparing the heroin result, at the state level we detect a positive effect on heroin mortality from OxyContin. In aggregate, our results lose some significance when we replace lifetime OxyContin misuse with last-year OxyContin misuse. The loss of significance, however, is in the direction predicted by the unfair advantage given to OxyContin. This exercise highlights the importance of treating the two misuse measures equally. When we use measures that more accurately capture recent OxyContin misuse than recent generic oxycodone misuse, we could mistakenly attribute effects of generic oxycodone to OxyContin.

## 1.6 Conclusion

Researchers have attributed the prescription opioid crisis and recent increase in heroin use to OxyContin. Previous studies have documented how Purdue Pharma's marketing downplayed the risks of OxyContin's abuse potential, which fomented the prescription opioid crisis; recent studies identified the OxyContin reformulation as the event that pushed users to switch to heroin, which precipitated recent increase in heroin use. This paper revisits the roles OxyContin and the Oxycontin reformulation played in the opioid crisis with fine-grained sales data that includes OxyContin's most immediate substitute, generic oxycodone. We have three main findings.

First, we find direct evidence of substitution to from OxyContin to generic oxycodone post-reformulation. Our difference-in-difference estimation indicates a 68% substitution from OxyContin to generic oxycodone due to the reform. Looking at the decline in OxyContin sales and rise in generic oxycodone sales from 2002-2006, we believe this substitution (for different reasons, namely Purdue's loss of its patent) also happened years before the reformulation. The size of this substitution, and indeed the size of the generic oxycodone market pre-reform, may come as a surprise to researchers. Paulozzi and Ryan, 2006 estimate that in 2002 OxyContin's market share was 68%. By the time of the reformulation in 2010, it had fallen by more than half. OxyContin played an essential part in igniting the prescription opioid crisis but, after losing its patent in 2004, other companies took up the torch and surpassed Purdue by selling generic oxycodone.

Our second main finding is that the OxyContin reformulation had no overall effect on opioid mortality. In our estimation, the OxyContin coefficients are not significant in the entire sample period, suggesting that higher OxyContin exposure is not predictive of either higher or lower opioid death. The lack of any trend indicates that the benefits of the OxyContin reformulation, if they exist, are offset by substitution to

oxycodone. In addition, we do find that high oxycodone exposure is predictive of rise in opioid mortality from 2011, confirming the increasingly important role of generic oxycodone in the recent prescription opioid crisis.

Third and most importantly, we show that the heroin overdose deaths after 2010 were predicted by generic oxycodone exposure, not OxyContin exposure. Our main event-study model shows positive and significant effects from oxycodone exposure on heroin deaths after 2012, but OxyContin exposure is not predictive of heroin deaths once we control for oxycodone. The difference-in-difference results are similar, showing that oxycodone exposure was predictive of heroin deaths before or after the reformulation, and OxyContin exposure after the reformulation is weakly positive but not statistically significant. We also do not observe an additional rise in heroin deaths immediately after reformulation in areas where OxyContin sales declined the most post-reformulation. In particular, without including generic oxycodone in the analysis, we recover the same results from the literature that OxyContin was responsible for the rise in heroin deaths. The evidence shows that omitting oxycodone, an important substitute to OxyContin, produces erroneous results. This paper demonstrates the pernicious effects of generic oxycodone, which had thus far escaped scrutiny until the *Washington Post* acquired data and reported on it.

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*Chapter 2*

# GEOGRAPHIC SPILLOVER EFFECTS OF PRESCRIPTION DRUG MONITORING PROGRAMS (PDMPS)

DANIEL GUTH AND SHIYU ZHANG

Prescription Drug Monitoring Programs (PDMPs) seek to potentially reduce opioid misuse by restricting the sale of opioids in a state. We examine discontinuities along state borders, where one side may have a PDMP and the other side may not. We find that electronic PDMP implementation, whereby doctors and pharmacists can observe a patient's opioid purchase history, reduces a state's opioid sales but increases opioid sales in neighboring counties on the other side of the state border. We also find systematic differences in opioid sales and mortality between border counties and interior counties. These differences decrease when neighboring states both have ePDMPs, which is consistent with the hypothesis that individuals cross state lines to purchase opioids. Our work highlights the importance of understanding the opioid market as connected across counties or states, as we show that states are affected by the opioid policies of their neighbors.

## 2.1 Introduction

Over the past two decades, the opioid epidemic has claimed more than 415,000 American lives (National Center for Health Statistics, Centers for Disease Control and Prevention, 2020). To stem the rising tide of opioid misuse, in the early 2000s, states began to regulate prescription opioid sales. Among the different policies that were implemented, we focus on Prescription Drug Monitoring Programs (PDMPs) that require prescribers and dispensers to submit data to a centralized system. In this paper, we study the effects of states' implementation of electronic-access PDMPs, a version of the law that allows doctors and pharmacists to query the patient's prescription history in real-time, on different regions in the same state and on the nearby states. Specifically, we focus on how sales in counties that border other states react differently to new PDMP regulations from sales in "inland" counties.

Our analysis shows that electronic-access PDMPs reduce prescription opioid sales and opioid mortality. The effect is economically and statistically significant despite

the fact that endogenous adoptions of such regulations bias our estimates of their impact downward. We find that border counties (counties that are immediately adjacent to another state) are systematically different from inland counties (counties not immediately adjacent to a county in a different state) and the enactment of ePDMP laws disproportionately affects border counties. These findings are consistent with our hypothesis that the border counties are destinations for consumers who are doctor or pharmacy shopping due to their proximity to another state. We also find a small but significant spillover effect in the form of increased opioid sales and overdose deaths when the neighboring state adopts stricter PDMP regulations.

Using the novel ARCOS data, we confirm the literature's general finding that PDMPs reduce opioid sales and mortality. We also contribute to resolving a debate in the literature about what features of PDMPs are more effective than others. We find that one specific implementation, electronic-access PDMPs (ePDMPs), is most effective at reducing opioid sales and mortality. Compared to a regular PDMP, this version not only requires doctors to submit information, but also allows doctors to see what other opioids a patient has received in real time. To the extent doctors and pharmacists consult the databases, ePDMPs mitigate the problem of individuals going to multiple doctors or pharmacies to secure opiates. We find that ePDMP laws reduce per person sales by 0.006 mg in active ingredient weight[1], which is equivalent to a 5.6% drop the 2006 national average, and per 100,000 mortality by 0.279, which is equivalent to a 12.3% decline from the 2006 national average.

We perform our analyses at the county level, which allows us to measure systemic differences in opioid markets of inland versus border counties due to the presence of state borders. Border counties appear similar to inland counties on observable demographics, but they have significantly higher opioid sales and lower opioid overdose deaths. These differences are consistent with the hypothesis that border counties are more frequently the destinations of doctor or pharmacy shopping, largely because their proximity to other states leads to lower travel costs for out-of-state residents. This difference between border counties and inland counties falls after the state adopts ePDMP, which further confirms our hypothesis that a higher percentage of sales in border counties were trafficked elsewhere for consumption. Our findings challenge the states-as-islands model often assumed in the opioid literature.

[1] The active ingredient weight is equivalent to the morphine milligram equivalent (MME) divided by 1500.

We also document negative externalities from these ePDMP laws, in the form of opioid sales and mortality increases in the border counties of neighboring states. We argue that these externalities come from the demand-side response of individuals using opioids, who now acquire these prescription drugs from out-of-state. The substitution to opioids from other states potentially reduces the effectiveness of ePDMPs as a policy intervention. The spatial substitution identified in this paper builds upon our previous work (Zhang and Guth, 2021) showing that partial supply-side interventions, like the OxyContin reformulation, can lead to drug substitution instead of preventing misuse. In the case of ePDMPs, the policy intervention was at the state and not the national level, so sales shifted across state lines instead of across products. This paper adds to the growing literature on the side effects of supply-side intervention curbing the opioid crisis (Alpert, Powell, and Pacula, 2018; Kim, 2021).

Our work speaks to the importance of not analyzing individual state policies in a vacuum. Individuals frequently cross these invisible borders in their day-to-day lives, and they may thus be subject to different regulatory regimes. The ability for individuals to evade one state's regulations for another extends to all markets regulated at the state level. One of the policy implications of our work is that there are costs to regulating opioids at the state level, and there would be benefits in enacting a national ePDMP. The American College of Physicians has called for a national prescription drug monitoring program, and for standardized PDMP laws across states until that point (Kirschner, Ginsburg, and Sulmasy, 2014).

The rest of the paper is as follows. Section 2 gives a background on PDMP laws as well as an overview of the literature understanding their effects. Section 3 describes the county-level sales and mortality data we use, the spread of PDMP laws during this time period, and our categorization of border counties. Section 4 describes how we model PDMP-border counties as well as our predictions based on economic theory and known trafficking patterns. Section 5 provides our results on sales and mortality, and finally, Section 6 concludes.

## 2.2 Background and Literature Review

Our paper connects three different strands of literature. First, we contribute to the literature on the opioid crisis and policies curbing opioid misuse. Second, we take methods from spatial economics and apply them to cross-border opioid sales and misuse. Third but not least, we build upon modern analyses of the effects of PDMP

laws.

**The Opioid Crisis and Interventions**

Over the past two decades, millions of Americans have misused prescription opioids. In 2019 alone, 1.6 million people had an opioid use disorder and 70,630 people died from an opioid overdose[2]. Opioid use disorder has devastating consequences for the individual, the family, and the community. The CDC estimates the total "economic burden" of prescription opioid misuse to be 78 billion dollars a year.

Many victims of the epidemic got their first access to an opioid from a doctor's prescription. Previous research has documented large variations in opioid prescribing and sales, both within and across states. McDonald, K. Carlson, and Izrael (2012) shows that the ratio of per-capita oxycodone sales in counties in the 75th percentile to counties in the 25th percentile is approximately seven to one. Their best model can only predict one-third of the variation in sales by county. Finkelstein, Gentzkow, and Williams (2018) uses Medicare data to track individuals who move between counties, and the paper finds that location has a noticeable effect on an individual's access to opioids. The paper estimates that 30% of the difference in opioid prescribing between counties can be explained by these place-specific factors. Our work is connected to the opioid prescription literature, in that we both study location-specific effects, but our data is on opioid shipments to pharmacies which occurs further down the prescription pipeline. We add to this literature by showing that being on the state border is one of these factors that affects local opioid sales and misuse.

Over the last two decades, states have made repeated attempts to regulate the sales of prescription opioids in the hope of preventing further opioid misuse. Litigation against Purdue Pharma, the manufacturer of the drug that ignited the opioid crisis, led the company to reformulate OxyContin in 2010. The reformulation led to reduced sales of OxyContin, but spurred on an increase in alternative oxycodone and heroin misuse (Zhang and Guth, 2021; Alpert, Powell, and Pacula, 2018; Evans, Lieber, and Power, 2019). Many states enacted new PDMP laws or tightened existing ones. The evidence of the effectiveness of such laws is mixed (for more detail see Section 2.2), and some argue that the new restrictions led to increases in heroin mortality (Kim, 2021; D. Dave, Deza, and Horn, 2021). Some states, Florida included, passed legislation that requires pill mills—rogue pain management clinics that were

[2]The US Department of Health and Human Services on the Opioid Epidemic (link).

inappropriately prescribing and dispensing opioids—to register with the state. The pill mill laws have led to a moderate decrease in opioid prescription and use (Rutkow et al., 2015; Kennedy-Hendricks et al., 2016). One common theme in this strand of the literature is a substitution toward alternative drugs when the original supply became restricted. We add to this literature by evaluating the effectiveness of PDMP laws while taking into consideration potential spatial spillovers.

**Spatial Spillover and Opioids**

Our work ties tightly into the literature studying the distribution of economic activities across space. Many works have noted how geographic characteristics have a direct impact on manufacturing, sales, and trade. Holmes (1998) finds sharp increases in manufacturing activity across the border in so-called "pro business" states. Similarly, Nachum (2000) finds that location and agglomeration effects can explain which states transnational corporations choose to put their headquarters in. Fox (1986) examines border counties and finds that changes in state taxes can shift purchases across state lines. Garrett and Marsh (2002) examines lottery sales in Kansas and estimates that the state loses $10.5 million dollars in net lottery revenue to cross-border shopping in 1998. We use border counties, a concept from this literature, to show how state policy differentially affects different locations. Our setting provides the perfect environment to test for spillovers because we have detailed sales data on exactly where opioids are sold, which is not common in other settings.

We also contribute to a small but significant literature on cross-border prescription shopping. Crossing state and national borders to taken advantage of favorable regulatory environments to obtain drugs is not a new concept in the literature. Casner and Guerra (1992) documents patients crossing the US-Mexico border to purchase prescription medication cheaply and without a prescription. McDonald and K. E. Carlson (2014) estimates that approximately 30% of "doctor shoppers" had opioid prescriptions from multiple states. Cepeda et al. (2013) finds that 4% of non-shoppers visited more than one state to purchase opioids, and for individuals who visited multiple pharmacies to purchase opioids, the median distance between pharmacies was about 12.6 miles. We add to this literature by leveraging decentralized policy change to systematically identify the impact of cross-border shopping on opioid sales.

**Prescription Drug Monitoring Programs**

Before any Prescription Drug Monitoring Programs, or PDMPs, individuals can freely doctor or pharmacy shop[3], and there is no way for doctors or pharmacists to know how many other prescriptions an individual has. PDMPs are state-level databases that track controlled substance prescriptions in a state. The modern precursor to the PDMP was California's "Triplicate Prescription Program" enacted in 1939[4]. The law required the dispensing pharmacist to fill out standardized forms for controlled substances and mail a copy to a centralized state repository. The California program set the blueprint for PDMPs and many states followed suit in subsequent decades. The legality of PDMPs was tested in *Whalen v. Roe*, where the Supreme Court unanimously ruled that storing this personal medical information did not violate a person's right to privacy.

These original PDMPs collected information from doctors and pharmacists via mail or fax, and doctors and pharmacists could not immediately query a patient's opioid history. Oklahoma implemented the first fully electronic PDMP in 1990 that directly and routinely sent records to a state database (Holmgren, Botelho, and Brandt, 2020). Currently, the electronic-access PDMPs allow registered doctors and pharmacists to query the data set in real-time and see all opioids an individual received in that state. The 21st century saw a wave of expansion to electronic PDMPs and by 2019, all but one US state have implemented e-access PDMPs (Mallatt, 2019). The next wave of PDMP regulation is the must-access or mandatory PDMPs. These laws require doctors and pharmacists to check an individual's opioid history before dispensing opioids. Absent the mandate, only filling the information is mandatory; checking a patient's history is voluntary. The must-access laws are often based on, and enacted after, electronic PDMPs. By 2017, 19 states have enacted some version of must-access PDMPs.

Most states do not share any information collected from PDMPs with other states[5]. The lack of information sharing made it feasible for individuals to partially circumvent the regulation by shopping across state borders. If state A adopted an ePDMP,

[3]Doctor shopping refers to the behavior of individuals going to multiple doctors to get opioid prescriptions to evade scrutiny, and pharmacy shopping refers to going to multiple pharmacies to get the prescriptions filled.

[4]New York had the first PDMP law in 1918, but rescinded it three years later.

[5]Lin et al., 2019 shows that in 2014, 23 states had some sort of data sharing agreement, but many of these agreements were one-way, and only Michigan and Indiana shared this information with all of their neighboring states.

an individual would face greater difficulty getting a second opioid prescription filled in-state. This difficulty could occur either from doctors, who upon observing that a patient already has an opioid prescription do not write another, or from pharmacists who refuse to fill it for the same reason. However, an individual could attempt to get and fill a second prescription in a neighboring state. We aim to evaluate the propensity for individuals to get opioid prescriptions outside of their state, specifically to avoid PDMP regulations.

There is a wide array of studies on the effects of PDMPs. One typical corroborated result in the literature is that PDMPs decrease prescription opioid sales (Simeone and Holland, 2006; Reisman et al., 2009; Kilby, 2016) and reduce abuse and mortality (Simeone and Holland, 2006; Patrick et al., 2016). Some papers note that specific formulations of PDMP are more effective than others. Effective features include monitoring more drugs and updating weekly (Patrick et al., 2016), and identifying and investigating cases proactively (Simeone and Holland, 2006). Bao et al. (2016) look at 22 states from 2001 to 2010 that implemented electronic access to PDMPs and showed it reduced oxycodone prescriptions from ambulatory visits to physician offices by 30%. A set of papers claim that only must-access PDMPs (MA-PDMPs) are effective in reducing opioid misuse (Buchmueller and Carey, 2018; Grecu, D. M. Dave, and Saffer, 2019; D. Dave, Deza, and Horn, 2021; Meinhofer, 2018; Kim, 2021) which conflicts with existing results on effectiveness of non-mandatory PDMPs. We contribute to this debate by showing that ePDMP laws are effective at reducing sales and overdose deaths during our sample period.

The disagreement in the literature on what features of PDMPs are more effective than others is partly the result of each paper employing its own categorization of laws and testing the effectiveness on different outcome variables. Assembling an accurate policy data set across all 50 states is inherently challenging (Schuler et al., 2021). Horwitz et al. (2018) point out that the inconclusive and contradictory results may be due to the large variations in dates used in different studies. Existing sources of enactment dates rarely acknowledge the researchers' decisions in creating such a data set, and the public sources have a large disagreement. In this paper, we use the "modern system operational date" variable from Horwitz et al. (2018) in our main analysis. We will elaborate on the choice of "modern system operational date" over other implementation dates in Section 2.3.

States adopted PDMP policies at different times, but the literature generally does not

address potential endogeneity concerns. For our regression specification, one particularly worries that states might be more likely to adopt PDMPs because they have the infrastructure to make the laws effective. If so, a naive regression's coefficients would be biased in favor of the hypothesis that the laws matter but such upward biases are unlikely. We argue that adoptions of PDMP laws are endogenous to local conditions but in ways that bias coefficients downwards toward zero rather than upwards. Specifically, places that are experiencing more opioid misuse or higher growths in sales or overdoses are the most likely to adopt measures like PDMPs. A simple difference-in-difference estimation of the effect of the law underestimates its impact and biases against the key hypotheses we want to test. Our estimation of the impact of PDMP laws on sales and mortality both suffer from this bias, but we are capturing statistically significant coefficients nonetheless. Moreover, since the enactment of PDMPs in a state is independent of the differences between border and inland counties in that state, and independent of conditions in nearby states, our estimation of the border effect and spillover will not be affected by the endogeneity problem.

## 2.3 Data

In this section, we introduce the data source of our sales and mortality data, describe our choice of PDMP implementation dates, define how we characterize border counties, and present summary statistics.

### ARCOS sales data and NVSS Mortality data

As part of the Controlled Substances Act, distributors and manufacturers of controlled substances are required to report all transactions to the DEA. This Automation of Reports and Consolidated Orders System (ARCOS) database contains the record of every pain pill sold in the United States. The complete database from 2006 to 2014 was recently released by a federal judge as a result of an ongoing trial in Ohio against opioid manufacturers.[6] The part of ARCOS that we use in this paper is shipments of oxycodone from manufacturers to pharmacies. In theory, the manufacturer to pharmacy shipments are not equivalent to sales to the consumers. However, since pharmacies do not keep large stocks of opioids, the aggregated annual data of sales from manufacturers to pharmacies is practically equivalent to the annual sales of pharmacies to consumer sales. The benefit of ARCOS data is that it allows disaggregation to arbitrarily fine geographical levels, which is essential for

[6]Link to the ARCOS Data published by the *Washington Post*.

the identification of the border effects, and it contains all opioid sales which allows us to identify spatial substitution. The ARCOS sales data is the primary outcome variable in our regressions.

We care about how PDMP laws affect opioid sales, but ultimately we're interested in preventing their effects on overdoses and deaths. The second outcome of interest in our main regression is opioid mortality. We use the restricted-use multiple-cause mortality data from the National Vital Statistics System (NVSS) to track opioid overdoses. The dataset covers all deaths in the United States from 2006 to 2014. We follow the literature's two-step procedure to identify opioid-related deaths. First, we code deaths with ICD-10 external cause of injury codes: X40–X44 (accidental poisoning), X60–64 (intentional self-poisoning), X85 (assault by drugs), and Y10–Y14 (poisoning) as overdose deaths. Second, we use the drug identification codes, which provide information about the substances found in the body at death, to restrict non-synthetic opioid fatalities to those with ICD-10 code T40.2.

**PDMP Enactment Dates**

As discussed in the background section, there are multiple sets of PDMP enactment dates, and the literature disagrees on which is the most effective in reducing opioid misuse. In this paper, we consider three sets of dates: (a) the legislated start date (any PDMP), which is the year that dispensers or prescribers would be required to send prescriptions to a central database, (b) the electronic access date (ePDMP), which is the year that the PDMP data becomes accessible to the dispensers or prescribers through a centralized electronic system, and (c) the must-access date (MA-PDMP), which is the year when certain dispensers or prescribers are required to check an individual's opioid history before dispensing. In Figure B.1 in Appendix, we graph the three enactment dates for each state. Most states started with the most basic version of PDMP and gradually adopted e-access in the 2000s. Only a handful of states adopted must-access PDMP during our time period.

We use ePDMP dates in our main regression analysis. The reasons are twofold. First, ePDMPs have large impact on prescriptions and sales both conceptually and empirically. Conceptually, an ePDMP streamlines the process by which the prescribers and dispensers check a patient's prescription history. Before an ePDMP, prescribers and dispensers are required to report opioid prescriptions but could not easily tell what other prescriptions an individual had. ePDMPs allows them to check a patient's opioid history online in real-time, so they could more easily refuse opioids

to questionable patients. Although an ePDMP is less restrictive than a MA-PDMP, it is reasonable to assume a large number of doctors who are conscious of the severity of the opioid crisis would have taken advantage of the electronic system when it became available. Empirically, Horwitz et al., 2018 finds this set of dates is most correlated with reductions in opioid sales after comparing it with nine other sets of dates[7].

Second, our sample period has higher coverage of enactment of ePDMP as compared to the other two dates. There is reasonable consensus in the literature that each wave of new PDMP legislation tightens the legal supply of opioids and reduces misuse (although the literature disagrees on which version is the most effective). Given that each round of legislation may have some impact, we want to work with the one that gives us the most identification power. The switch from no PDMP to any PDMP happened in the 1990s and early 2000s, and by 2006, the start of our sample period, 31 states have already adopted it. The adoption of ePDMPs took place mainly during our sample period: 37 states adopted ePDMP between 2006 and 2014. Only 10 states enacted MA-PDMP during our sample period. Working with ePDMPs allows us to use data from more states to estimate the impact of the law. Our $\beta$ estimations would be less reliant on trends from a few states.

Our ePDMP dates are obtained from Horwitz et al. (2018)[8]. We use Horwitz as our main source because this paper is the most systematic methodological paper on PDMP implementation timing that we've reviewed. In robustness, we use ePDMP dates published by the Prescription Drug Abuse Policy System (PDAPS), an organization funded by the National Institute on Drug Abuse to track state laws related to prescription opioid abuse. To check if other PDMP laws have similar spillover effects, we use any PDMP dates from Horwitz et al. (2018) and MA-PDMP dates from Sacks et al. (2021). We list all sets of dates in the Chapter 2 Appendix.

**Defining Border Counties and Assigning ePDMP Status**

We define a border county as a county that neighbors at least one county in an adjacent state and an inland county as a county that borders only counties of the

[7]To be fair, the paper did not compare ePDMP with MA-PDMP.

[8]The authors coined their e-access dates the "modern system operational date." Although the naming is different, the two definitions are conceptually identical.

same state. After excluding Alaska, Hawaii[9], and Florida[10] from our data, we have 2906 counties, 37.3% of which are border counties (see Figure B.2 in Appendix for a visual representation of border and inland counties). For each inland county, we document whether an ePDMP law has been implemented. For each border county, we document whether a law has been implemented in that state and the bordering state(s). If a county is bordering multiple states and these states have different ePDMP status, the nearby law of the county will be the ePDMP status under which the majority of the nearby population live[11]. We only need to do this calculation on 653 county-year observations, which is 6.6% of all border county-year observations. See Figure B.3 in Appendix for an example of the calculation.

The transition from states not having an ePDMP to having an ePDMP is key to our identification. During our sample period, over 60% of all counties transitioned from no ePDMP to ePDMP (see adoption rate in Figure B.4 in Appendix). Identification of border coefficients relies on law change in a county and law changes in nearby border counties. The majority of the transitions in border counties also took place during our sample period: over 80% of border counties has no ePDMP regulation in 2006 and that number decreases to less than 20% by the end of 2014 (see detailed transitions in Figure B.5 in Appendix).

**Summary Statistics**

Since we are comparing border counties to inland counties of the same state, it is important that we acknowledge any potential differences between the two sets of counties, especially those associated with opioid use. In Table 2.1, we document the population-weighted average of opioid sales, mortality, and important demographics, and ePDMP coverage of the two sets of counties. Border counties have a significantly higher level of opioids sales throughout the sample period. They have lower levels of opioid mortality in 2006, but the difference loses significance since 2010. We will discuss these differences in outcome variables in our hypotheses and result section. The two sets of counties are quite similar on all demographic

[9] Alaska and Hawaii neighbor no US states.

[10] Florida experienced a dramatic rise in opioid supply in the 2000s and then a significant drop due to crackdown on pill mills in 2010–2011. It is common practice in the literature to exclude Florida from the analysis.

[11] The underlying idea is that the ePDMP status of more populous nearby counties would have a bigger impact on the local county than the ePDMP status of less populous nearby counties. Specifically, we sum up the population adjacent to a border county by ePDMP status. If more nearby population resides under the states with PDMP law than no law, the county's nearby law variable will be 1; if more nearby population resides under the states with no law, it will be 0.

dimensions. Since some of these demographic factors are associated with higher levels of opioid misuse, it is important we control for demographic differences in our regressions.

Table 2.1: Summary Statistics.

| Variables | All counties | Border counties | Inland counties | Test of equality (p-value) |
|---|---|---|---|---|
| *Opioid-related statistics* | | | | |
| Sales per person (2006) | 0.101 | 0.113 | 0.095 | 3.28e-11 |
| Sales per person (2010) | 0.163 | 0.185 | 0.150 | 0.004 |
| Sales per person (2014) | 0.158 | 0.181 | 0.145 | 0 |
| Opioid overdose per 10,000 (2006) | 2.22 | 2.01 | 2.33 | 0.003 |
| Opioid overdose per 10,000 (2010) | 3.35 | 3.29 | 3.38 | 0.574 |
| Opioid overdose per 10,000 (2014) | 3.84 | 4.02 | 3.75 | 0.164 |
| *Demographics (2009)* | | | | |
| Average Population | 98,853 | 92,914 | 102,392 | 0.397 |
| Average Age | 36.11 | 36.8 | 35.7 | 0.149 |
| Male (%) | 49.2 | 49.0 | 49.3 | 1.53e-07 |
| Separated (%) | 18.2 | 18.6 | 18.1 | 0.001 |
| High School and above (%) | 83.4 | 83.9 | 83.1 | 0.002 |
| Bachelor and above (%) | 27.4 | 27.2 | 27.6 | 0.004 |
| Mean income | 70,130 | 71,063 | 69,625 | 0.05 |
| Low income (%) | 33.2 | 33.3 | 33.2 | 0.703 |
| White (%) | 78.6 | 79.0 | 78.4 | 0.279 |
| Black (%) | 12.8 | 13.6 | 12.4 | 0.015 |
| Asian (%) | 4.94 | 3.87 | 5.51 | 0 |
| Native American (%) | 0.178 | 0.141 | 0.197 | 8.41e-05 |
| *PDMP-related statistics* | | | | |
| Number of counties | 2906 | 1085 | 1821 | |
| Have ePDMP by 2006 (%) | 18.6 | 17.8 | 20.1 | |
| Have ePDMP by 2010 (%) | 50.5 | 52.0 | 49.5 | |
| Have ePDMP by 2014 (%) | 87.2 | 85.1 | 88.3 | |

*Notes:* Means are weighted by county population. For opioid-related statistics, border counties have significantly higher levels of opioid sales throughout the sample period. Mortality is higher in inland counties, but the difference is not significant in all three years we tested. Many of the differences in demographics between border and inland counties are statistically significant but not economically. The adoption rates of ePDMP laws are similar between the two sets of counties.

## 2.4 Hypotheses and Empirical Framework

### Hypotheses

In this section, we lay out our hypotheses and discuss the underlying assumptions and their implications on the market structure of prescription opioids. We start with a simplified model with no spatial spillover.

**The state-as-island model.** Consider states as isolated islands in an ocean. Due to the separation, opioids sold in each state can only be consumed in that state. Since county location bears no significance in this model, sales patterns and mortality should be similar in border and inland counties of the same state after controlling for demographic differences. For example, San Bernadino County, on the state border between California and Arizona, should behave similarly to Fresno County, landlocked within California. Although the adoption of a PDMP is endogenous to local conditions, a priori we would not expect the law to have differential effects on border and inland counties. Since all opioids sold locally are consumed locally, changes in sales due to PDMP laws should translate directly to changes in use patterns, and by extension, to changes in local opioid mortality, ignoring any substitution to illegal opioids[12]. The adoption of PDMP in one state should have no impact on opioid sales or mortality in the neighboring state. The testable hypotheses of the state-as-island model are:

> *Hypothesis 1a:* Under the state-as-island model, sales and mortality patterns are similar in border and inland counties.
>
> *Hypothesis 1b:* Under the state-as-island model, changes in sales translates into changes in mortality.
>
> *Hypothesis 1c:* Under the state-as-island model, adoption of PDMP in one state has no impact on sales or mortality in the neighboring states.

However unrealistic the above model is, it is assumed in many important studies on the opioid crisis. States are treated as isolated markets where all pills sold are consumed locally with the exception of Florida, which most papers exclude. The state-as-island model is applicable in situations when the spillover effect is small compared to the main effect, or if the spillover's impact is tangential to the main question. The literature has documented many occasions when the state-as-

[12] We focus on opioid mortality, but as described in the literature review, some papers do find substitution to heroin following implementation of MA-PDMP laws.

island model fails. Individuals cross the state border to take advantage of favorable lottery situations (Garrett and Marsh, 2002); patients cross the US-Mexico border to purchase prescription medication cheaply and without a prescription (Casner and Guerra, 1992). The decentralized enactment of PDMP creates differences in regulatory environments and incentivizes individuals to seek out the less regulated market. Next, we consider a model with spatial spillover.

**The spatial spillover framework.** Consider two states not separated by an imaginary ocean. Both opioids and people can cross the state line. As a result, opioids purchased in one state may or may not be consumed in that state. When individuals are incentivized to purchase opioids from a neighboring state, their cost of doing so is highly dependent on the distance traveled. Under these assumptions, vicinity to the state border has consequences on opioid sales and diversion. For someone living on the Arizona side of the Arizona-California state border, the cost of travelling to San Bernadino County for additional pills is much lower than that of travelling to some inland county within California.

The question remains as to when individuals are incentivized to cross the state border. Before any PDMP law, patients could obtain multiple prescriptions and get them filled in the same state with minimal constraint. When states adopt some version of the PDMP, doctor and pharmacy shopping within the same state become more difficult. However, because most states do not share their PDMP data with the neighboring states, the cost of obtaining additional pills from the neighboring states remains the same despite enactment of PDMP locally. As the cost of within-state pill shopping increases due to progressively stricter PDMP regulations (from PDMP to ePDMP to MA-PDMP), more and more individuals would be incentivized to cross a nearby border. By the start of our time period, 31 states had enacted some version of the PDMP, which means that some individuals would already be going to other states for pills. Hence, we expect a higher share of the border counties' sales to be diverted elsewhere for consumption during our sample period. Because the diverted pills are not consumed locally, we expect the sales to mortality ratio to be higher in a border county.

> *Hypothesis 2a:* Under the spillover framework, border counties will have higher sales but lower moralities as compared to inland counties of the same state.

Variation in diversion rates between inland and border counties implies that the two sets of counties will respond differently to new PDMP regulations. When states enact stricter PDMPs, the local pill shoppers and the out-of-state pill shoppers are similarly affected by the new rule. Since a higher share of the border counties' sales is from pill shoppers, the law change will have a bigger impact on the border counties. The endogeneity of adoption may bias the overall estimation toward zero, but should not affect how the border counties react to the law change relative to the inland counties. In addition, as the cost of local pill shopping increases due to stricter laws, local pill shoppers are more incentivized to cross the state border, and hence sales in border counties of the neighboring states would increase.

> *Hypothesis 2b:* Under the spillover framework, when the local state adopts a stricter PDMP, border counties will experience a larger decrease in sales relative to inland counties of the same state.

> *Hypothesis 2c:* Under the spillover framework, when the nearby state adopts a stricter PDMP, border counties will experience a larger increase in sales relative to inland counties of the same state.

In this stylized model, the mapping from sales to mortality is less direct when spatial spillover was not possible. With the state-as-island model, the enactment of a PDMP law puts a hard constraint on the opioid misuser's ability to acquire prescription opioids. Assuming no other substitution, changes in opioid sales in one location translate directly into changes in opioid mortality in that location. With spatial spillovers, changes in opioid sales in one place may lead to changes in mortality elsewhere. Since a larger share of the border counties' sales was consumed elsewhere, the adoption of stricter PDMP will result in a smaller drop in opioid mortality in the border counties. The enactment of PDMP in a nearby state increases sales in the border counties but should have no additional impact on mortality, assuming that people traveling to acquire pills go back to their home counties to consume them. In reality, how mortality responds to a PDMP law depends on many factors, including the state of the black market, the availability of alternative drugs, and the ease of getting drugs from the nearby states. Since we cannot control for all of these relevant factors, we expect the mortality results to be less sharp than the sales results.

> *Hypothesis 2d:* Under the spillover framework, when the local state

adopts stricter PDMPs, border counties will experience a smaller decrease in mortality relative to inland counties of the same state.

*Hypothesis 2e:* Under the spillover framework, when the nearby state adopts stricter PDMPs, border counties will experience no additional change in mortality relative to inland counties of the same state.

See Figure B.6 in Appendix for a visual representation of the hypotheses of the spillover framework.

**Empirical Framework**

We want to test (1) how counties react to the enactment of ePDMP laws, (2) if border counties react differently as compared to inland counties, and (3) how the adoption of an ePMDP in one state affects border counties in the adjacent state. We use the following empirical framework to test our hypothesis:

$$\begin{aligned} Y_{ct} =& \alpha_s + \delta_t + \beta_1 \text{ Law}_{ct} + \beta_2 \text{ Border}_c + \beta_3 \text{ Law}_{ct} \times \text{Border}_c + \\ & \beta_4 \text{ Nearby Law}_{ct} \times \text{Border}_c + X_{ct}\gamma + \epsilon_{mt} \end{aligned}$$

where $Y_{ct}$ are the outcome variables of interest: sales and mortality in county $c$ in year $t$. Ideally, because each county has different initial conditions, we want to control for these conditions to get at the impact of the law change. However, because the location of a county and its border status does not change over time, any time-invariant differences between the border and inland counties would be absorbed by the county-fixed effects if added. Hence, we use a full set of state-fixed effects $\alpha_s$ and county characteristics $X_{ct}$ as controls. We also add year-fixed effects to control for national changes in drug use over time.

Our coefficients of interest are the full set of $\beta$'s: $\beta_1$ estimates the impact of ePDMP laws on sales and mortality; $\beta_2$ estimates the baseline difference in sales and mortality between border and inland counties of the same state; $\beta_3$ estimates how the law affects the border counties differently as compared to the inland counties; and $\beta_4$ estimates how the enactment of an PDMP in one state impacts sales and mortality in the bordering counties of the neighborhood state, as compared to inland counties in the neighborhood state.

One notable feature of our empirical strategy is that the identification of the border effects ($\beta_3$ and $\beta_4$) does not require any assumption about the exogeneity of law change. As we've discussed in the literature review, enactments of PDMP laws are

endogenous. When each state decides to implement ePDMP is a function of many factors, including its regulatory environment, the severity of its opioid crisis, the current political climate, and many others. These factors are highly correlated with the pre-enactment level of sales and mortality and the post-enactment response. If states are more likely to pursue stringent opioid regulations when conditions are bad, $\beta_1$ would underestimate the true impact of the law change. In terms of the estimation of the difference ($\beta_3$) and the spillover effect ($\beta_4$), law changes can be considered as random events.

## 2.5 Results

### PDMP Law and spatial spillover in sales

The full set of $\beta$ from our main regression is presented in column (5) of Table 2.2. We start with a simple two-way fixed effects model in column (1). We replace county fixed effects with the set of state fixed effects in (2) to (5) to estimate the border coefficients. In (2), we replicate the same regression as in (1) to show that changing from county to state fixed effects has no discernible impact on the ePMDP law coefficient. Starting in column (3), we add border status and interact it with ePDMP law to separately estimate the impact of ePDMP law on border counties. To ensure that differences in population characteristics between border and inland county are not driving the identification, in column (4) to (5), we control for county characteristics (average age, % male, % separated, education level, mean income, % low income, and ethnicity). These are variables that the literature has characterized as being influential in driving opioid use and overdose (Wright et al., 2014). In column (5), we add an indicator for whether the nearby state adopted ePDMP for each border county. We repeat the same analysis using the alternative ePDMP enactment dates from PDAPS. The results are documented in in Table B.1 in the Appendix.

Table 2.2: Impact of ePDMP laws on opioid sales using Horwitz (2018) modern system operational dates.

| | Dependent variable: | | | | |
|---|---|---|---|---|---|
| | Sales per person | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_1$ - PDMP law | -0.006*** | -0.005*** | -0.002 | -0.003 | -0.003 |
| | (0.001) | (0.002) | (0.002) | (0.002) | (0.002) |
| $\beta_2$ - Border county | | | 0.006*** | 0.004*** | 0.004*** |
| | | | (0.002) | (0.001) | (0.002) |
| $\beta_3$ - Law x border | | | -0.009*** | -0.008*** | -0.008*** |
| | | | (0.002) | (0.002) | (0.002) |
| $\beta_4$ - Nearby law x border | | | | | 0.0004 |
| | | | | | (0.002) |
| County FE | Yes | | | | |
| State FE | | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Controls | | | | Yes | Yes |
| Observations | 26,154 | 26,154 | 26,154 | 26,154 | 26,154 |
| $R^2$ | 0.000 | 0.458 | 0.458 | 0.519 | 0.519 |

$\beta_1$ estimates the average effect of the enactment of ePMDP laws on opioid sales. Before adjusting for differential response due to the location of the county, we find that ePDMP reduces opioid sales. The coefficient is consistently negative in all specifications, but only significant before the inclusion of border coefficients. The border coefficient $\beta_2$ is consistently positive from (3) to (5), indicating that border counties start with higher sales as compared to inland counties of the same state. The estimation of $\beta_2$ supports *hypothesis 2a* (spillover framework) over *hypothesis 1a* (state-as-island framework). $\beta_3$, the law and border interaction term, is consistently negative. Although border counties start with higher per person sales, they experience a much larger drop in sales post-ePDMP than inland counties in the same state. The results are consistent with *hypothesis 2b* (spillover framework) that a higher percentage of sales in border counties are diverted elsewhere for non-medical use. Comparing the size of $\beta_1$ across specifications, we see that the estimated impact of ePMDP law on sales is largest in columns (1) and (2) and decreases and loses significance once we interact law with border status. If we don't separately account for abnormal behaviors in the border counties, the coefficients in (1) and (2) over-estimate the effect of the law change on opioid sales in a "normal" county. We observe the same pattern using our alternative e-access dates in Table B.1.

In regression (5) of Table 2.2, $\beta_4$ is not well identified. Using our alternative e-access

date, $\beta_4$ is significant and positive. We need to be careful in interpreting $\beta_4$ since the coefficient is measured with respect to sales in inland counties of the same state. Suppose A and B are two neighboring states and A experiences a law change. We have tentative evidence that counties in B that border A experience a faster growth (or slower decline) in sales than the inland counties in B. The findings support *hypothesis 2c* (spillover framework) over *hypothesis 1c* (state-as-island framework). Implementation of an ePDMP in one state increases the sales of opioids in border counties of nearby states.

Putting the coefficients together, border counties start with higher sales, experience a larger decrease if the local state enacts the ePDMP, and an additional increase if the nearby state enacts the ePDMP (only if we use alternative ePDMP dates). When states on both sides of the border adopt ePDMPs, most of the border effects cancel out. As the difference in regulation disappears between states, border counties lose their higher-than-average sales and their significance in cross-border opioid trafficking. In addition, the decrease in sales due to ePDMP laws is driven mostly by decreases in the border counties. The inland counties experience no significant drops in sales once we control for the border-law interactions. In the robustness section, we discuss what impact adoption timing has on how border states react to the enactment of electronic PDMP locally and nearby.

Translating our coefficients to real terms using Table 2.2, if we don't differentiate the border counties from inland counties, (1) shows that the law reduces per person sales by 0.006 MME, which is equivalent to a 5.6% drop from the national average in 2006. Since only a portion of sales are diverted for non-medical use, a 5.6% overall decrease is large if we translate it into drops in diversions. When we account for border status, our estimation shows that the law reduces inland county sales by 0.003 mg in active ingredient on average (2.8%). In addition, the law reduces the border county's sales by 0.011 mg in active ingredient weight (10.2%), which is more than three times as much as the drop in inland counties.

**PDMP Law and spatial spillover in mortality**

We've shown that the adoption of PDMP laws decreases local sales but has spillover effects on nearby states. Ultimately, however, what we care about is the consequences these laws have on actual opioid misuse and overdose. In this section, we use the same econometric specifications to test what impact an ePDMP law enacted in a state has on mortality in local and nearby counties. We expect the mortality results to be

less sharp than sales results since there are many intervening factors between access to prescription opioids and opioid overdoses. Spatial spillovers, as identified in the previous section, are one. Substitution toward other alternative drugs is another. The literature has many examples of how restricting access to one drug resulted in substitution toward another potentially more lethal substance (Alpert, Powell, and Pacula, 2018; Zhang and Guth, 2021; Kim, 2021).

Table 2.3: Impact of ePDMP laws on opioid mortality using Horwitz (2018) modern system operational date.

| | Dependent variable: | | | | |
|---|---|---|---|---|---|
| | Mortality per 100,000 residents | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_1$ - PDMP law | -0.217*** | -0.192*** | -0.302*** | -0.318*** | -0.279*** |
| | (0.051) | (0.069) | (0.075) | (0.073) | (0.074) |
| $\beta_2$ - Border county | | | -0.580*** | -0.666*** | -0.763*** |
| | | | (0.063) | (0.062) | (0.067) |
| $\beta_3$ - Law x border | | | 0.320*** | 0.366*** | 0.254*** |
| | | | (0.085) | (0.083) | (0.088) |
| $\beta_4$ - Nearby law x border | | | | | 0.297*** |
| | | | | | (0.076) |
| County FE | Yes | | | | |
| State FE | | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Controls | | | | Yes | Yes |
| Observations | 26,154 | 26,154 | 26,154 | 26,154 | 26,154 |
| $R^2$ | 0.000 | 0.283 | 0.285 | 0.318 | 0.318 |

The coefficients on *PDMP law* are straightforward to interpret. Across the specifications, PDMP laws reduce opioid overdose. The reduction is economically significant. Using estimates from column (5), a -0.279 drop per 100,000 people translates into a 12.3% drop from the national opioid fatality rate in 2006. A negative and significant $\beta_2$ indicates that border counties have a lower level of baseline overdoes rate, which is consistent with our hypothesis that border counties don't abuse as many opioids but export a high percentage of their sales for misuse elsewhere (*hypothesis 2a*). Given that the extra sales originating from border counties are not consumed locally, the adoption of PDMP laws should have no extra impact, if not less, on mortality in these counties. In columns (3) to (5), our estimation of $\beta_3$ is positive and significant. The size of $\beta_3$ is almost as large as $\beta_1$ in all three specifications, suggesting that the adoption of ePDMP has nearly no impact on a border county, which supports *hypothesis 2d*.

In regression (5), we find $\beta_4$ to be positive and significant, which suggests that the mortality rate in border counties neighboring a state with a new ePDMP law increases faster (or decreases slower) than that in the inland counties of the same state. We get similar findings using the alternative ePDMP dates (Table B.2). A positive $\beta_4$ does not support *hypothesis 2e* that nearby enactment of ePDMP has no addition impact on the border counties. While the sales results suggest that people from recently restrictive states cross the state line to acquire opioids from the neighbor county, the mortality results suggest that these people not only shop across state lines, but also stay in the neighbor county to consume these opioids. Validating this mechanism is beyond the scope of the data we have, and we leave it to future researchers.

The differences in the mortality and the sales results are direct evidence that prescription opioids are trafficked across state lines. If opioids sold in each county are consumed locally, the mortality result should mirror that of the sales result. However, we find that border counties start with higher levels of sales but lower levels of mortality. Enactment of ePDMP leads to additional drops in sales in border counties, but fewer drops in mortality. The overall evidence supports the spillover framework over the state-as-island framework.

**Effectiveness of alternative PDMP laws**

To check if other PDMP laws have similar spillover effects, we run our main regression using two additional dates: any PMDP dates from Horwitz et al. (2018) and MA-PDMP dates from Sacks et al. (2021). The results on sales are documented in Table B.3 in Appendix. The enactments of PDMP and must-access PDMP are not associated with reductions in opioid sales during our sample period. These findings are not conclusive evidence that PMDPs or MA-PDMPs are ineffective in reducing opioid sales. As we've stated in Section 3.2 and shown in Figure B.1, our sample period covers very few enactments of PDMPs and MA-PDMPs. Most states had already enacted some version of the PDMP by the start of our sample period, hence we only observe PDMP law change in few states that had been slow in action. Similarly for MA-PDMP, we only observe law change in the few early-mover states. The limited data combined with the endogeneity of adoption means that we do not have enough power to identify the effects of PMDP and MA-PDMP using 2006 to 2014 data.

We identify no border or spillover effect using the two alternative dates. The results

suggest that identification of the border and spillover effects is sensitive to using the "correct" PDMP law. On the border coefficient, we know from previous regressions that the enactment of ePDMPs on both sides of the border makes the border counties lose their significance in cross-border shopping. Not finding a border effect using PDMP or MA-PDMP dates further validates our main hypotheses. On the spillover effect, if the law itself did not lead to a significant reduction in opioid sales in the first place, there is no reason to expect individuals to cross-border shop.

## 2.6 Conclusion

In this paper, we examined the effects of ePDMP laws on both the states they were enacted in and neighboring states. Following the literature, we find that opioid sales fall in states that adopted electronic access PDMPs. After controlling for border and spillover effects, we estimate that ePDMP laws reduce per-person opioid sales by 5.6% from the median sales in 2006, a considerable drop because the laws should only affect the fraction of users doctor or pharmacy shopping. We find that the decrease was driven by border counties in particular, where sales decreased 10.2% post-ePDMP. We also find that ePDMP laws reduce opioid overdoses in a state, with approximately a 12.3% decrease relative to per-capita mortality in 2006. These findings confirm the understanding in the literature that PDMP laws are effective in curbing the opioid epidemic.

The decentralized adoption of ePDMPs created opportunities for individuals to cross the state border to acquire opioids from a less restrictive state. Counties on the border are more likely to be destinations for doctor or pharmacy shopping, due to the lower travel cost from other states. Our paper is the first to document a differential pattern in opioid use and a differential response to law changes in counties due to their proximity to the state border. Before the enactment of an ePDMP, border counties have significantly higher opioid sales and lower rates of overdose as compared to inland counties of the same state. When the state adopts an ePDMP, its border counties experience a larger drop in sales and a smaller decrease in mortality. In addition, when the nearby state adopts an ePDMP, we observe a larger increase (or smaller decrease) in sales in counties neighboring the law change state as compared to inland counties in that same state. The spillover effect indicates that the benefits of ePDMPs are partially mitigated because individuals purchase opioids from neighboring states when their state adopts an ePDMP.

The qualitative differences between border and inland counties in opioid sales and

overdose have implications for all studies on the opioid crisis. Previous studies treat each state as an independent market and assume that local opioid sales have a one-to-one mapping to local opioid consumption. This simplifying assumption is the correct one to make in many situations. For example, in the study of the OxyContin reformulation, each state is treated with the same regulatory change. Spillover effects due to preexisting regulatory or cultural differences still exist, but they are irrelevant to measuring the impact of OxyContin reformulation on opioid use. However, in many other situations, where change takes place on a state-by-state basis, treating each state as an independent market may bias the estimation. In the case of PDMP laws, not accounting for cross-border sales overestimates the benefits of the law change.

The spillovers we have identified in this paper have implications beyond the opioid crisis. We have documented a direct negative externality from having state-based opioid policies instead of a national one. In a counterfactual world where all states adopt electronic access of PDMPs at the same time, all states would get the sales reduction without the increased sales from cross-border trafficking. These findings speak to the advantages and disadvantages of a federalist system. On one hand, decentralization allows each state to experiment and adopt politics based on their own conditions. Information from early adopters could flow to late adopters, thereby providing late adopters with real-world data on policy effectiveness. On the other hand, decentralization kills coordination and there is often a cost in failures to coordinate. Individuals, resources, and businesses are often not confined to one location. Regulatory differences among states allow entities unwilling to comply to move to a different state, thereby offsetting the positive benefit of new regulations.

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*Chapter 3*

# AIR POLLUTION AND SCHOOL ABSENCES IN NEW YORK CITY

DANIEL GUTH

In this paper, I analyze the effects of changes in day-to-day air pollution levels on daily absences for New York City schools from 2006 to 2019. I combine EPA air quality data with absences for more than 1,600 schools and use wind as an instrument for transport of air pollution. I estimate that an additional 1 $\mu g/m^3$ of PM2.5 pollution increases absences across all schools by 44 per 100,000 students, and an extra part-per-billion (PPB) of Ozone increases it by 29 per 100,000 students. PM2.5 pollution has the largest effects on elementary and middle schools, but Ozone has largest effects on high schools. Examining trends across 14 years of pollution and absences, my results suggest that the decrease in average daily PM2.5 pollution of 5 $\mu g/m^3$ from 2006 to 2019 led to at least 381,000 fewer absences across NYC schools and increased education spending by $19 million every year. This work shows the improvements over time in air quality in New York City but also highlights the disparate impacts of air pollution.

## 3.1 Introduction

Air pollution has darkened American skies for a century. It is well understood on the individual level that air pollution is a risk factor for a variety of respiratory and cardiovascular diseases. However, measuring the effects of air pollution on human health is difficult because the consequences unfold over time and many other factors influence health outcomes. In this paper, I estimate the consequences of exposure to air pollution in day-to-day life by analyzing its effects on school absences for millions of students in New York City. I find strong evidence that increased levels of PM2.5 and Ozone cause additional students to be absent over the next few days, and this effect persists even when pollution levels are below federal limits.

Much of the research on air pollution in the US was done when when carbon monoxide (CO) levels were frequently above the federal limit and the smallest particulate matter that could be measured was 10 microns (PM10). More recent

work demonstrates that small particle pollutants (PM2.5) are particularly harmful to human health because their smaller size allows them to penetrate deeper into the lungs. Deryugina et al., 2019 found large increases in mortality and medical spending among Medicare recipients due to PM2.5 pollution, as well as annual benefits of more than $24 billion from national reductions in PM2.5 emissions from 1999 to 2013. I contribute to this literature by providing evidence for the negative effects of PM2.5 and Ozone on NYC children while also showing in the same context that CO and other pollutants do not cause similar harms. I analyze school absences from 2006 to 2019 and find suggestive evidence that the reduction in average PM2.5 pollution over that time contributed to a citywide decline in student absences. My data and methodology also allow me to separately identify the effects of air pollution on each school, and I find novel results for high school absences caused by Ozone.

Previous work on pollution and school absences took place in different pollution environments and with less data. Currie, Hanushek, et al., 2009 analyzed school absences aggregated by six-week attendance blocks for Texas in the late 1990s and found that carbon monoxide (CO) was responsible for large increases in absences, PM10 and Ozone had a small effect on absences, and they could not evaluate PM2.5 because atmospheric measurements did not exist at the time. I analyze absences from 2006 to 2019 in NYC at pollution levels much lower than in Currie, Hanushek, et al., 2009, and I find a statistically significant number of absences caused by PM2.5 and Ozone pollution. I use absences at the daily level and develop a framework that allows me to estimate the effects of increased air pollution on different days while controlling for seasonal variation. I further leverage modern climate modeling to use exogenous variation in daily wind direction to show that wind-carried PM2.5 and Ozone directly causes absences. NYC has PM2.5 pollution comparable to the median county in the United States but Ozone pollution higher than the median county, so school districts across the country may have similar amounts of absences caused by air pollution. My estimates represent a lower bound for the harms experienced by other countries, though, most of which have much higher PM2.5 or Ozone concentrations.

School absences represent a child missing class for whatever reason. In papers that track individual students and further break down the cause of absences, respiratory-related absences are the most responsive to pollution. Gilliland et al., 2001 found that Ozone led to increases in both upper and lower respiratory illnesses. Gilliland et al. note that these kinds of respiratory issues often do not land children in the hospital,

and so “school absences caused by respiratory illnesses may usefully represent the first tier of adverse effects that are far more common than severe adverse effects." S. Chen, Guo, and Huang, 2018 studied the effects of pollutants in China from 2013 to 2015, finding that PM2.5 and Ozone particularly increased respiratory-illness related absences. I do not observe reasons for school absences in my NYC data, so for each school I control for the average number of absences on less-polluted days and seasonal effects using multiple years of data. After netting out those effects, the remaining absences caused by pollution are likely due to respiratory issues.

There are two advantages to my approach. First, by analyzing school absences at the daily level I can look at the same school cohort before and after a high-pollution event. This analysis allows each school to act as both a control and treatment group on different days, and I can test multiple lag structures to identify the time frame at which pollution causes absences. Specifically, I find differences between the time it takes PM2.5 pollution to cause absences (largest same-day effects) and the time takes Ozone pollution to cause absences (one or two days). Second, by using wind as an instrument, pollution shocks (after accounting for snow) are uncorrelated with school absences except through wind-carried pollutant transport. This allows me to get causal estimates of pollution’s effects on school absences using years of variation in air quality levels.

I use two kinds of regression models to estimate the effects of air pollution on absences at the school-day level. In the first set of models, I use the pollutant concentration (for PM2.5 in terms of $\mu g/m^3$, and Ozone in parts-per-billion) and directly regress school absences on air pollution through ordinary least squares (OLS). The second set of regressions uses wind direction as an instrumental variable (IV) in a two-stage least squares procedure (2SLS). In the first stage, daily PM2.5 or Ozone pollution is regressed on wind direction, and then the wind-fitted pollution is used in the second stage absence regression. All models include a panel of fixed effects for day of week, school, school times school year, and school times month to control for unobserved school and seasonal characteristics. To the extent that wind satisfies the exclusion restriction (that is, wind being uncorrelated with school absences except through pollution-transport), this IV regression produces causal estimates of PM2.5 and Ozone on absences. I also show that snowfall in NYC has large effects on absences and is correlated with wind direction, so the main regressions are run on months without snow to avoid model misspecification and violating this exclusion principle. In the robustness section I include OLS

regressions run on all months and directly controlling for snow, but measurement error makes it difficult to separate absences caused by snowfall from absences caused by air pollution.

Before previewing the results, it's worth considering these effects relative to the overall burden of pollution. First, we should expect the coefficient on pollution for absences to be relatively small, because there are many factors that cause sickness or for students to otherwise miss school. For instance, snowfall in NYC sometimes cancels school but short of that, icy roads often cause some students to be absent. Other non-illness related absences can be caused by students travelling for holidays or otherwise being unable to get transportation to school. Illnesses can also be caused by seasonal flu or infectious respiratory diseases, although it is generally understood that air pollution impairs the body's immune system and ability to fight off disease. At the same time, air pollution negatively affects everyone and in many more ways than are captured in school absences. Elevated levels of air pollution have been shown to be associated with reductions in test scores, increases in mortality and hospital spending, as well as increase in crime rates.[1] This paper provides a precise estimation of one of the short-run causal effects of air pollution, but these estimates are lower bounds on the societal costs of pollution.

Analyzing school absences allows me to test multiple lag structures without issues of displacement or "harvesting" that arise when analyzing mortality or hospitalization data. In those settings, the effects of pollution might be strongest among people who were going to die or require hospitalization soon regardless of exposure, so researchers often average pollution measurements or mortality over many days. Absences are high-frequency indicators of student health where I can separately identify the effects of pollution on day-of absences from its effects on absences one or more days later. I can identify these changes because I have absence data on the same group of students across many days, and wind direction provides day-specific pollution shocks. My extended results show that PM2.5-caused absences are largest on the day-of pollution exposure, with somewhat smaller effects one or two days afterwards, and the effect become approximately zero three or more days later. I find that Ozone-caused absences are also approximately zero after three days, but I find the largest effect on absences two days later. These findings are significant because

[1] Papers on air pollution and test scores include Xin Zhang, X. Chen, and Xiaobo Zhang, 2018 and Ebenstein, Lavy, and Roth, 2016. Mortality effects from Medicare populations are analyzed in Deryugina et al., 2019 and Di et al., 2017. Crime papers include Bondy, Roth, and Sager, 2020 and Herrnstadt et al., 2021.

they represent illnesses that are generally too minor to appear in hospital data but are common in the everyday life of children. In this paper I am able to quantify both the effect size as well as timescale at which pollution causes absences.

I find that elevated levels of PM2.5 and Ozone each lead to absences across all schools and in a variety of regression specifications. My preferred specification, the IV regression with full set of fixed effects, finds that every additional 1 $\mu g/m^3$ of PM2.5 leads to 44.3 more absences per 100,000 students and every additional part-per-billion (PPB) of Ozone leads to 29.8 more absences per 100,000 students. The average school day in NYC has an absence rate of approximately 8%, and going from the median PM2.5 concentration of 8.05 $\mu g/m^3$ to the top decile of most polluted days at 16.98$\mu g/m^3$ times that coefficient implies an additional 0.395% (one-twentieth the daily mean) absences that day. Doing the same comparison for Ozone, going from the median pollution day of 32.6 PPB to the top decile of 60.8 PPB implies an additional 0.84% (one-tenth the daily mean) absences two days later. I document a decline in both school absences and daily PM2.5 concentration over this period, and multiplying the decrease of 5 $\mu g/m^3$ from 2006 to 2019 suggests that there are 0.2% (approximately 2100) fewer absences across NYC schools every day because air quality improved. In contrast to the PM2.5 trends, I find that Ozone concentration slightly increased over this period and continues to cause school absences through the present day. These are lower bounds for the effects of air pollution, though, and cumulative exposure may cause harms not captured in my analysis.

Finally, to understand the treatment heterogeneity I separately analyze each school's absences and compare regression coefficients across schools. I find that elementary and middle schools had much larger PM2.5-induced absences compared to high schools. This difference in effect size is consistent with medical literature on these kinds of respiratory issues, because children's immune systems are developing and they are known to be more susceptible to a variety of illness. Ozone, in contrast, has largest effect sizes for high schools, followed by elementary schools, and smallest effect sizes for middle schools. I am unaware of research that would suggest high school students are more (and middle school students less) susceptible to Ozone-related health issues, but high school students who participate in after school sports programs might spend hours outside during hours when Ozone concentration is highest. If that is the case, high school students might have the most exposure to Ozone pollution and further research should investigate the relationship between

Ozone and other health outcomes. Breaking down the effect size for schools by measures of student poverty, I find the elasticity of PM2.5-related absences is higher for schools with more students classified as economically disadvantage. This means that the reduction in PM2.5 pollution from 2006 to 2019 might have had largest benefits for poorer students. Repeating that exercise for Ozone, I find that economic status has less of an effect on pollution elasticity.

The rest of the paper is as follows. Section 2 provides background on air quality regulations and previous papers on school absences. Section 3 outlines the absence, air pollution, and weather data. Section 4 describes the methodology and instrumental variables (IV) approach. Section 5 provides results for OLS, IV, and individual school regressions. Section 6 includes extensions of the model to compare lagged days of pollution and OLS results including winter months controlling for snow. Section 7 concludes and discusses the implications for future pollution regulations.

## 3.2 Background

This section gives background on air pollution and related literature of its effects on humans. Most of the research on air pollution has been done using observational studies which can identify correlations but are less effective at estimating the effects from marginal changes in pollutant concentrations. More recently, environmental economists have developed methods for exploiting quasi-random pollution shocks or changes in regulation that allows for causal identification and estimation of counterfactuals for different pollution levels. I continue this line of research by analyzing the effects of changes in air pollution on school absences, which allows for comparing the same set of students on days of high and low pollution.

### Effects of Air Pollution

Air pollution leads to many health problems, but mortality is the most studied because it is the worst possible outcome and governments have kept detailed death records for decades. Anderson, 2009 describes the history of research on pollution and mortality, which have improved over time due to better pollution measurements and computational power. The landmark six-cities study of Dockery et al., 1993 found large increases in mortality from PM2.5 over a 14-year observational period. Follow-up studies, such as Lepeule et al., 2012, found that average PM2.5 concentrations declined but were still associated with higher mortality. Deryugina et al., 2019 analyzed mortality among Medicare populations using daily changes in pollution from wind and found significant effects from PM2.5 across the US. The

other population that is often studied in this context is newborn infants, who have developing lungs and are especially affected by pollution. Currie and Neidell, 2005 finds that reductions in California of CO and PM10 pollution in the 1990s likely prevented more than one thousand infant deaths. Several papers studying pollution from car exhaust, such as Knittel, Miller, and Sanders, 2016, find similar effects of particulate matter on infant mortality. Currie and R. Walker, 2011 finds that the introduction of E-ZPass reduced congestion and resulted in nearly 10% fewer premature births near toll plazas. School-aged children are underrepresented in these kinds of studies because they are rarely hospitalized for respiratory issues, so absences are a useful kind of high-frequency data to analyze as health outcomes.

A separate strand of literature analyzes the educational effects of pollution on test scores. Ebenstein, Lavy, and Roth, 2016 analyzed students retaking exams in Israel and found that increased PM2.5 pollution on the day of the exam reduced student test scores. Lower test scores then lead to reductions in average earnings and university completion later in life. Marcotte, 2017 finds that students score between 1 to 2% lower on tests on days with high pollen or PM2.5 concentration. Xin Zhang, X. Chen, and Xiaobo Zhang, 2018 showed negative effects of both transient and cumulative air pollution on verbal and math test scores. Heissel, Persico, and Simon, 2022 finds that students who transitioned to schools downwind of highways had lower test scores and more absences.

**Regulations on Air Pollution**

There are costs associated with reducing air pollutant emissions, and a better understanding of the harms caused by air pollution informs cost-benefits analysis for regulation. W. R. Walker, 2013 examines industrial plants newly affected by the 1990 Clean Air Act Amendments and finds they led to more than $5 billion in lost wages, but notes that this is approximately two orders of magnitude less than the health benefits from pollution reduction. Currie and R. Walker, 2019 reviews the economic literature on the Clean Air Act, distinguishing between papers using casual identification on short-run changes in pollution versus longitudinal changes in yearly pollution. The EPA updated the PM2.5 regulations in 1997[2], but PM2.5 and Ozone pollution below the federal limits still has negative effects. Counties in the US that are above the annual limits are designated as "non-attainment", but in this paper I am able to identify costs from pollution at levels that are well below

[2]The updated thresholds were in large part due to Dockery et al., 1993 which found large increases in mortality from PM2.5 even below the previous threshold.

these limits. I show large reductions of PM2.5 concentration in NYC from 2006 to 2019 [3], but Ozone concentration increased over this time and continues to cause absences.

**Literature on School Absences**

Ransom and Pope III, 1992 was one of the first papers to study the association between PM10 and school absences for schools in Utah. Hales et al., 2016 continues that Utah analysis into the 2010s using PM2.5 and uses a "control" district exposed to lower levels of pollution to explain residual absences caused by pollution. Gilliland et al., 2001 identified a longitudinal sample of fourth graders and tracked the cause of their absences over six months, finding increases in Ozone concentrations were associated with more respiratory-illness related absences. L. Chen et al., 2000 similarly found absences caused by CO and Ozone for Nevada in 1996-1998, but found a negative association from PM10. The most recent paper on school absences is S. Chen, Guo, and Huang, 2018, which analyzed school absences and their causes for two years in Guangzhou, and found that air pollution increased respiratory illness-related absences. The mean daily absence rate in that paper was 0.22%, though, with worse air quality than in NYC so comparatively each of those absences represents more severe illness. Currie, Hanushek, et al., 2009 is the most comprehensive school absence study in the US, but it was limited to analysis of 6-week absence periods and PM10 instead of PM2.5 concentrations. I update analysis of school absences in the United States for years 2006 - 2019, which allows me to analyze Ozone and PM2.5 to identify causal effects of absences at the individual day level. Daily absence data across years allows me to identify the timescale at which pollution causes absences as well as document a decline in absences over time due to reductions in PM2.5 pollution.

In addition to the negative health effects, missing school causes a child to also miss opportunities to learn. Research that uses individual student-level data is able to study the effects of absences themselves on other educational outcomes. Most of the research on absenteeism in this area analyzes the educational effects of chronic absenteeism, such as Chang and Romero, 2008 or Allen, Diamond-Myrsten, and Rollins, 2018, because that has large effects on graduation rates. Less attention is focused on students who are infrequently absent, they but still experience learning loss. Goodman, 2014 finds that school absences had more effect than school

[3] This link, shows the reductions in pollution by neighborhood using NYCCAS measurements from approximately 100 locations.

closures on test performance, suggesting that teachers are less able to accommodate students who miss school at different times. Liu, Lee, and Gershenson, 2021 connects school absences in specific class periods and shows that they can reduce the probability of graduating. I do not directly test for those educational outcomes in my analysis, but my research provides a method and preliminary evidence showing that PM2.5 and Ozone in NYC over this period caused school absences. Further research is warranted to understand the effects of these pollution-induced absences on educational outcomes.

## 3.3 Data

This section describes the different data sources combined to analyze school absences. Table 3.1 provides school and pollution summary statistics for each school year. School absence data is described first, then air pollution data, and then finally weather data comprising wind and snow.

Table 3.1: NYC daily absences and pollution concentration by school year, with PM2.5 and Ozone calculated starting in August. Average absences are expressed as a daily percent weighted by school size, and elementary absences calculated for the approximately 1,182 schools that are either elementary or middle schools.

| School Year | Average Daily PM2.5 ($\mu g/m^3$) | Average Daily Ozone (PPB) | Number of Schools | Average Absences (%) | Average Absences Elementary (%) | Total Enrollment |
|---|---|---|---|---|---|---|
| 2006-2007 | 12.8 | 32.73 | 1313 | 9.88 | 7.85 | 917876 |
| 2007-2008 | 13.06 | 34.36 | 1369 | 9.58 | 7.42 | 924888 |
| 2008-2009 | 10.54 | 31.32 | 1428 | 9.47 | 7.56 | 931849 |
| 2009-2010 | 9.85 | 35.27 | 1491 | 8.8 | 6.88 | 957476 |
| 2010-2011 | 10.36 | 36.25 | 1525 | 9.01 | 7.03 | 967880 |
| 2011-2012 | 9.08 | 34.99 | 1551 | 8.37 | 6.36 | 969586 |
| 2012-2013 | 9.08 | 35.03 | 1577 | 8.53 | 6.72 | 976174 |
| 2013-2014 | 8.92 | 34.25 | 1607 | 8.58 | 6.92 | 980377 |
| 2014-2015 | 8.22 | 34.48 | 1615 | 8.21 | 6.63 | 983312 |
| 2015-2016 | 7.83 | 36.49 | 1608 | 7.97 | 6.37 | 977592 |
| 2016-2017 | 7.22 | 35.07 | 1594 | 8.19 | 6.69 | 972781 |
| 2017-2018 | 7.69 | 35.44 | 1594 | 8.41 | 6.97 | 964161 |
| 2018-2019 | 7.35 | 35.16 | 1565 | 8.38 | 6.97 | 947389 |

### School Absence Data

School absence data for NYC is publicly available from 2006 to 2021. This paper ends at the 2018-2019 school year to avoid Covid-related school absences starting

in 2020.[4] The absence rates are reported at the school level, so all analysis will be done at the school level. I analyze a total of 1669 different schools in NYC from 2006 to 2019, representing more than three million school-day absence records and twelve million student-years. Figure 3.1 shows the number of students as well as the average absences for all schools and for elementary-to-middle schools. The data includes a total of 1182 elementary schools, and previous research has suggested that younger children are most susceptible to pollution, but I also analyze high schools. High schools also have higher absence rates because older students may be absent for work, sports, or other reasons. One advantage of my analysis is that NYC schools are all in the same district and thus have the same regulations and absences procedures.[5] For methodological reasons, I do not directly include school demographics as fixed effects in the absence regressions.[6] In one specification I run each school separately and group the results by type (elementary, middle, or high) and socioeconomic information (fraction of students classified as living in poverty).

**Air Pollution Data**

Air pollution data comes from the EPA's Air Quality System (AQS) that maintains a network of outdoor monitors across the United States.[7] Using this data, pollution concentrations are measured multiple times throughout the day and then aggregated to form daily readings. The EPA regulates and measures six principal pollutants: particulate matter (PM2.5)[8], Ozone, NO2, SO2, CO, and lead. AQS data on airborne lead concentrations is sparse, so lead is not analyzed in this paper. Outdoor concentrations of CO never went above 50% of the daily limit or near harmful levels from Currie, Hanushek, et al., 2009, so it is analyzed in Appendix Section C but has no significant effect.[9] OLS results for NO2 and SO2 are also reported in

[4]School absence data is available at this link. NYC School locations were obtained using location data from 2012 to 2013 school data and a few dozen school locations from later years.

[5]In NYC when a student is suspended (Suspension Link), they receive alternative instruction and are not marked absent unless they miss those activities. Alternative Reference.

[6]Including yearly school student demographic variables in the model corresponds to trying to estimate and control for the average daily absence rate by race. Across all of NYC and 14 years of data, I do not think it is meaningful to try and estimate those quantities using ecological inference at the school level, so I instead use school fixed effects. Including school by year fixed effects similarly controls for changes in the student body by year without assuming race has a constant absence elasticity across the entire sample.

[7]This link allows you to download daily data by state and year.

[8]The AQS stopped measuring PM10 concentrations for New York in approximately 2006, so PM10 is not analyzed in this paper.

[9]Different pollutants are aggregated on different time-scales: PM2.5 is reported as daily mean concentration; Ozone is reported as daily maximum 8-hour concentration; SO2 is reported as daily maximum 1-hour concentration; CO is reported at daily maximum 8-hour concentration; NO2 is

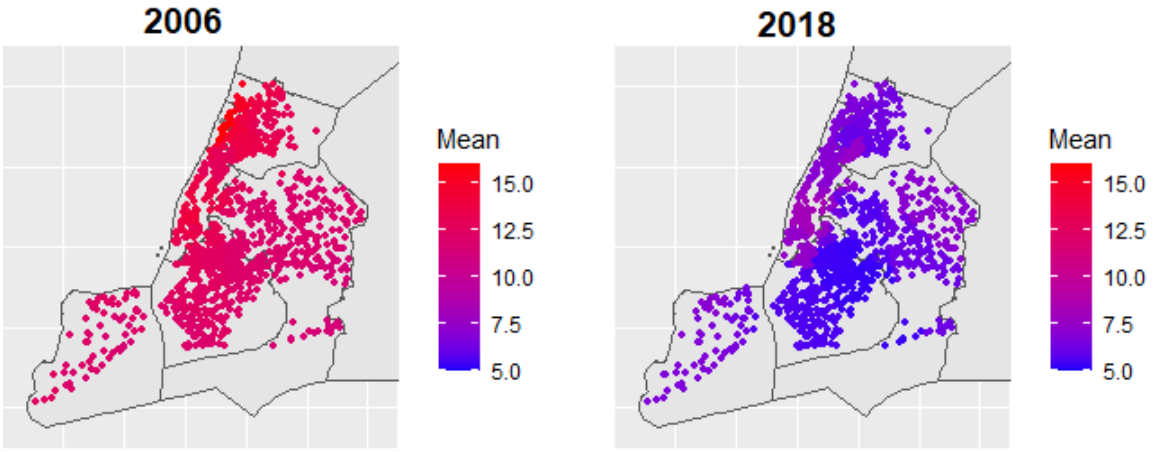


Figure 3.1: NYC average PM2.5 by school using a common scale for 2006-2007 and 2018-2019 school years. A total of 1,312 schools are plotted in 2006 and 1,551 schools in 2018. Borough-wide PM2.5 averages for (Bronx, Brooklyn, Manhattan, Queens, Staten Island) school days in 2006 were (13.49, 11.73, 13.44, 11.83, 11.77) and in 2018 were (6.50, 5.71, 7.40, 6.36, 6.61)

the appendix, with no significant results. Thus, the main pollutants of interest are PM2.5 and Ozone.[10]

For every school and every day over this period, I assigned each school the pollution measurement from the closest PM2.5 or Ozone monitoring site. As described in Zou, 2021, many PM2.5 monitoring sites are active on 1-in-3 or 1-in-6 day schedules, so individual schools are assigned multiple monitoring stations in a single year. The median distance between schools and PM2.5 monitoring station across all days was 1.91 miles, 93% of all school-day monitor distances were below 5 miles, and 99.5% of all distances were less than 10 miles. There are fewer Ozone monitoring stations, so the median distance between schools and Ozone monitoring station across all days was 4.14 miles, 97.2% of all school-day monitor distances were less than 10 miles, and 99.6% of all distances were less than 12 miles. Figure 3.1 shows the average PM2.5 measurement for each school in school years 2006-2007 and 2018-2019 across all school days. PM2.5 pollution in 2006 was highest in the Bronx and Manhattan whereas in 2018 Manhattan is most polluted, but PM2.5 air quality improved everywhere in NYC over this time. NYC is dense and so the absolute difference in pollution between boroughs on the same day is not large, but there is significant variation in average pollution across days and years. Wind is discussed

reported at maximum 1-hour concentration.

[10]Pollution is used here in direct atmospheric concentrations, whereas some other papers use the scaled Air Quality Index (AQI) with Technical Documentation here.

in the next section and acts as a daily pollution shock common to all schools in NYC.

**Weather Data**

**Wind Data**

As shown in Deryugina et al., 2019, wind-carried transport is responsible for significant variation in daily PM2.5 pollution across the US. In this paper, I leverage changes in wind direction using an instrumental variable analysis that exploits this quasi-random pollution shock. Wind data comes from the North American Regional Reanalysis (NARR) from 2006 to 2019[11], with implementation details borrowed from Deryugina et al., 2019. NARR combines multiple data sets to produce consistent and longitudinal atmospheric data at a 32 km by 32 km resolution. Wind conditions are reported as an east-speed (u-component) and a north-south speed (v-component). Simple trigonometric functions allow for the combination of u- and v-wind into a wind direction.[12] Specifically, the wind angle is calculated as $\theta = \frac{180}{\pi}\text{Arctan}(\frac{|v|}{|u|})$ and then converted from 0-360 degrees depending on the signs of $u$ and $v$ following conventions in Deryugina et al., 2019 and elsewhere:

$$WINDDIR = \begin{cases} 270 - \theta \text{ if } u > 0 \text{ and } v > 0 \\ 270 + \theta \text{ if } u > 0 \text{ and } v < 0 \\ 90 + \theta \text{ if } u < 0 \text{ and } v > 0 \\ 90 - \theta \text{ if } u < 0 \text{ and } v < 0 \end{cases}$$

In this form, $WINDDIR$ of zero corresponds to wind blowing from the north into the south, and increasing angle moves clockwise with 90 degrees corresponding to east-west, 180 degrees south-north, and 270 degrees west-east. New York is covered by a single 32km by 32km grid[13], and all school-monitor pairs are assigned the same daily wind direction.

Figure 3.2 shows a regression of average daily PM2.5 in NYC from 2006 to 2019 against daily wind directions. The median daily PM2.5 concentration across this period was 8.05$\mu g/m^3$ and so wind coming out of the south-to-southwest adding an additional 6-7$\mu g/m^3$ is a nearly 75% increase relative to wind from the north.

[11] Link to data at NARR Monolevel Data Catalog, data used is near-suface (10m) uwnd and vwnd. Data description can be found at NARR homepage.

[12] The current model does not use wind speed, which is calculated as $\sqrt{u^2 + v^2}$.

[13] Using NARR's grid, the row coordinate is 259 and the columnn coordinate is 130, centered at latitude and longitude of (40.656, -73.816), near JFK airport.

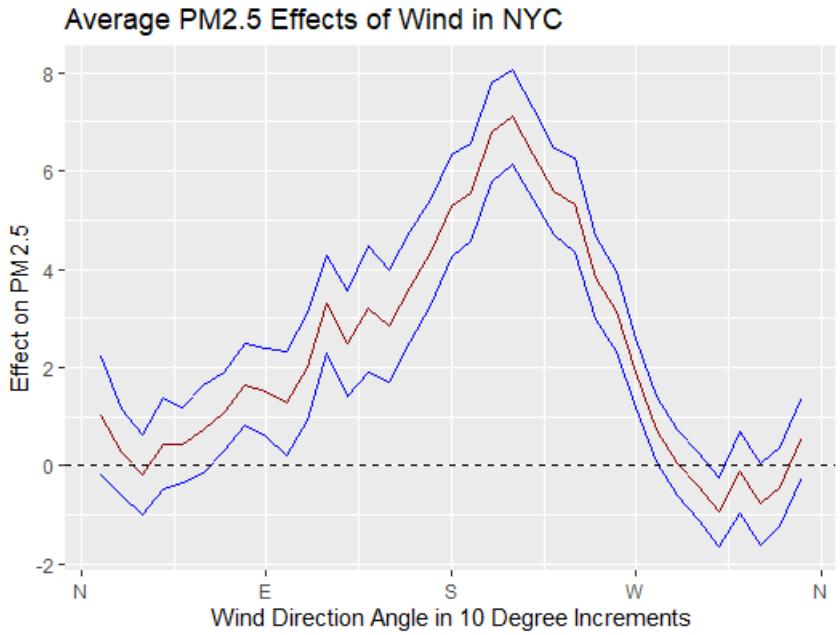


Figure 3.2: Regression of average daily PM2.5 measurements against 10-degree binned wind direction from 2006 to 2019 with Month by Year fixed effects. Regression coefficients are in red with blue lines representing 95 percent confidence intervals using robust standard errors. Omitted comparison angle is zero degrees.

This relationship between PM2.5 and wind in NYC is very similar to a figure in the appendix of Deryugina et al., 2019 for King's County, New York from 1999 to 2013. Figure 3.3 shows the same regression of average daily Ozone on wind direction, and the effect size is significant but smaller (relative to the mean) than for PM2.5 pollution. Against a median daily average of 32.6 PPB, an additional 10 PPB from south-west originating wind compared to east-originating wind adds 30% more Ozone pollution.

### Snow Data

NYC gets snow multiple times a year, and snow can lead to school delays or closings as well as icy roads that cause individual students to be unable to get to school. Controlling for snow is important in analyzing winter school absences, and snow data from 2006 to 2019 is taken from NOAA's Global Historical Climatology Network (GHCN) of snowfall observations. Unfortunately, there are only a few snow surface stations compared the pollution monitoring stations, and so most schools are 10 miles away from where their nearest snow measurements were taken. Even at that distance measured snowfall has a large effect on each school's daily absences, but it

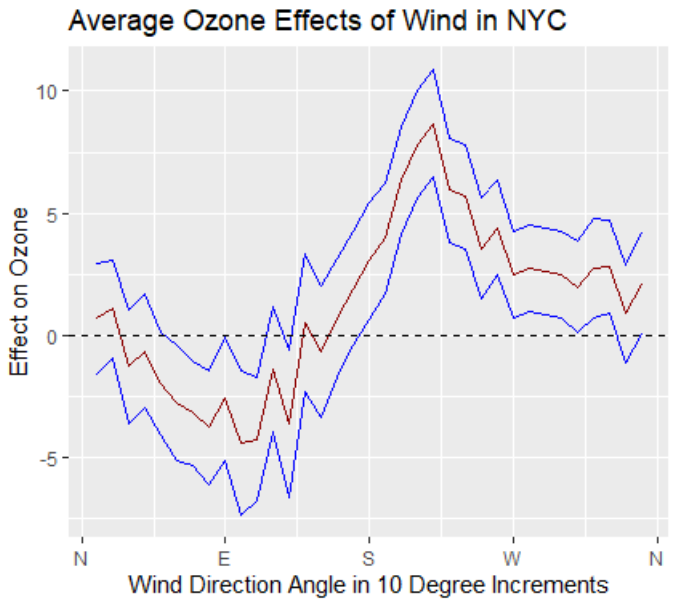


Figure 3.3: Regression of average daily Ozone measurements against 10-degree binned wind direction from 2006 to 2019 with Month by Year fixed effects. Regression coefficients are in red with blue lines representing 95 percent confidence intervals using robust standard errors. Omitted comparison angle is zero degrees.

represents significant measurement error from the amount of snow on the roads near each school. I create indicator variables for whether any snowfall was measured on the day of absences or the day before, as well as additional indicator variables if cumulative snowfall over the last four days was more than 30cm or 50cm. These snow variables are included in an OLS regression for the robustness section, but the main regressions are restricted to the months of April through November to avoid snow's effect on absences.

## 3.4 Methodology

This section considers how to analyze the effects of air pollution on school absences. To inform policy, we want estimates of the harms of pollution that are either unbiased or biased downwards. Daily records of absences and pollution allow for testing multiple lag or dose-response functions, but require controlling for seasonal and non-pollution related absences. I first describe the OLS specification of regressing daily school absences on air pollution. The next subsection describes threats to identification, particularly omitted variables that affect both pollution and absences. I then propose using wind in an instrumental variable regression, because daily wind direction is quasi-random and wind transports air pollution. This section concludes by discussing snowfall, which is measured with error and violates the exclusion

restriction.

**OLS Regression Specification**

The first regression specification directly regresses daily absences on daily pollutant concentration:

$$Y_i^t = \beta_c[\text{Pollution}_i^{t-n}] + \delta_d + \gamma_s + \theta_{sy} + \zeta_{sm} + \epsilon_i^t \tag{3.1}$$

$Y_i^t$ is the percent of students absent for school $i$ at day $t$, the coefficient of interest is $\beta_c$ for each different pollutant. I include $\delta_d$ as fixed effects for day-of-the-week because absences are higher on Mondays and Fridays. I also include school fixed effects ($\gamma_s$), school times year ($\theta_{sy}$), and school times month fixed effects ($\gamma_{sm}$). I test several lag structures using pollution from previous days (shown in Section 3.6) and find different time-lags based on pollutant. The main results for PM2.5 uses the same day of pollution (n = 0) on absences, but Ozone has delayed effect on absences and so I use two-days lagged (n = 2). Results are presented in the main section for PM2.5 and Ozone, while CO, NO2, and SO2 have non-significant results and are relegated to the appendix.

Following papers such as Hales et al., 2016 and S. Chen, Guo, and Huang, 2018, I use day-of-the-week fixed effects because absences are statistically more likely on Mondays and Fridays compared to the middle of the week. To further control for unobserved school and seasonal characteristics, I also include school, school times month, and school times year fixed effects. The remaining error term $\epsilon_i^t$ is also at the school $i$ and day $t$ level, with results weighted by school population and the coefficients calculated using HC2 standard errors.

**Threats to Identification**

Consider the above OLS regression of daily air pollution on school absences. The first problem we might encounter is selection, where students who live areas with low pollution might have different probabilities of being absent than students who live in areas with high pollution. I can control for differences in average absence rates by using school and school times year fixed effects, so that kind of selection is unlikely to be an issue. Differences in elasticity or response to pollution by school, however, would lead to biased estimates. For this reason I also have a specification where I directly analyze each school's absences separately and then compare results across schools. I find that type of school (elementary, middle, or high) changes the elasticity for absences caused by Ozone or PM2.5, and school poverty has effects

on absences caused by PM2.5 pollution.

The next possible threat to identification involves potential changes across years in school absences or the way they are calculated. I am unaware of any such rule changes, but over a period of 13 calendar years there might also be changes in the availability of school busing or prevalence of mental health related absences. Papers like Twenge et al., 2019 and Bitsko et al., 2018 find increases of anxiety and depression among students over this sample period, which could translate into more absences. Empirically, however, I document a significant decline in NYC average absence rates from 2006 to 2019, combined with a decline in PM2.5 pollution and a slight increase in Ozone pollution. My analysis uses day-to-day variation in pollution that is present across all years and is thus an unbiased estimate of effects across time.

The final threat to identification in the school absence context is omitted variables or seasonality that affects both pollution and absences. For PM2.5, concentrations are highest in winter months which have higher absences due to holiday breaks as well as seasonal influenza. For Ozone, concentrations are highest in summer months which have higher absences potentially due to end-of-year effects. Comparing school days from different months could lead to coefficients biased either upwards or downwards depending on the size of these seasonal correlations. For this reason I add month fixed effects, which makes the comparison between high pollution and low pollution days while attempting to keep seasonal effects constant by comparing days within the same month. To analyze pollution variation that is uncorrelated with these kinds of seasonal effects, I use wind direction in an instrumental variables specification. This IV specification also prevents any possible issues of reverse causality, because wind transports air pollution that causes absences but school absences have zero effect on wind direction.

**IV Regression Specification**

Using wind direction as an instrument on daily pollution, this regression attempts to determine the causal effects of air pollution. Following Deryugina et al., 2019 I use wind direction as an instrument for pollution in 2SLS. Wind is binned into eight 45-degree indicator variables. The first-stage specification for PM2.5 is

$$PM2.5_i^t = \alpha_c[\sum_{k=0}^{3} \text{WindDirection}_i^{t-k}] + \delta_d + \gamma_s + \theta_{sy} + \zeta_{sm} + \epsilon_i^t \qquad (3.2)$$

Pollution is estimated for each day $t$ and school $i$. Deryugina et al., 2019 includes two days lagged wind measurements, and I include three lags because absences are calculated in the morning. In the second stage I use this estimated $\widehat{PM2.5}$ based on each school's closest pollution monitor to get the daily measurements:

$$Y_i^t = \beta_c[\widehat{PM2.5_i^{t-n}}] + \delta_d + \gamma_s + \theta_{sy} + \zeta_{sm} + \epsilon_i^t \tag{3.3}$$

I use the same fixed effects as the OLS regression of day of the week ($\delta_d$), school ($\gamma_s$), school times year ($\theta_{sy}$), and school times month ($\zeta_{sm}$) are used in both equations. Figure 3.2 shows a simplified version of the first-stage regression with large variations in daily PM2.5 pollution based on wind direction. The above equations are written in terms of PM2.5, but I repeat the 2SLS procedure for Ozone using two-days lagged pollution and the corresponding lagged wind. Figure 3.3 shows a version of the first-stage regression for Ozone, with significant variation based on wind direction but smaller effects (relative to the mean concentration) compared to the regression of PM2.5 on wind direction. The remaining error term $\epsilon_i^t$ is also at the school $i$ and day $t$ level, with results weighted by school population and the coefficients calculated using HC2 standard errors.

### Individual School Regressions

To directly compare absences between students at the same school as well as examine treatment heterogeneity across schools, I also run the OLS and IV regressions on absences for each school individually in the Results section. I analyze both PM2.5 and Ozone and report coefficients for individual schools, as well as schools grouped by type (elementary, middle, or high) and by fraction of students that are classified as economically disadvantaged. The OLS and IV regressions are set up in the same way as in equations 3.1, 3.2, and 3.3 except without school fixed effects (because these regressions include only one school at a time); instead, the fixed effects are $\delta_d + \theta_y + \gamma_m$ for day of the week, month, and year for all individual school results.

### Snow Problems

Snow poses two challenges in analyzing school absences in New York City. The first problem is that snow has large effects on school attendance but is measured with error. As described in the Data section on snow, most schools are 10 miles away from where their nearest snow measurements were taken. Even at that distance snow has a large effect on each school's daily absences, but it represents significant

measurement error from the amount of snow on the roads near each school. Snow might melt at different rates across NYC depending on plow timing and road salt usage, so the effects on absences for following days might also vary. For this reason the main OLS and IV regressions in the results section are run on months that do not have snow, but I include OLS results using all months in Section 3.6.

The second issue with snow is that it is correlated with wind direction. For the IV wind regression to satisfy the exclusion restriction, there must be no channel by which daily wind affects school absences except through wind-carried pollution. In a meteorological analysis Blechman, 2002 finds that "New York City exhibited preferences for snow with east and northeast winds with few westerly wind events." Figure 3.4 updates the analysis of wind direction on snowy days for NYC from 2006 to 2019, and shows there remains a strong correlation between wind direction and snowfall. Specifically, snow most often occurs on days that have low PM2.5 pollution due to winds from the north and north-east and rarely occurs on high PM2.5 days with winds coming from the south and west. Snowfall has a strong effect on school absences, so the IV exclusion restriction fails on those days and the coefficient is biased downwards. The main IV results are thus run on months without snow, April though November. More localized snow data or improved functional forms for the snow-absence relationship could allow for analysis of winter months. I am unaware of other omitted variables or correlations that would violate the exclusion restriction for my IV regressions, but if there were, my OLS estimates still provide evidence of the same magnitude and timing of absences caused by PM2.5 and Ozone pollution.

## 3.5 Results

### OLS Results

This section provides OLS regression results for PM2.5 and Ozone across all years from 2006 to 2019. As described in Section 3.4, snow is measured with error and causes large amounts of absences unrelated to pollution, so the main OLS regressions omit snow-containing months December through March. OLS estimates for all months and directly controlling for snow are presented in Section 3.6.

Table 3.2 shows the PM2.5 OLS results of same-day pollution on absences. Against an average of approximately 8% (8000 per 100,000 students) students absent per day across NYC during this period, the coefficient represents absences per 100,000 students caused by an additional 1 $\mu g/m^3$ of daily PM2.5 pollution. The coefficients are positive in all specifications, although adding more fixed effects reduces the

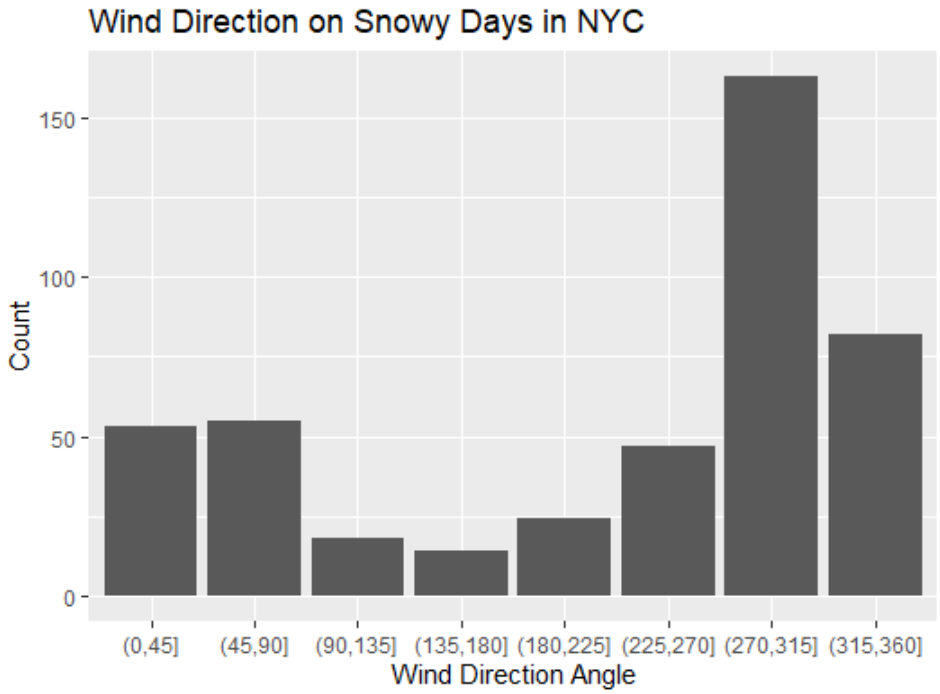


Figure 3.4: Wind direction of NYC days that had non-zero snowfall from 2006 to 2019. With a total of 456 days with snow accumulation over that range, 245 (53.7%) had wind coming from north-to-northwest (270 to 360 degrees).

magnitude. The preferred specification is Model 5 with all fixed effects, showing an extra 25.1 absences per $\mu g/m^3$ of PM2.5. Going from the median NYC day of PM2.5 pollution of $8.05\mu g/m^3$ to the 90th percentile day of $16.98\mu g/m^3$ times that coefficient implies an additional 224 absences per 100,000 students that day.

Table 3.2: OLS regression of same-day PM2.5 pollution on daily absences for months without snow (April through November).

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_1$ - $\mu g/m^3$ PM2.5 | 83.66*** | 60.18*** | 39.14*** | 48.45*** | 25.06*** |
| | (1.33) | (1.00) | (0.89) | (1.03) | (0.92) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.000 | 0.536 | 0.616 | 0.538 | 0.618 |

Table 3.3 shows the Ozone OLS results of two-days lagged pollution on absences. Against an average of approximately 8% (8000 per 100,000 students) students absent

per day across NYC during this period, the coefficient represents absences per 100,000 students caused by an additional part-per-billion (PPB) of daily Ozone pollution two days earlier. As shown in later Section 3.6, Ozone has largest effects on absences one or two days following exposure. The coefficients are positive in all specifications, and school times month fixed effect especially reduces the coefficient magnitude. This is because Ozone is highest in hot summer months, and comparing absences between months may attribute seasonal effects to differences in average Ozone, so it is important to include fixed effects to control for monthly variation. The preferred specification is Model 5 with all fixed effects, showing an extra 20.9 absences per 100,000 students for every PPB of Ozone. Going from the median NYC day of Ozone pollution of 32.6 PPB to the ninetieth percentile day of 60.8 PPB times that coefficient implies an additional 589 absences per 100,000 students two days later.

Table 3.3: OLS regression of two-days lagged Ozone pollution on daily absences for months without snow (April through November).

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_2$ - PPB of Ozone | 57.42*** | 76.31*** | 16.71*** | 79.82*** | 20.91*** |
| | (0.52) | (0.38) | (0.42) | (0.38) | (0.42) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.011 | 0.554 | 0.616 | 0.557 | 0.619 |

**Wind IV Regression Estimates**

This section provides IV regression results for PM2.5 and Ozone across all years from 2006 to 2019. As described in Section 3.4, snow is measured with error and causes large amounts of absences unrelated to pollution, so the main IV regressions omit snow-containing months December through March. Figures 3.2 and 3.3 show the first stage regressions of daily PM2.5 or two-days lagged Ozone on school absences.

Table 3.4 shows the PM2.5 IV results of same-day pollution on absences. Against an average of approximately 8% (8000 per 100,000 students) students absent per day across NYC during this period, the coefficient represents absences per 100,000 students caused by an additional 1 $\mu g/m^3$ of daily PM2.5 pollution. The coefficients

are positive in all specifications, although adding more fixed effects reduces the magnitude. The preferred specification is Model 5 with all fixed effects, showing an extra 44.2 absences per 100,000 students for every $\mu g/m^3$ of PM2.5 pollution. Going from the median NYC day of PM2.5 pollution of $8.05\mu g/m^3$ to the 90th percentile day of $16.98\mu g/m^3$ times that coefficient implies an additional 395 absences per 100,000 students that day. Comparing the PM2.5 IV coefficient magnitude to the OLS results, with no fixed effects the OLS coefficient is approximately 60% larger than the IV coefficient, but in Model 5 the IV coefficient is 75% larger than the OLS coefficient.

Table 3.4: IV regression of same-day PM2.5 pollution on daily absences for months without snow (April through November).

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_1$ - $\mu g/m^3$ PM2.5 | 50.82*** | 50.53*** | 49.21*** | 57.19*** | 44.29*** |
| | (2.29) | (1.59) | (1.44) | (1.63) | (1.46) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.013 | 0.508 | 0.597 | 0.511 | 0.600 |
| $R^2$ Adj. | 0.013 | 0.508 | 0.596 | 0.510 | 0.600 |

Table 3.5 shows the Ozone IV results of two-days lagged pollution on absences. Against an average of approximately 8% (8000 per 100,000 students) students absent per day across NYC during this period, the coefficient represents absences per 100,000 students caused by an additional part-per-billion (PPB) of daily Ozone pollution two days earlier. As shown in later Section 3.6, Ozone has largest effects on absences one or two days following exposure. The coefficients are positive in all specifications and, similarly to Table 3.3, the school-times-month fixed effect especially reduces the coefficient magnitude. The preferred specification is Model 5 with all fixed effects, showing an extra 29.8 absences per 100,000 students for every PPB of Ozone. Going from the median NYC day of Ozone pollution of 32.6 PPB to the ninetieth percentile day of 60.8 PPB times that coefficient implies an additional 840 absences per 100,000 students two days later. Comparing the Ozone IV coefficient magnitude to the OLS results, the IV coefficient is consistently larger than the OLS coefficients regardless of fixed effects, and it is approximately fifty percent larger in Model 5.

Table 3.5: IV regression of two-days lagged Ozone pollution on daily absences for months without snow (April through November).

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_2$ - PPB of Ozone | 79.05*** | 88.00*** | 30.16*** | 91.38*** | 29.83*** |
| | (1.48) | (1.07) | (1.04) | (1.04) | (1.03) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.020 | 0.526 | 0.596 | 0.531 | 0.601 |
| $R^2$ Adj. | 0.020 | 0.525 | 0.596 | 0.531 | 0.600 |

**Individual School IV Analysis**

This section presents PM2.5 and Ozone IV results from individual schools, with the corresponding OLS results relegated to the appendix. Results are shown first for all schools in histogram form, then grouped by school type, and finally grouped by percentage of students considered economically disadvantaged.

Figure 3.5 shows the PM2.5 IV results run on each individual school. The vertical red line marks zero effects on absences, and 176 out of 1623 (10.8%) schools have negative PM2.5 coefficients. Running each school individually increases the standard errors, though, and so only 319 out of 1623 (19.7%) of schools have IV coefficients that are significantly above zero. Comparing across all schools, the median IV coefficient is 49.4 and the mean IV coefficient is 52.2, both of which are slightly larger than the full regression IV coefficient of 44.3. This distribution of effect size by school is consistent with PM2.5 causing absences combined with some noise by school, but it is possible some schools do not have absences caused by PM2.5 pollution.

Figure 3.6 shows the Ozone IV results run on each individual school. The vertical red line marks zero effects on absences, and 188 out of 1623 (11.6%) schools have negative coefficients. Running each school individually increases the standard errors, though, and so only 321 out of 1623 (19.8%) of schools have IV coefficients that are significantly above zero. Comparing across all schools, the median IV coefficient is 32.2 and the mean IV coefficient is 38.6, both of which are slightly larger than the full regression IV coefficient of 29.8. The distribution of effect size by school is consistent with Ozone causing absences combined with some noise by

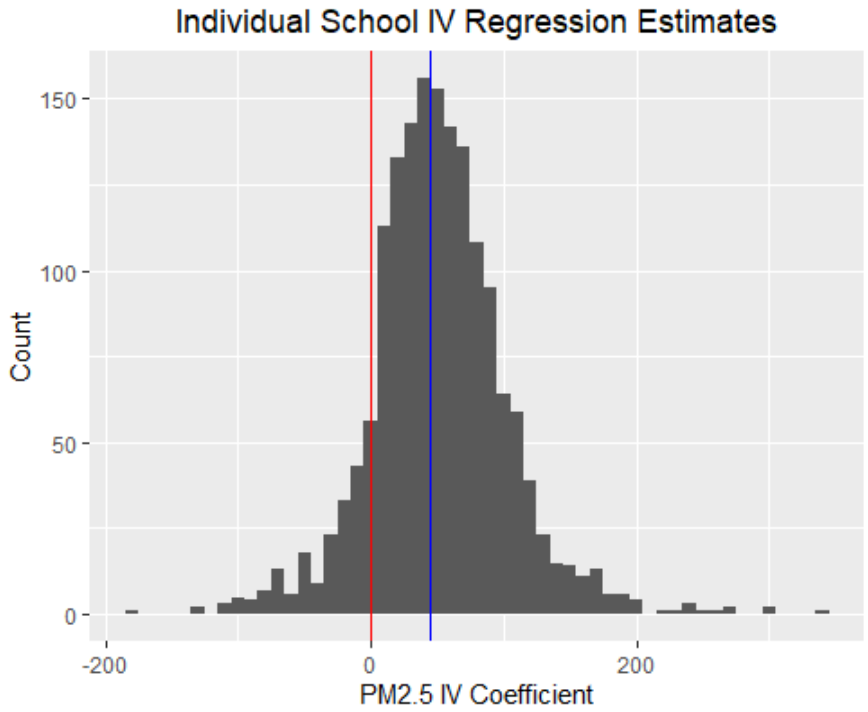


Figure 3.5: PM2.5 IV regression with every school run individually. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school. The red line is at 0 and the blue line represents the coefficient in the full data.

school, but it is also possible some schools do not have absences caused by Ozone.

Figure 3.7 presents a box plot of PM2.5 IV coefficients grouped into elementary, middle, and high schools. The median PM2.5 coefficients by type are 53.9 for elementary schools, 55.6 for middle schools, and 35.3 for high schools. The difference between the average elementary and middle school coefficient is not statistically significant ($p = 0.12$), but both are statistically larger than the average coefficient among high schools; in fact, 25% of all high schools have a negative PM2.5 coefficient. Previous work has generally found that PM2.5 causes absences for elementary or middle schools, but high schools' absences are often not analyzed. I include all types of schools in the main analysis, but this difference by school type fits with the medical understanding that PM2.5 harms younger children more than high school aged children.

Figure 3.8 presents a box plot of Ozone IV coefficients grouped into elementary, middle, and high schools. The median Ozone coefficients by type are 30.5 for elementary schools, 17.8 for middle schools, and 49.7 for high schools. Pairwise

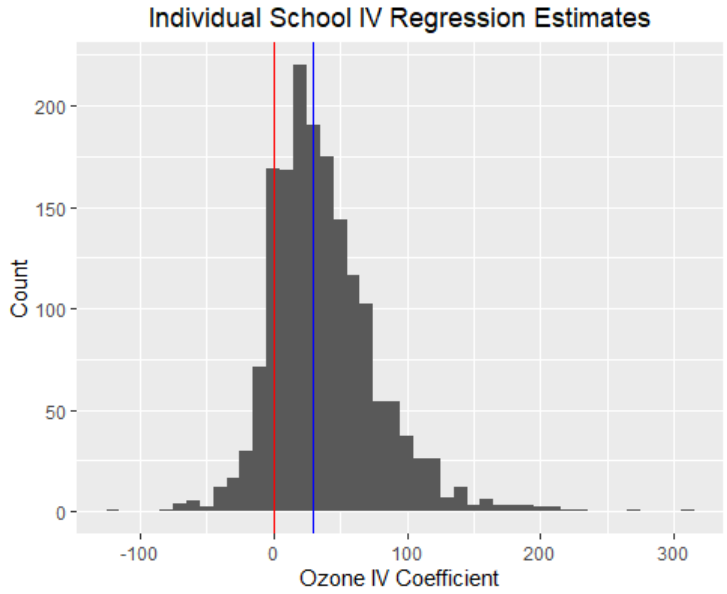


Figure 3.6: Ozone IV regression with every school run individually. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school. The red line is at 0 and the blue line represents the coefficient in the full data.

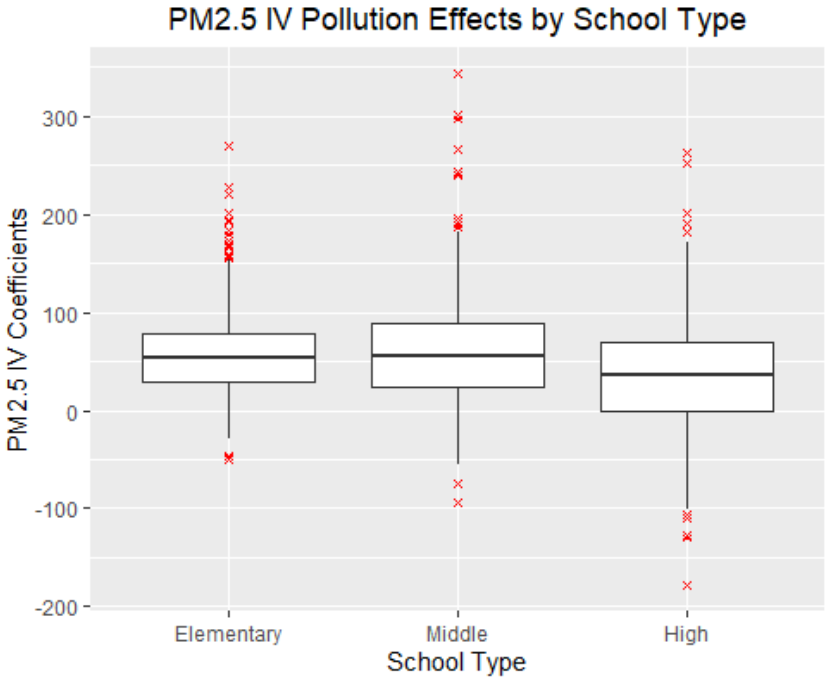


Figure 3.7: PM2.5 IV regression with every school run individually and grouped into school type. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

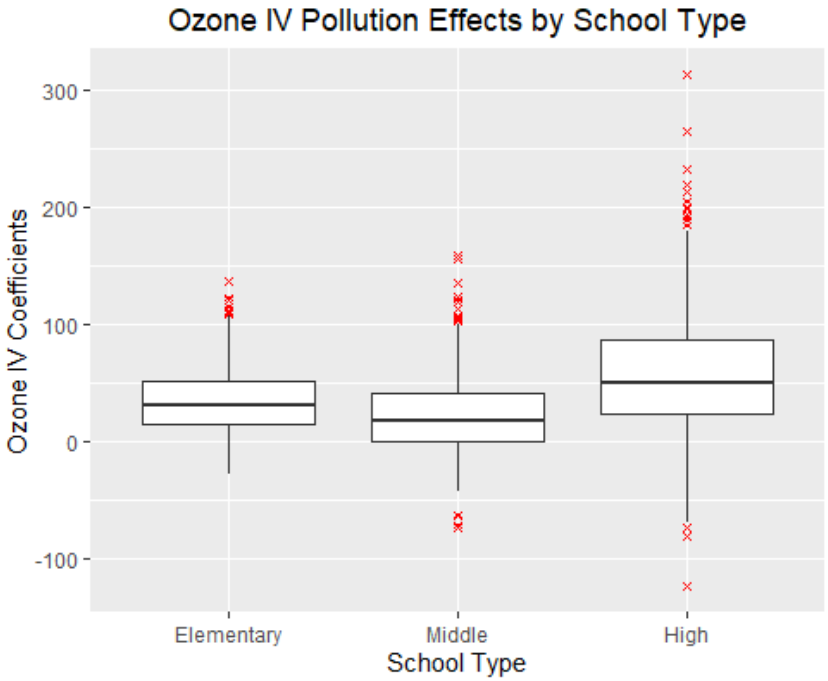


Figure 3.8: Ozone IV regression with every school run individually and grouped into school type. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

differences between school types are all statistically significant, with high schools having statistically larger effects than elementary schools which have larger effects than on middle schools. Most of the medical literature suggests that younger children are generally more at risk because of their developing immune systems, so this large Ozone effect on high school absences (and small effect on middle school absences) is surprising. I conjecture that this difference might be due to after-school sports in high schools, which would have them exercising outdoors during peak Ozone hours. Previous analysis of school absences often focused only on elementary or middle school, so these Ozone-caused absences among high school students warrant further investigation.

Figure 3.9 presents a box plot of Ozone IV coefficients grouped by a school's percentage of students considered economically disadvantaged. Most of NYC public school students are classified as poor, with poverty quartiles by school of Q4 = [0, 76.07], Q3 = (76.07, 87.92], Q2 = (87.92, 93.08], and Q1 = (93.08, 100], with Q1 being the richest schools and Q4 being the poorest schools. The median coefficients by poverty quartile are 38.1 for Q1, 46.5 for Q2, 55.2 for Q3, and 68.9 for Q4. Average effect sizes are increasing in poverty quartile, with statistically significant

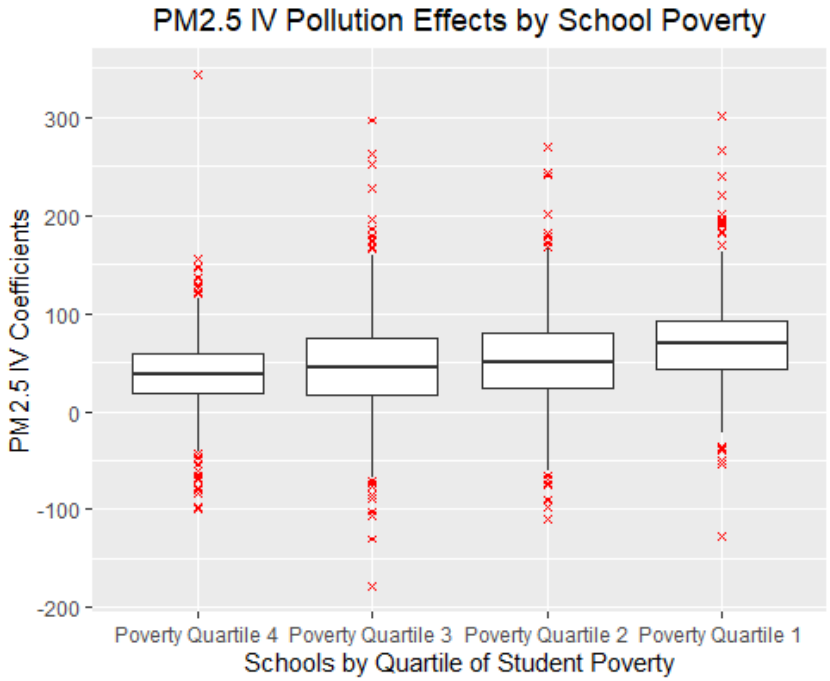


Figure 3.9: PM2.5 IV regression with every school run individually and grouped by percentage of students considered economically disadvantaged, where Quartile 4 is richest and Quartile 1 is poorest. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

increases from Q4 to Q3 (p = 0.016), from Q3 to Q2 (p = 0.021), and from Q2 to Q1 (p = 0.0001). Previous research has shown that socioeconomic status affects both the frequency and severity of asthma, and higher asthma rates at schools with more students in poverty could explain these findings. Alternatively, students from families that are more well-off may have smaller cumulative exposure from pollution or more ways to mitigate the harms of pollution through air filtration or increased healthcare spending.

Figure 3.10 presents a box plot of Ozone IV coefficients grouped by a school's percentage of students considered economically disadvantaged, using the same poverty quartile bins as in the Figure 3.9 with Q1 being the richest schools and Q4 being the poorest schools. The median coefficients by poverty quartile are 22.0 for Q1, 44.0 for Q2, 42.7 for Q3, and 45.6 for Q4. In contrast to PM2.5, average effect sizes are not increasing in poverty quartile, with statistically significant increases from Q4 to Q3 (p = 0.0000) but not from Q3 to Q2 (p = 0.652) or Q2 to Q1 (p = 0.248). This analysis suggests that socioeconomic status has less of an intermediary effect on absences caused by Ozone than on absences caused by PM2.5 pollution.

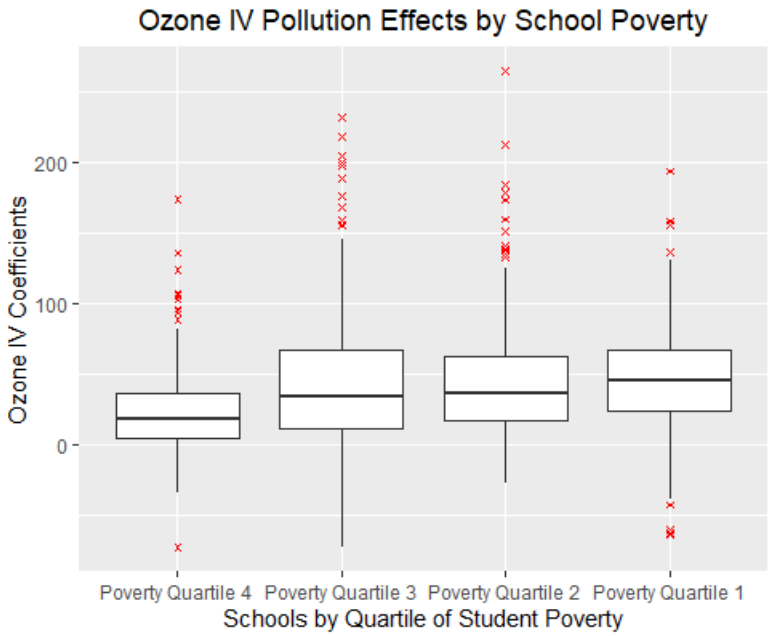


Figure 3.10: Ozone IV regression with every school run individually and grouped by percentage of students considered economically disadvantaged, where Quartile 4 is richest and Quartile 1 is poorest. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

## 3.6 Extensions and Robustness

This section contains extensions of the main IV regressions and robustness tests. The first subsection compares the effects of pollution on different days, which motivates the choice of same-day PM2.5 pollution and two-days lagged Ozone. The final subsection runs OLS regressions on all data (including winter months) and directly controlling for snow.

### Effects of Lagged Pollution

This section analyzes different pollution lag structures for Ozone and PM2.5 pollution. These two chemicals are measured differently (maximum 8-hour concentration for Ozone, average 24-hour concentration for PM2.5) and the process of Ozone formation is heavily influenced by heat, so Ozone is highest in the afternoon after school absences are determined by morning attendance. For this reason, we might expect different time-responses for absences caused by PM2.5 versus Ozone pollution. PM2.5 measurements are averaged over the entire day, so it also includes after-school hours, but ambient concentrations do not change as much throughout the day as Ozone concentrations do.

Table 3.6 runs the full fixed effects model of PM2.5 separately for each day of pollution. For the IV models, in the first stage each day of pollution is regressed on wind from that day and three additional days of lagged wind. In both the IV and OLS models days 0 through 2 all have positive and significant coefficients, but day 3 has a negative and/or insignificant coefficient. This regression is evidence of the timescale at which pollution causes respiratory issues that lead to absences, and it appears that the short-run increase in absences caused by PM2.5 subsides after 2 days. Average PM2.5 over the past week or month is likely to affect school absences, but my econometric design can only test for short-run changes in pollution. Effects are largest on the same-day that PM2.5 increases, but there are (smaller) statistically significant absence effects on the following day and two days later. Based on this analysis of the effects by day, I run the main regressions on same-day PM2.5 concentrations, but results are similar if I include all pollution from days 0 through 2 as explanatory variables.

Table 3.6: PM2.5 IV and OLS regression comparing each day of lagged pollution separately for months without snow (April through November) and including all fixed effects.

| | Dependent Variable: | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | | | | |
| | IV(1) | IV(2) | IV(3) | IV(4) | OLS(5) | OLS(6) | OLS(7) | OLS(8) |
| Same Day PM2.5 | 44.29*** | | | | 25.06*** | | | |
| | (1.45) | | | | (0.92) | | | |
| 1 Days Lag | | 32.38*** | | | | 21.20*** | | |
| | | (1.62) | | | | (0.90) | | |
| 2 Days Lag | | | 25.41*** | | | | 13.86*** | |
| | | | (1.68) | | | | (0.91) | |
| 3 Days Lag | | | | -18.2*** | | | | -0.938 |
| | | | | (1.60) | | | | (0.90) |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.600 | 0.600 | 0.600 | 0.600 | 0.618 | 0.618 | 0.618 | 0.618 |
| $R^2$ Adj. | 0.600 | 0.600 | 0.600 | 0.599 | | | | |

Table 3.7 runs the full fixed effects model of Ozone separately for each day of pollution. For the IV models, in the first stage each day of pollution is regressed on wind from that day and three additional days of lagged wind. In both the IV and OLS models days 0 through 2 all have positive and significant coefficients, but day 3 has a insignificant IV coefficient and smaller OLS coefficient. Same-day Ozone pollution also has much smaller effects than lagged Ozone; the same-day coefficient magnitude is three times smaller compared to two-days IV lagged coefficient and twelve times

smaller than the comparable OLS coefficient. This regression is evidence of the timescale at which Ozone causes minor respiratory issues that lead to absences, and it appears that the short-run increase in absences caused is strongest one or two days later, but then this increase subsides. As with PM2.5, average Ozone concentration over the past week or month is likely to affect school absences, but my econometric design can only test short-run changes in pollution. Based on this analysis of the effects by day, I run the main regressions on two-days lagged Ozone concentrations, but results are similar if I include all pollution from days 0 through 2 as explanatory variables.

Table 3.7: Ozone IV and OLS regression comparing each day of lagged pollution separately for months without snow (April through November) and including all fixed effects.

| | Dependent Variable: | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | | | | |
| | IV(1) | IV(2) | IV(3) | IV(4) | OLS(5) | OLS(6) | OLS(7) | OLS(8) |
| Same Day Ozone | 9.37*** | | | | 16.7*** | | | |
| | (0.85) | | | | (0.40) | | | |
| 1 Day Lag | | 26.11*** | | | | 19.80*** | | |
| | | (0.91) | | | | (0.39) | | |
| 2 Days Lag | | | 29.83*** | | | | 20.91*** | |
| | | | (1.03) | | | | (0.42) | |
| 3 Days Lag | | | | -1.57 | | | | 11.65*** |
| | | | | (0.99) | | | | (0.42) |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.600 | 0.601 | 0.601 | 0.600 | 0.618 | 0.619 | 0.619 | 0.618 |
| $R^2$ Adj. | 0.599 | 0.600 | 0.600 | 0.599 | | | | |

**OLS Results Including Snow**

This subsection runs OLS regressions on the full school absence data set and directly controls for snow. Snow is included as binary indicators for {Any snow at $t$ or $t-1$, between 0.1 and 0.5 meters of snow from $t$ to $t-3$, More than 0.5 meters of snow from $t$ to $t-3$}. Table 3.8 shows the panel of fixed effects and coefficients on the snow variables. The PM2.5 OLS coefficients in Model 1 and 2 are similar to the corresponding coefficients in the main results, but adding school times year and school times month fixed effects reduces the coefficient and makes it negative in Model 5. The issue is that snow is measured with error and has much larger effects on absences than pollution. Using coefficients from Model 5, any snow that day or the day before causes 1862 absences per 100,000 students, which is added to either 2539 or 6322 absences if there is more than 0.1 or 0.5 meters of accumulated snow. A day of heavy snowfall can outweigh the average absence effects of a month

of high-pollution, so month and year fixed effects end up fitting average snowfall instead of average pollution. This is an econometric and measurement issue, though, and pollution still causes absences during winter months.

Table 3.9 shows the Ozone OLS regression using all months of data and directly controlling for snow. In contrast to PM2.5, the Ozone results are remarkably similar to the main Ozone OLS results run on months without snow. Specifically, all specifications for Ozone with snow are positive and significant and the Model 5 coefficient is only twenty percent larger in the comparable regression of non-winter months. Ozone formation depends on heat and is lowest during winter months, so it is not too surprising that this regression is able to distinguish snow absences from Ozone-caused absences.

Table 3.8: OLS regression of same-day PM2.5 pollution on daily absences for all months and directly controlling for snow.

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_1$ - $\mu g/m^3$ PM2.5 | 43.25*** | 17.51*** | 1.68** | 2.72*** | -16.30*** |
| | (0.96) | (0.64) | (0.64) | (0.66) | (0.67) |
| >0 Snow | 1733*** | 1895*** | 1887*** | 1899*** | 1862*** |
| | (21.1) | (15.8) | (15.7) | (15.6) | (15.5) |
| 10-50cm of Snow | 2181*** | 2310*** | 2398*** | 2447*** | 2539*** |
| | (38.2) | (32.0) | (32.0) | (32.1) | (32.0) |
| >= 50cm of Snow | 5097*** | 6106*** | 5962*** | 6437*** | 6322*** |
| | (242) | (221) | (220) | (222) | (221) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 3499057 | 3499057 | 3499057 | 3499057 | 3499057 |
| $R^2$ | 0.007 | 0.554 | 0.604 | 0.555 | 0.605 |

Table 3.9: OLS regression of two-days lagged Ozone pollution on daily absences for all months and directly controlling for snow.

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_2$ - PPB of Ozone | 32.20*** | 49.03*** | 12.48*** | 52.34*** | 17.30*** |
| | (0.40) | (0.29) | (0.34) | (0.29) | (0.34) |
| >0 Snow | 1973*** | 2205*** | 1904*** | 2220*** | 1894*** |
| | (21.1) | (15.6) | (15.5) | (15.4) | (15.4) |
| 10-50cm of Snow | 2332*** | 2490*** | 2462*** | 2642*** | 2603*** |
| | (38.1) | (31.8) | (31.9) | (31.9) | (32.0) |
| >= 50cm of Snow | 5240*** | 6203*** | 6091*** | 6544*** | 6402*** |
| | (241) | (220) | (220) | (220) | (221) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 3490118 | 3490118 | 3490118 | 3490118 | 3490118 |
| $R^2$ | 0.024 | 0.532 | 0.580 | 0.536 | 0.584 |
| $R^2$ Adj. | 0.024 | 0.531 | 0.580 | 0.535 | 0.583 |

## 3.7 Conclusion

This paper estimates the causal effects of air pollution encountered in day-to-day life in the United States. Previous papers analyzing the absences in the US took place either at smaller scale or using less fine-grained data. I analyze 1669 schools in NYC from 2006 to 2019 and using daily attendance data from millions of students find absences caused by PM2.5 as well as Ozone. Because I can analyze responses to wind-induced pollution shocks across different days, I show that PM2.5 pollution causes absences the same day it increases but Ozone has largest effects on absences one or two days later. I separately analyze schools by type and find, consistent with the literature, that elementary and middle schools experience larger effects from PM2.5 pollution than do high schools. For Ozone, by contrast, the largest absence effect is on high schools, followed by elementary and then middle schools.

Although the magnitudes of the estimated absences caused by PM2.5 and Ozone are not especially large, they are robust to multiple specifications, are estimated for all NYC students, and can be interpreted as causal. This means that, across NYC with an average yearly enrollment of 960,000, an additional $\mu g/m^3$ of PM2.5 pollution causes 425 students to stay home sick that day and an extra PPB of Ozone causes 286 students to be sick from school two days later. Going from the median PM2.5 day to the 90th percentile day causes 2150 students to be absent that day and going from

the median Ozone day to the 90th percentile day causes 5654 students to be absent two days later. The decline in average daily PM2.5 pollution of 5 $\mu g/m^3$ from 2006 to 2019 caused a reduction in school absences of 2125 students per day, or 381024 total absences over the entire school year. The state of New York funds individual schools on the basis of average daily attendance, with a school losing around $50 for every day that a student is absent, so this reduction in school absences increased annual NYC school funding by approximately $19 million dollars in 2019. My identification strategy compares school days in the same month to minimize bias from seasonal trends, but if some of the difference in absences between months is due to changes in pollution across months, then these are likely to be lower bounds. For example, if school absences are high in summer months not because of end-of-year reasons but only because Ozone concentrations are much higher, then including School $\times$ Month fixed effects biases the results towards zero. Assuming that all differences in absences across months are due to pollution corresponds to Model 4 in my results, and the Ozone coefficients are approximately 3 times larger than the corresponding Model 5 coefficients.

These pollutants were measured in NYC from 2006 to 2019 with concentrations below the respective federal limits on approximately 99.3% of days for PM2.5 and 98.3% of days for Ozone. The measured harms were larger for days above those thresholds, but there were absences caused by pollution below those thresholds as well. I was able to identify effects of pollution at these levels because of daily school absence data representing more than twelve million student years. Reporting absences by school means that the absence data is not sensitive and is publicly available in multiple settings. Student-level data allows for the tracking of students exposed to high pollution and the long-run effects, but that data is harder to access and typically can't be released for replication. In contrast, the NYC school-level data is available online and anyone can replicate this paper with different econometric specifications. Other variables of interest to connect to absences in future work include influenza rates, asthma rates, or seasonal allergies.

These absence results also have implications for the rest of the country. PM2.5 concentrations declined significantly over this period, but Ozone remained high, and these pollutant concentrations are common to the US and other countries in the modern era. A back-of-the-envelope calculation suggests that: counties containing more than 140 million people have higher yearly average PM2.5 pollution than NYC; counties containing more than 103 million people have higher 98th percentile daily

PM2.5 compared to NYC; counties containing more than 76 million people have higher Ozone concentrations.[14] While those other counties might have different distributions of polluted days, it suggests that schools in those counties would have similar numbers of absences due to pollution.

[14]Calculation done using the EPA's Air Quality Statistics by County data for 2021, Link Here, with 2010 Population. "Wtd AM" is weighted annual mean concentration, (24-hr) is the 98th percentile 24-hour PM2.5 concentration, and "O3" is the fourth daily maximum 8-hour concentration. NYC is calculated as the mean of New York County (Manhattan), Kings County (Brooklyn), Bronx County (The Bronx), Richmond County (Staten Island), and Queens County (Queens).

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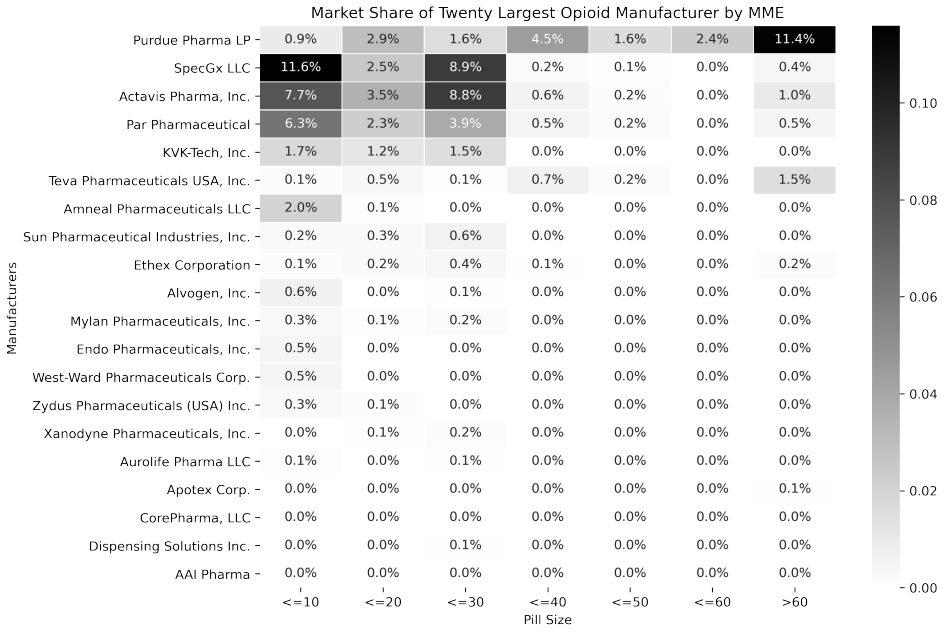
*Appendix A*

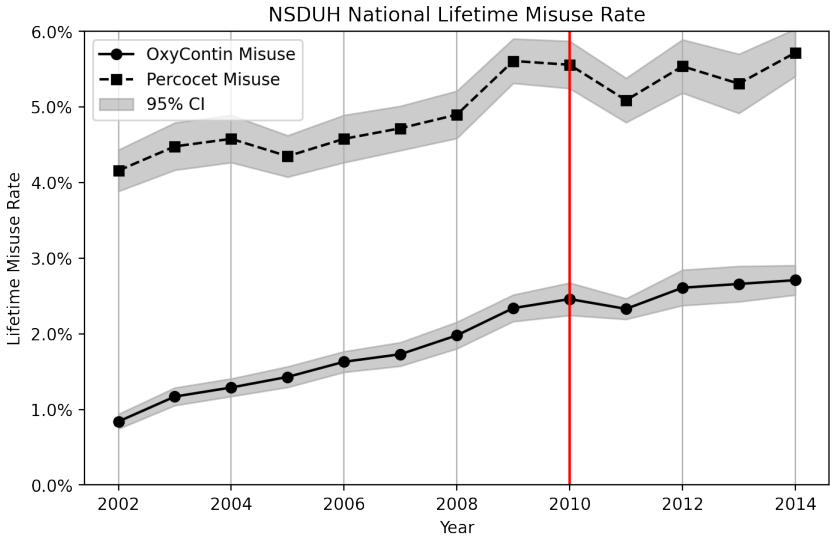
# CHAPTER 1 APPENDIX

## Additional Information



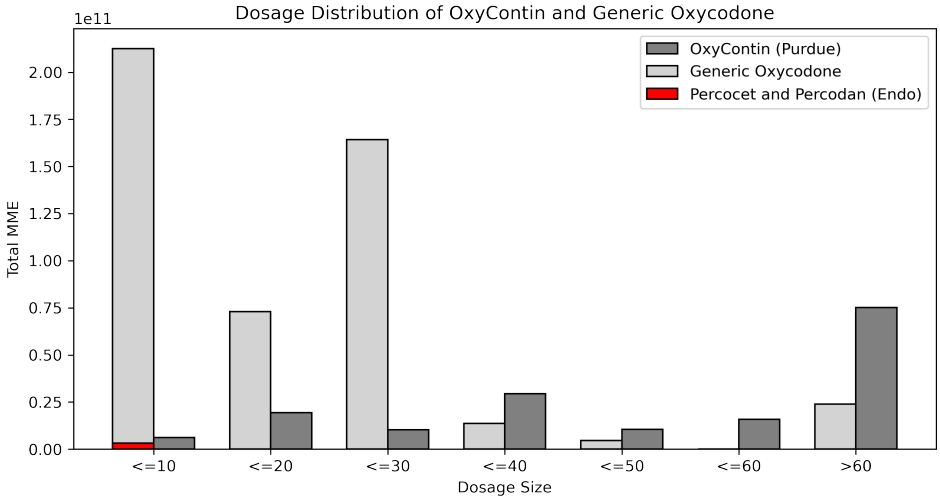

*Note:* We compute market share based on the average of 2006 to 2014 sales data. We kept only the top twenty manufacturers for better readability of the table. The rest of the 35 manufacturers combined contribute 0.18% of total sales. During this sample period, Purdue Pharma was the dominant manufacturer of high dosage oxycodone pills ($\geq$ 40mg). In the lower dosage market, three manufacturers (SpecGx, Actavis Pharma and Phar Phamaceutical) had higher share of the market than Purdue Pharma.

Figure A.1: Market share of different opioid manufacturers.



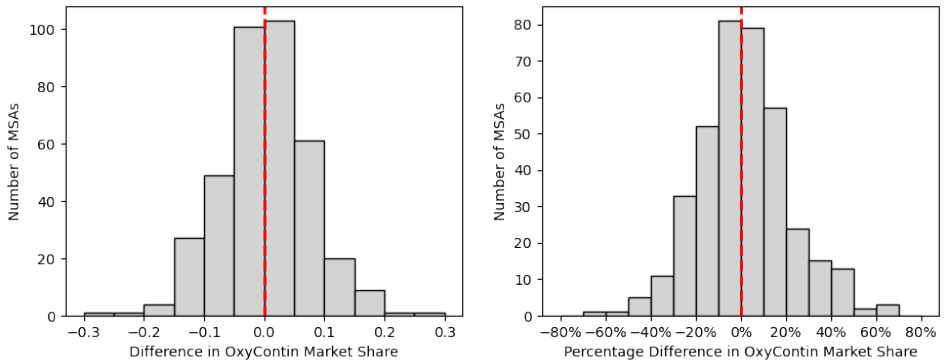

*Note:* The figure shows the misuse rate of OxyContin (OXYFLAG or OXYCONT2) and the misuse rate of Percocet, Percodan, and Tylox (PERCTYL2). Data obtained from annual NSDUH. Percocet was a popular prescription oxycodone to misuse in the pre-OxyContin period. We see in this graph that the PERCTYL2 misuse rate increased 30% from 2002 to 2009, suggesting that the lifetime misuse rate captures more than historical Percocet, Percodan, and Tylox misuse.

Figure A.2: NSDUH national lifetime misuse rate.



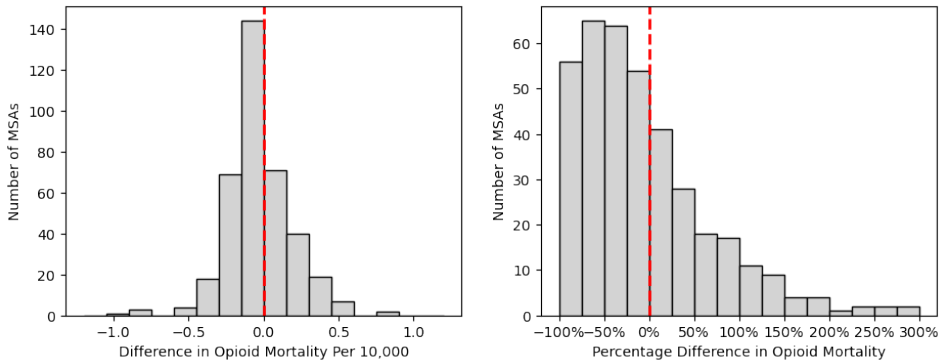

*Note:* This graph shows the difference in oxycodone sales between Purdue and Endo Pharma. The small market share of Endo Pharma leads us to believe that individuals misreport the drugs they consume on the NSDUH.

Figure A.3: Comparison of sales of Purdue and Endo Pharma.



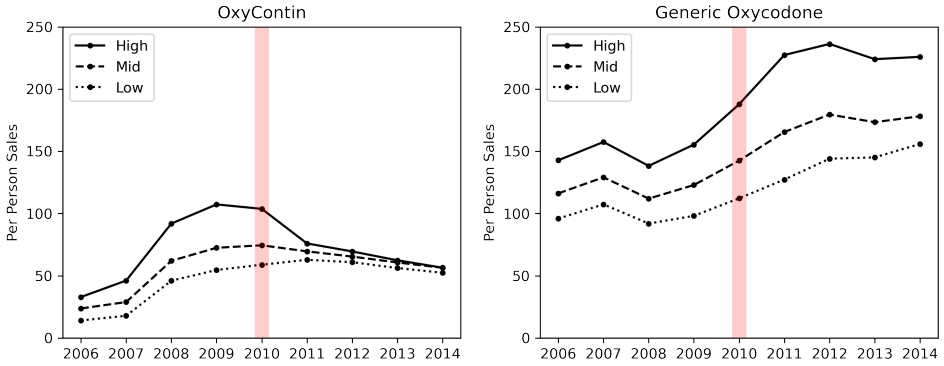

*Note:* Left is the absolute difference in market share (0.1 means that MSA share is 10% higher than the state average) and right is percentage difference (10% means that MSA share is 1.1 times the state average).

Figure A.4: Within-state variation in OxyContin market share.



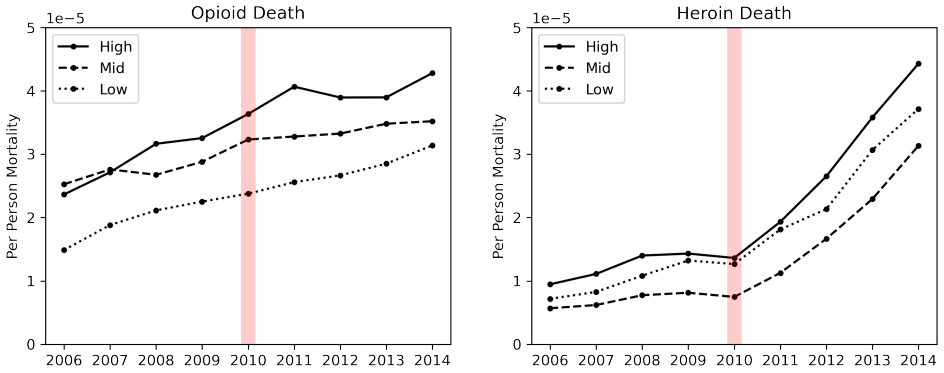

*Note:* Left is the absolute difference in opioid mortality (0.1 means that MSA mortality per 10,000 people is 0.1 higher than the state average) and right is percentage difference (10% means that MSA mortality per 10,000 people is 1.1 times the state average).

Figure A.5: Within-state variation in opioid mortality.




*Note:* We categorized all MSAs into high, mid, and low by the drop in the observed per person OxyContin sales from 2009 to 2011. The series are population weighted and Florida is excluded. The high group saw a 30% drop in OxyContin sales, mid group a 3.9% drop, and low group a 15% increase. The high group experienced a 46% increase in generic oxycodone sales, mid group a 34% increase, and low group a 29% increase. The three groups share similar oxycodone growth trends until the reformulation.

Figure A.6: Opioid sales by empirical OxyContin drop.




*Note:* Similarly to the previous figure, we categorized all MSAs into high, mid, and low by the drop in the observed per person OxyContin sales from 2009 to 2011. The series are population weighted and Florida is excluded. No trend break in opioid mortality in the high drop group. The high group saw an 35% increase in heroin mortality, the mid group 38%, and the low group 37%. The similar increases in heroin mortality post-reform indicates that drops in OxyContin use post-reform did not lead to additional increase in heroin use.

Figure A.7: Opioid mortality by empirical OxyContin drop.

Table A.1: Testing constructed exposure measure against opioid mortality.

| | Opioid overdose deaths per 100,000 | | | | | |
|---|---|---|---|---|---|---|
| | OxyContin | | | Generic Oxycodone | | |
| | (1) | (2) | (3) | (4) | (5) | (6) |
| NSDUH misuse | 10.235 | | | 2.909 | | |
| | (1.719) | | | (0.570) | | |
| ARCOS sales | | 0.001 | | | 0.001 | |
| | | (0.0002) | | | (0.0001) | |
| Combined exposure | | | 0.093 | | | 0.087 |
| | | | (0.012) | | | (0.009) |
| Number of observations | 379 | 379 | 379 | 379 | 379 | 379 |
| R-square | 0.086 | 0.089 | 0.130 | 0.065 | 0.178 | 0.189 |
| Adjusted R-square | 0.084 | 0.086 | 0.128 | 0.062 | 0.176 | 0.187 |

*Notes:* Standard errors are in parentheses. We report coefficients from OLS regressions of opioid mortality on misuse, sales or exposure. NSDUH misuse rates is the 6-year average OxyContin or Percocet lifetime misuse rate from pre-reform period (2004-2009). ACROS sales is Oxycontin or generic oxycodone sales per person from 2009. Combined exposure is the product of the previous two measures normalized (see Equation 1). Overdose from 2009. Regressions are weighted by MSA population.

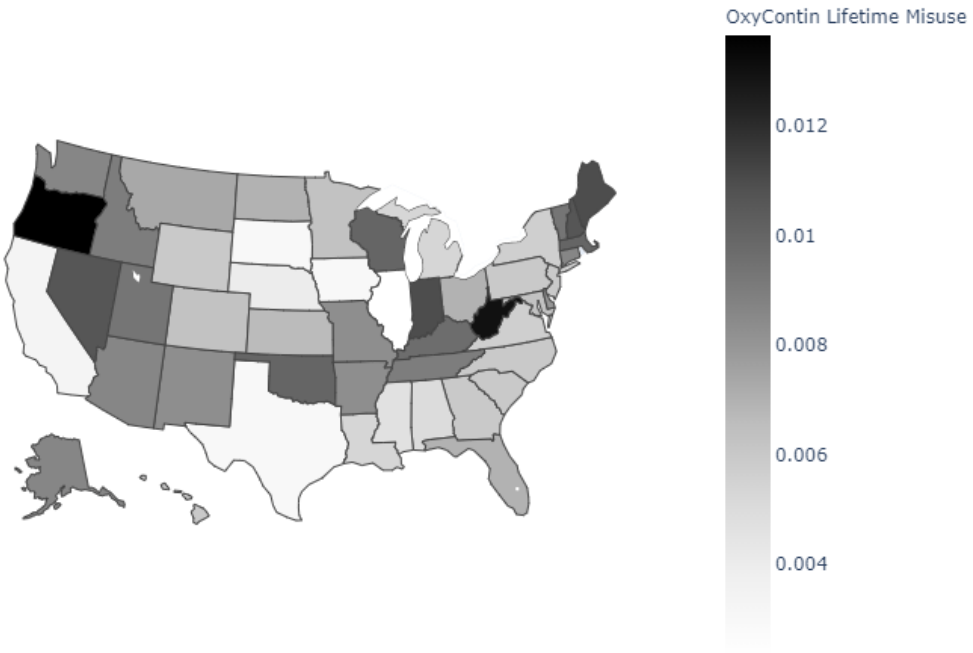
Table A.2: Difference in difference regression results.

| | Opioid sales per person | | Overdose per 10,000 | |
|---|---|---|---|---|
| | OxyContin | Oxycodone | Opioid | Heroin |
| | (1) | (2) | (3) | (4) |
| Post | -8.05 | 41.74 | 0.01 | 0.14 |
| | (2.86) | (4.92) | (0.02) | (0.02) |
| High OxyContin | 47.24 | 56.46 | -0.05 | -0.07 |
| | (5.78) | (13.36) | (0.03) | (0.02) |
| High Oxycodone | 26.84 | 95.90 | 0.14 | 0.08 |
| | (6.66) | (15.47) | (0.04) | (0.05) |
| Post x High OxyContin | -15.14 | 10.30 | 0.02 | 0.03 |
| | (6.39) | (8.90) | (0.02) | (0.02) |
| Post x High Oxycodone | -2.33 | 33.99 | 0.06 | 0.07 |
| | (6.37) | (8.80) | (0.02) | (0.02) |
| Number of observations | 2148 | 2148 | 2148 | 2148 |
| R-square | 0.665 | 0.737 | 0.517 | 0.469 |
| Adjusted R-square | 0.654 | 0.728 | 0.501 | 0.452 |

*Notes:* We report coefficients from the difference-in-difference estimation (see Equation (3)). All MSAs in Florida are excluded. In all specifications, we include MSA-level control variables, state fixed effects and year fixed effects. Standard errors are clustered at the MSA level.

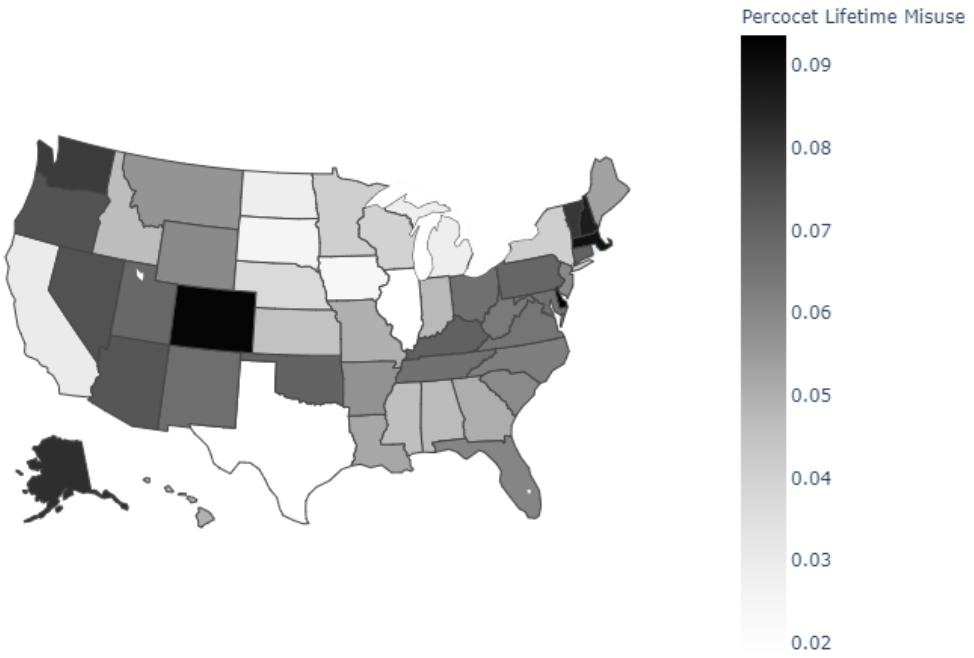
**Tables**

**Map**



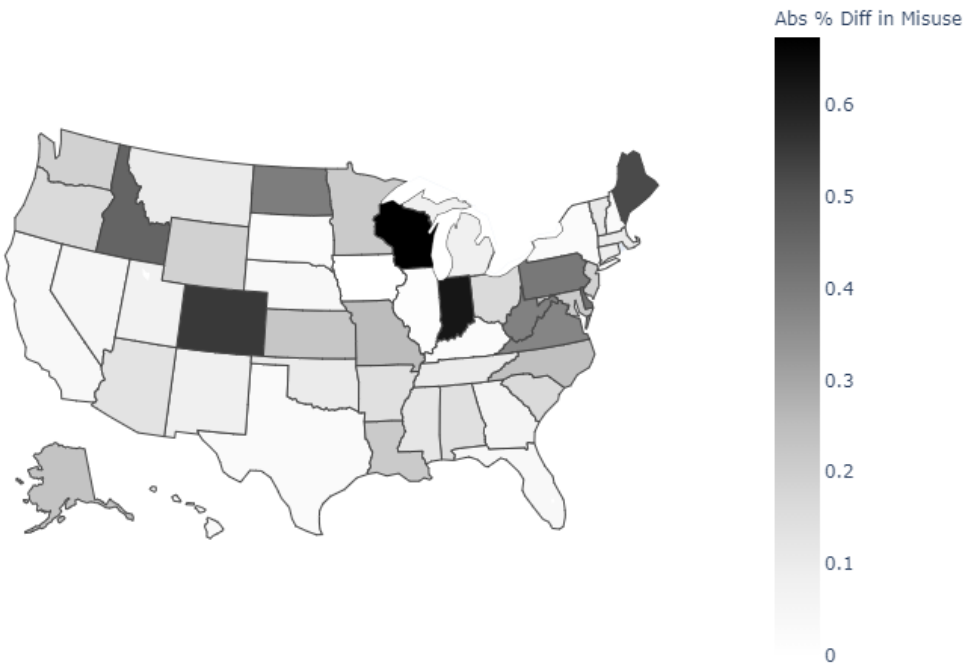

*Note:* Data from 2004-2009 NSDUH lifetime OxyContin misuse rate (NSDUH ticker OXXYR). 0.01 is interpreted as 1% of the state population have ever misused OxyContin.

Figure A.8: OxyContin lifetime misuse rate at state level.



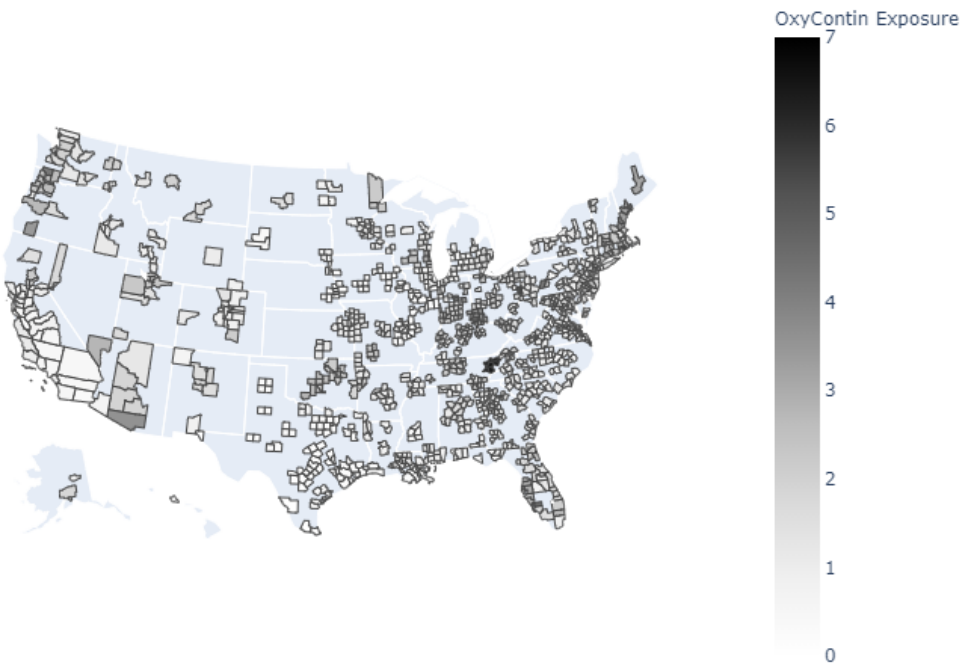

*Note:* Data from 2004-2009 NSDUH lifetime Percocet, Percodan, Tylox misuse rate (NSDUH ticker PERCTYL2). 0.01 is interpreted as 1% of the state population have ever misused one of the three drugs. Percocet lifetime misuse rate on average is much higher than OxyContin lifetime misuse rate.

Figure A.9: Percocet lifetime misuse rate at state level.



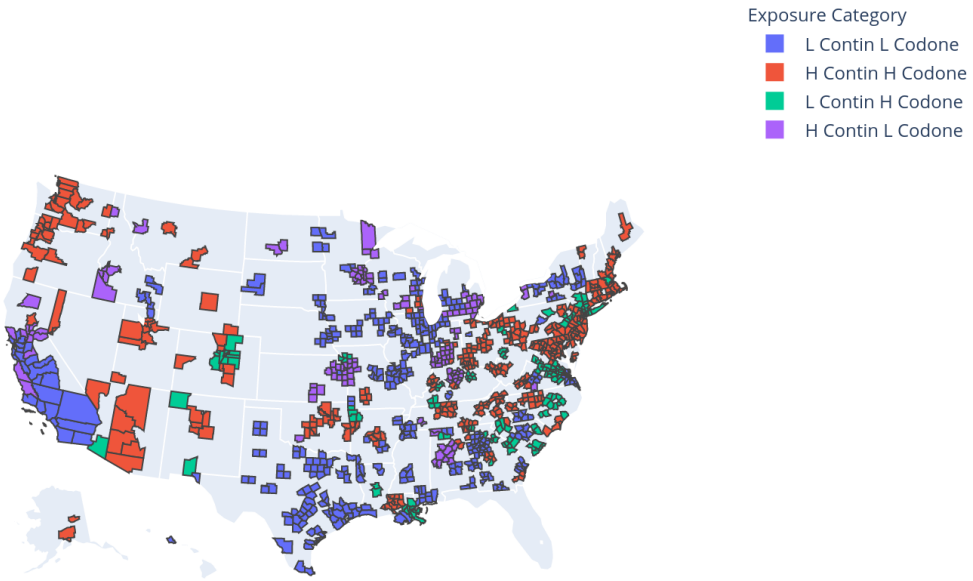

*Note:* The figure plots the absolute difference in percentile ranking of the two state level lifetime misuse rate. A 0.1 should be interpreted as a 10% difference in percentile ranking between OxyContin lifetime misuse rate and Percocet lifetime misuse rate. For example, Colorado's OxyContin misuse rate is 0.0063 (42 percentile) and it's Percocet misuse rate is 0.092 (97 percentile), which is a 55% difference in percentile ranking. We rely on the difference between two misuse rate to separately identify the impact of OxyContin and oxycodone.

Figure A.10: Difference in state level misuse rates.




*Note:* This figure shows OxyContin exposure by MSA. We show Florida here, which had very low OxyContin exposure/sales, but omit it from analysis because it had abnormally high generic oxycodone sales with large amounts being trafficked to other states.

Figure A.11: OxyContin exposure at MSA level.




*Note:* Florida is excluded in this analysis. MSAs grouped by high vs low OxyContin exposure and high vs low generic oxycodone exposure.

Figure A.12: Difference-in-difference regression categories.

## Alternative Regression Specifications

### MSA FE

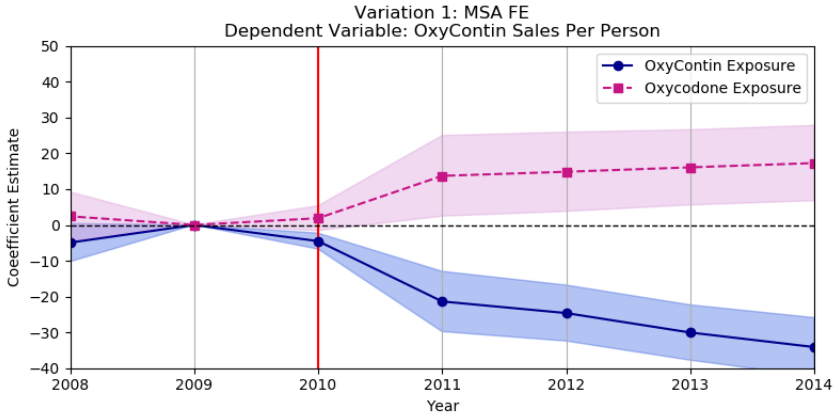



Figure A.13: Regression on OxyContin sales with MSA FE. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

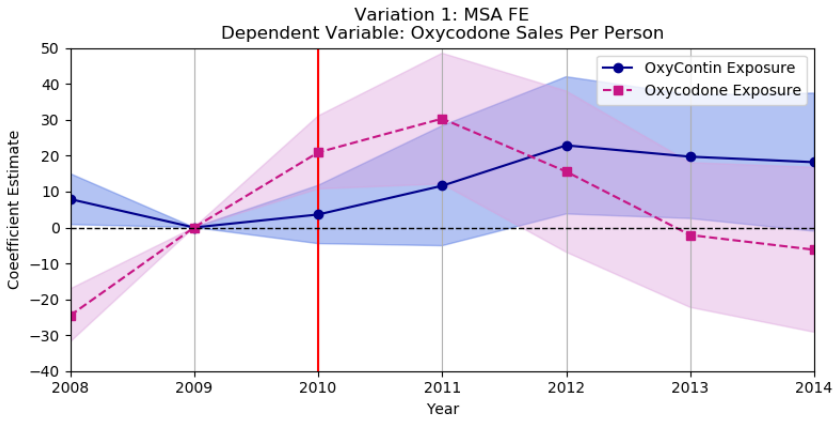



Figure A.14: Regression on oxycodone sales with MSA FE. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

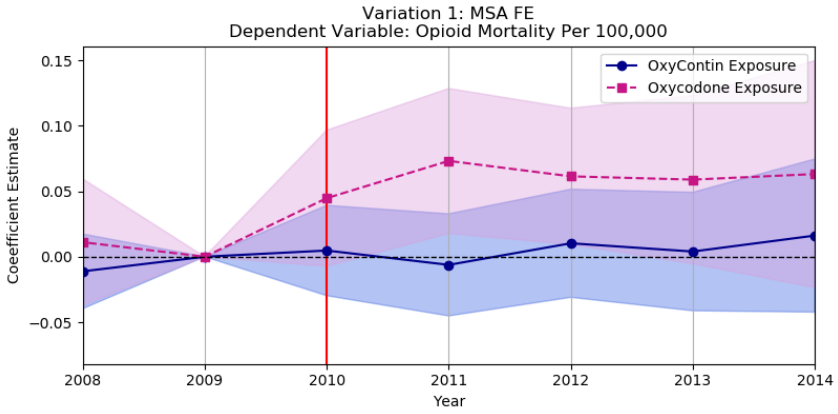



Figure A.15: Regression on opioid mortality with MSA FE. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

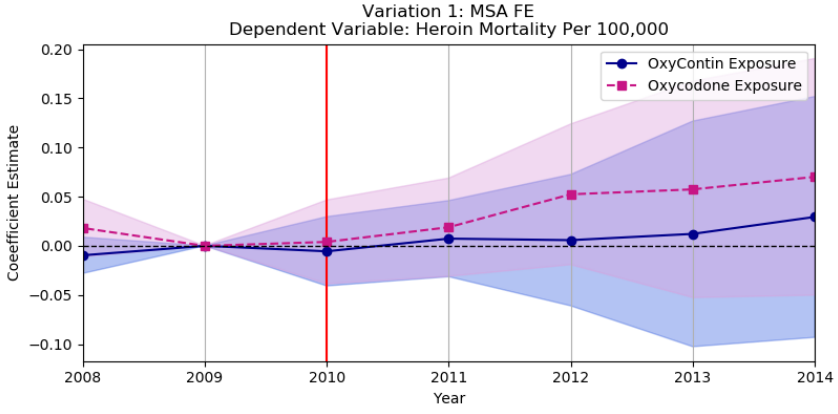



Figure A.16: Regression on heroin mortality with MSA FE. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

**Last Year OxyContin Misuse**

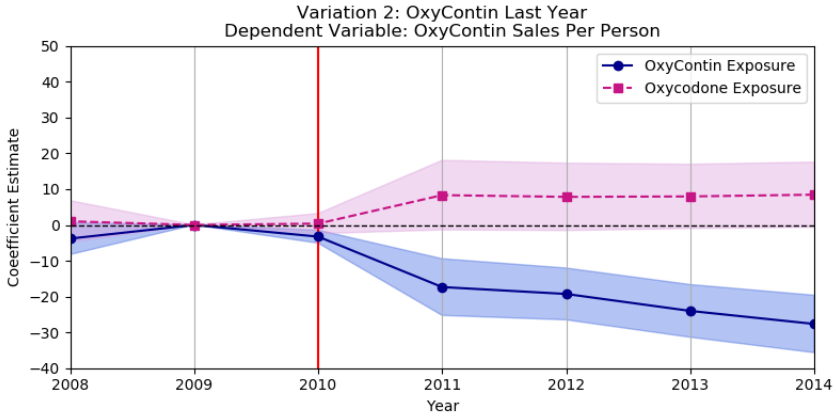



Figure A.17: Regression on OxyContin sales with last-year OxyContin. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

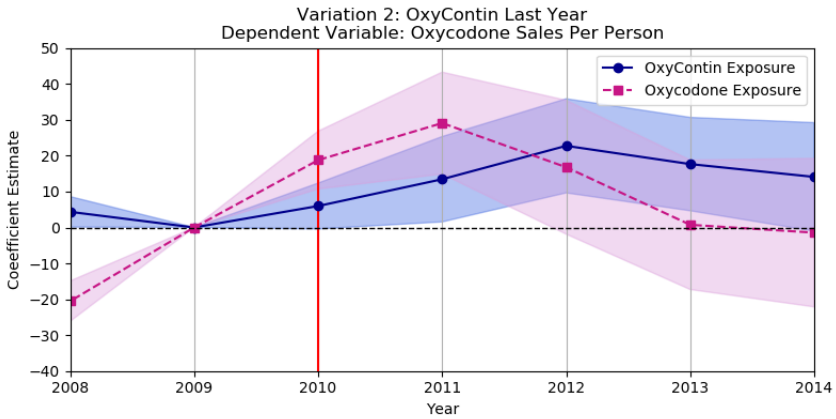



Figure A.18: Regression on oxycodone sales with last-year OxyContin. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

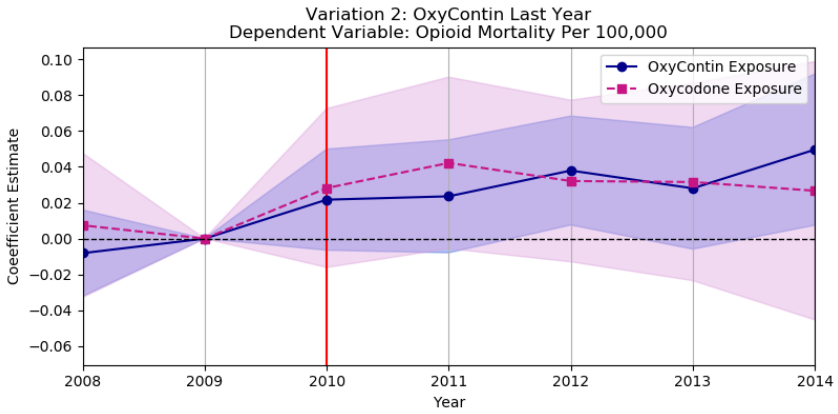



Figure A.19: Regression on opioid mortality with last-year OxyContin. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

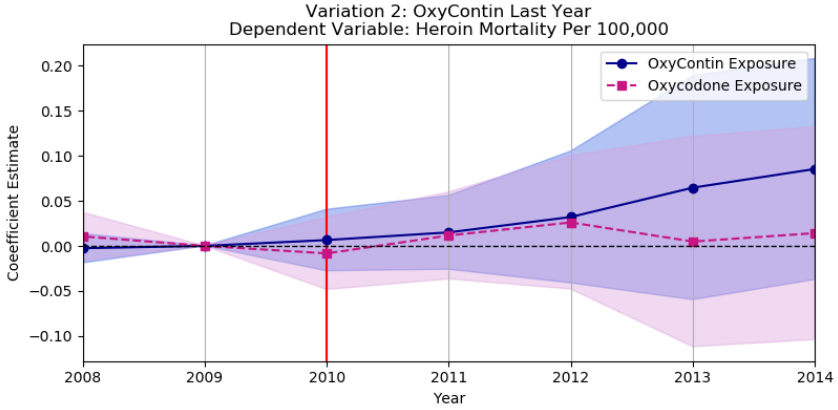



Figure A.20: Regression on heroin mortality with last-year OxyContin. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

**State Level Regression**

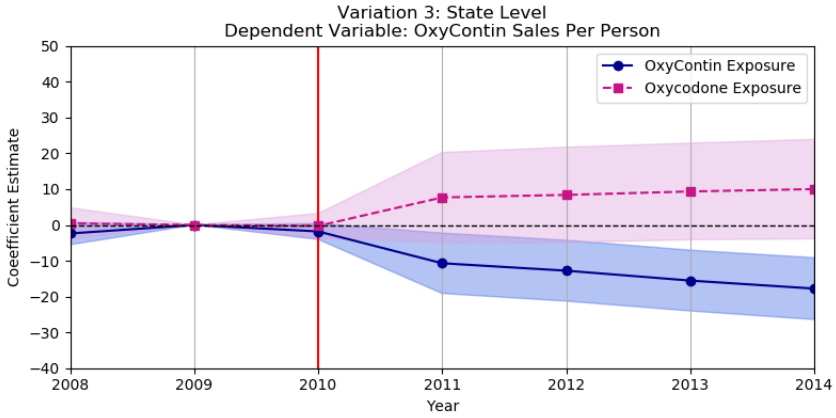



Figure A.21: Regression on OxyContin sales at state level. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

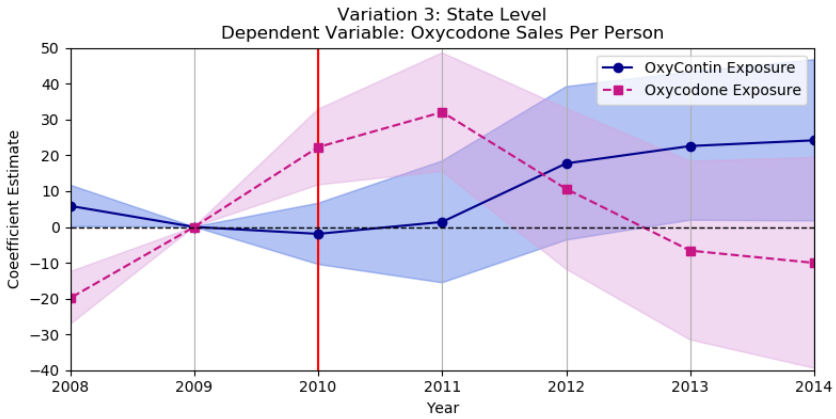



Figure A.22: Regression on oxycodone sales at state level. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

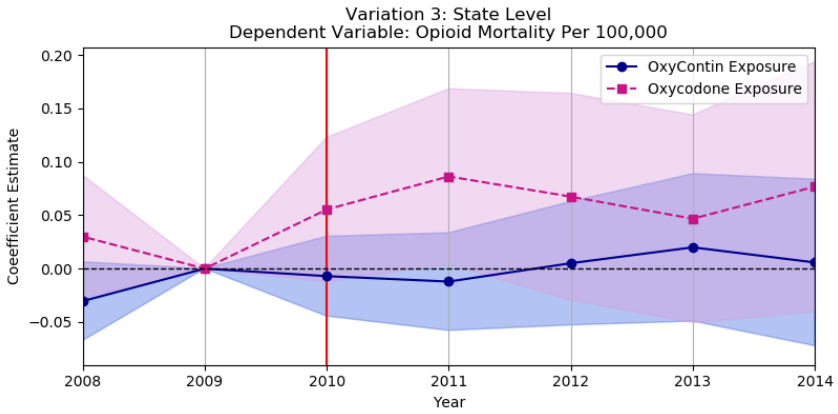



Figure A.23: Regression on opioid mortality at state level. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

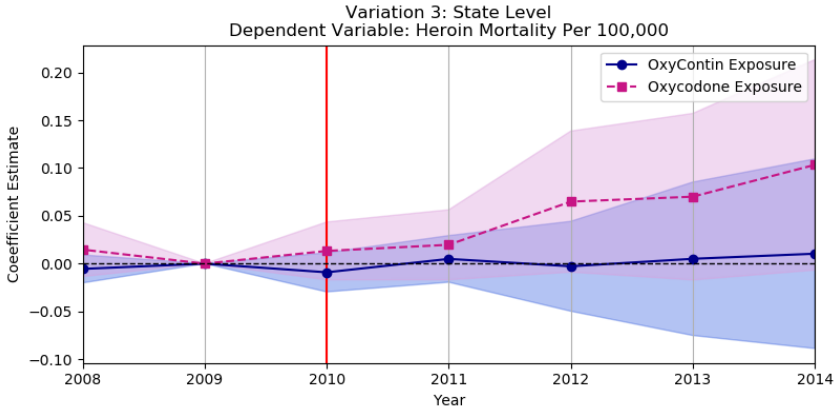



Figure A.24: Regression on heroin mortality at state level. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

**OxyContin Only**

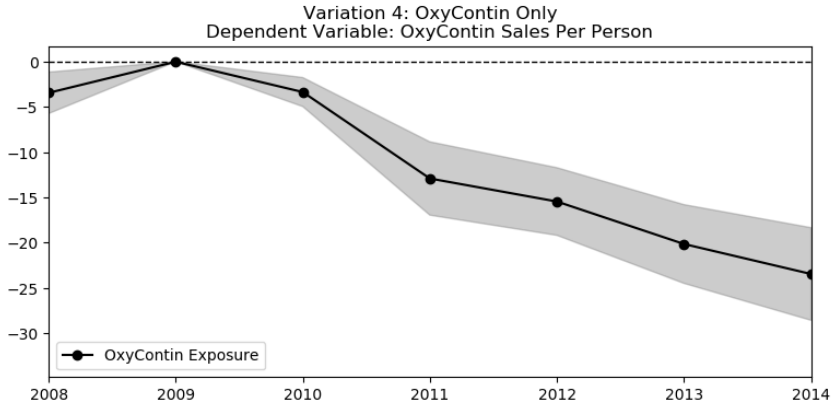



Figure A.25: Regression on OxyContin sales with OxyContin only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

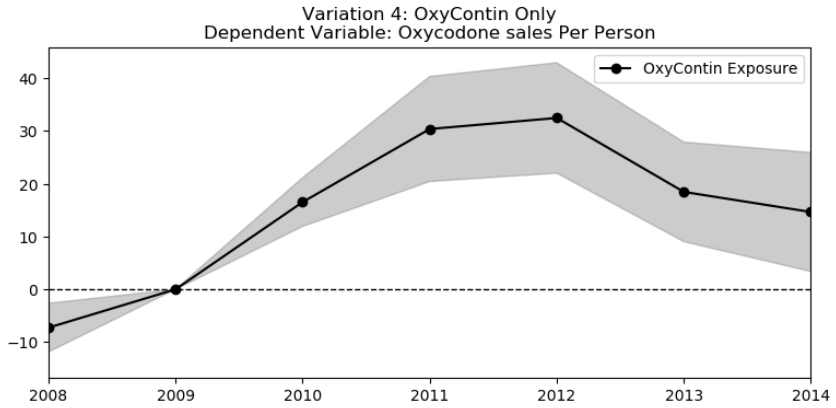



Figure A.26: Regression on oxycodone sales with OxyContin only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

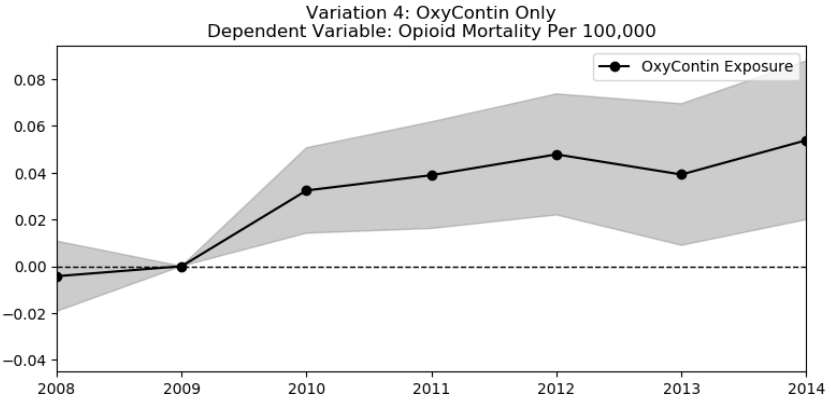



Figure A.27: Regression on opioid mortality with OxyContin only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

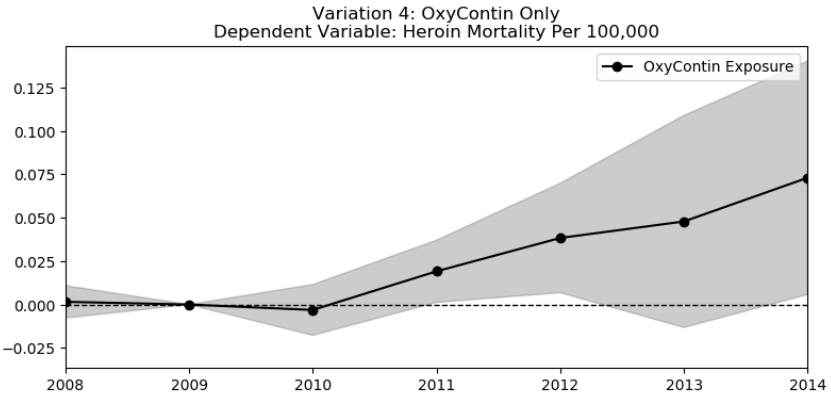



Figure A.28: Regression on heroin mortality with OxyContin only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

**Oxycodone Only**

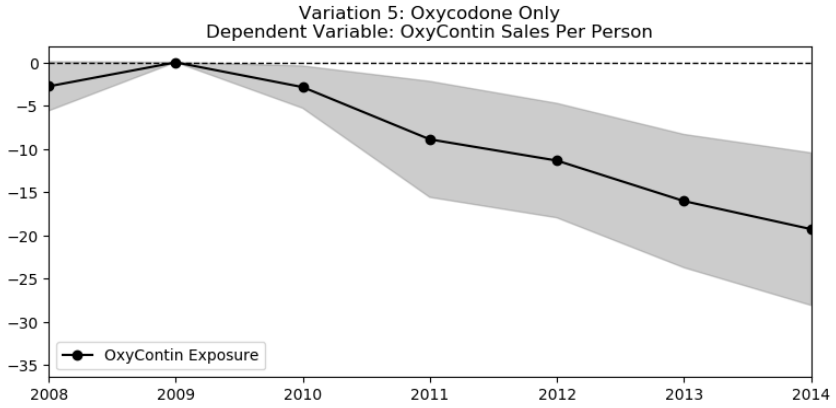



Figure A.29: Regression on OxyContin sales with oxycodone only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

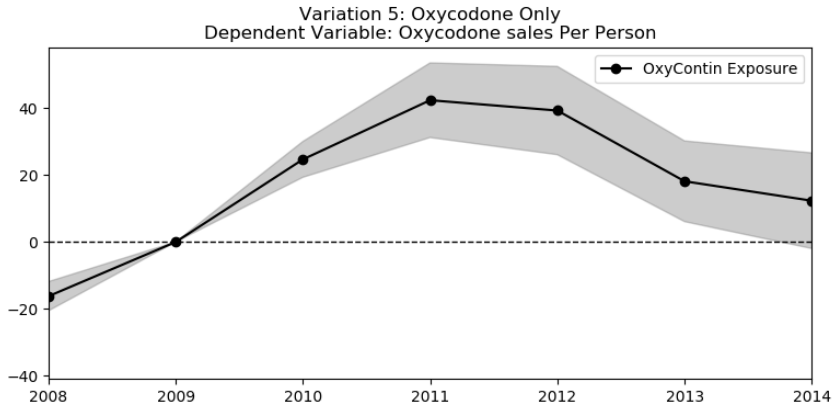



Figure A.30: Regression on oxycodone sales with oxycodone only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

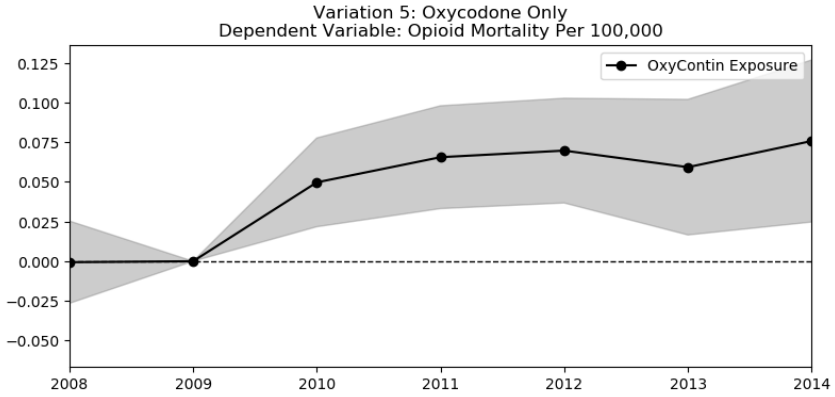



Figure A.31: Regression on opioid mortality with oxycodone only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

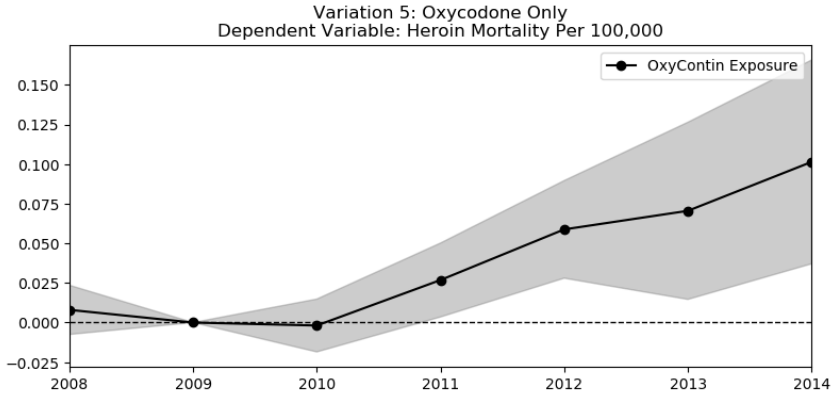



Figure A.32: Regression on heroin mortality with oxycodone only. Shaded regions are the 95 percent confidence intervals with standard errors clustered at the MSA level.

*Appendix B*

# CHAPTER 2 APPENDIX

**Additional Tables**

| State | PDMP | ePDMP (Horwitz) | ePDMP (PDAPS) | MA-PDMP |
|---|---|---|---|---|
| Alabama | 2005-11-01 | 2006-04-01 | 2007-06-28 | |
| Alaska | 2008-09-01 | 2012-01-01 | 2012-01-01 | |
| Arizona | 2007-09-01 | 2008-12-01 | 2008-12-01 | |
| Arkansas | 2013-03-01 | 2013-05-01 | 2013-05-16 | |
| California | 1939-01-01 | 2009-09-01 | 2009-09-01 | |
| Colorado | 2005-06-01 | 2008-02-01 | 2008-02-04 | |
| Connecticut | 2006-10-01 | 2008-07-01 | | 2015-10-01 |
| Delaware | 2011-09-01 | 2012-08-01 | 2012-08-21 | 2012-03-01 |
| District of Columbia | 2014-02-01 | 2016-10-01 | | |
| Florida | 2010-12-01 | 2011-10-01 | 2011-10-17 | |
| Georgia | 2011-07-01 | 2013-05-01 | 2013-07-01 | 2014-07-01 |
| Hawaii | 1943-01-01 | 2012-02-01 | 1997-01-01 | |
| Idaho | 1967-01-01 | 2008-04-01 | 1999-06-01 | |
| Illinois | 1961-01-01 | 2009-12-01 | | |
| Indiana | 1997-01-01 | 2007-07-01 | 2004-12-29 | 2014-07-01 |
| Iowa | 2006-05-01 | 2009-03-01 | 2009-03-19 | |
| Kansas | 2008-07-01 | 2011-04-01 | 2011-04-01 | |
| Kentucky | 1998-07-01 | 1999-07-01 | 1999-07-01 | 2012-07-01 |
| Louisiana | 2006-07-01 | 2009-01-01 | 2009-01-01 | 2008-01-01 |
| Maine | 2004-01-01 | 2005-01-01 | 2005-01-01 | |
| Maryland | 2011-10-01 | 2013-12-01 | 2013-12-20 | |
| Massachusetts | 1992-12-01 | 2011-01-01 | 2011-01-01 | 2014-07-01 |
| Michigan | 1988-01-01 | 2003-01-01 | 2003-01-01 | |
| Minnesota | 2009-01-01 | 2010-04-01 | 2010-04-15 | |
| Mississippi | 2006-06-01 | 2008-07-01 | 2005-12-01 | |
| Missouri | | | | |
| Montana | 2011-07-01 | 2012-10-01 | 2012-11-01 | |

| | | | | |
|---|---|---|---|---|
| Nebraska | 2011-08-01 | 2017-01-01 | 2011-04-14 | |
| Nevada | 1996-01-01 | 2011-02-01 | 1997-07-01 | 2007-10-01 |
| New Hampshire | 2012-06-01 | 2014-10-01 | 2014-10-16 | 2016-01-01 |
| New Jersey | 2009-08-01 | 2012-01-01 | 2012-01-05 | 2015-11-01 |
| New Mexico | 2004-07-01 | 2005-08-01 | 2005-08-01 | 2012-09-01 |
| New York | 1972-01-01 | 2013-06-01 | | 2013-08-01 |
| North Carolina | 2006-01-01 | 2007-07-01 | 2007-10-01 | |
| North Dakota | 2006-12-01 | 2008-10-01 | 2007-09-01 | |
| Ohio | 2005-05-01 | 2006-10-01 | 2006-10-02 | 2012-03-01 |
| Oklahoma | 1991-01-01 | 2006-07-01 | 2006-07-01 | 2011-03-01 |
| Oregon | 2009-07-01 | 2011-09-01 | 2011-09-01 | |
| Pennsylvania | 1972-01-01 | 2016-08-01 | | |
| Rhode Island | 1978-01-01 | 2012-09-01 | 2012-07-01 | 2016-06-01 |
| South Carolina | 2006-06-01 | 2008-02-01 | 2008-09-01 | |
| South Dakota | 2010-03-01 | 2012-03-01 | 2012-03-01 | |
| Tennessee | 2003-01-01 | 2010-01-01 | 2007-01-01 | 2013-07-01 |
| Texas | 1981-09-01 | 2012-08-01 | 2012-06-30 | |
| Utah | 1995-07-01 | 2006-01-01 | 1997-01-01 | |
| Vermont | 2008-06-01 | 2009-01-01 | 2009-04-01 | 2015-05-01 |
| Virginia | 2003-09-01 | 2006-01-01 | 2006-06-01 | 2015-07-01 |
| Washington | 2011-08-01 | 2012-01-01 | 2012-01-04 | |
| West Virginia | 1995-06-01 | 2013-05-01 | 2004-12-01 | 2012-06-01 |
| Wisconsin | 2010-06-01 | 2013-06-01 | 2013-06-01 | |
| Wyoming | 2003-07-01 | 2013-07-01 | 2004-10-01 | |

*Notes:* Date in the first column is the enactment/legislated start date for any PDMP from Horwitz et al. (2018). Date in the second column is the modern system operational date from Horwitz et al. (2018). Date in the third column is the electronic access dates from PDAPS. Date in the forth column is the must-access PDMP date from Sacks et al. (2021).

Table B.1: Impact of ePDMP laws on opioid sales using PDAPS dates

| | Dependent variable: | | | | |
|---|---|---|---|---|---|
| | Sales per person | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_1$ - PDMP law | -0.010*** | -0.011*** | -0.008** | -0.008** | -0.008** |
| | (0.001) | (0.002) | (0.002) | (0.002) | (0.002) |
| $\beta_2$ - Border county | | | 0.006*** | 0.005*** | 0.003* |
| | | | (0.002) | (0.001) | (0.002) |
| $\beta_3$ - Law x border | | | -0.009*** | -0.008*** | -0.009*** |
| | | | (0.002) | (0.002) | (0.003) |
| $\beta_4$ - Nearby law x border | | | | | 0.003* |
| | | | | | (0.002) |
| County FE | Yes | | | | |
| State FE | | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Controls | | | | Yes | Yes |
| Observations | 26,154 | 26,154 | 26,154 | 26,154 | 26,154 |
| $R^2$ | 0.005 | 0.459 | 0.459 | 0.520 | 0.520 |

*Notes:* We run the same regressions as Table 2.2 using alternative ePMDP dates. The results are very similar to our main findings: ePDMP reduces sales, but the reduction is less once we control for border counties; border counties have higher level of sales and they experience sharper decline when ePDMP is enacted; enactment of ePDMP in nearby states increases sales in border counties of the local state.

Table B.2: Impact of ePDMP laws on opioid mortality using PDAPS dates.

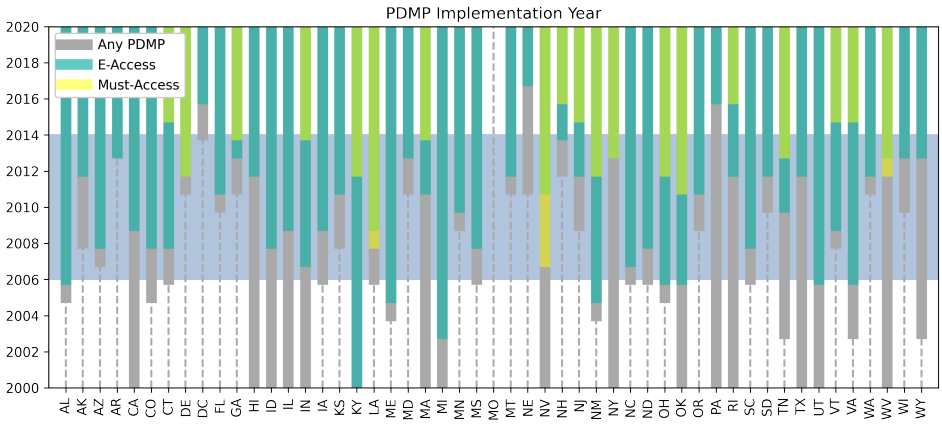
| | Dependent variable: | | | | |
|---|---|---|---|---|---|
| | Mortality per 100,000 residents | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_1$ - PDMP law | -0.419*** | -0.391*** | -0.432*** | -0.457*** | -0.431*** |
| | (0.052) | (0.071) | (0.075) | (0.073) | (0.074) |
| $\beta_2$ - Border county | | | -0.488*** | -0.594*** | -0.682*** |
| | | | (0.062) | (0.062) | (0.070) |
| $\beta_3$ - Law x border | | | 0.146* | 0.234*** | 0.173* |
| | | | (0.088) | (0.086) | (0.089) |
| $\beta_4$ - Nearby law x border | | | | | 0.210*** |
| | | | | | (0.075) |
| County FE | Yes | | | | |
| State FE | | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Controls | | | | Yes | Yes |
| Observations | 26,154 | 26,154 | 26,154 | 26,154 | 26,154 |
| $R^2$ | 0.001 | 0.283 | 0.286 | 0.318 | 0.319 |

*Notes:* We run the same regressions as Table 2.3 using alternative ePDMP dates. Again, the results are almost identical to our main findings. Enactment of ePDMP laws reduces overdose. Border counties start with lower opioid mortality but experience almost no drop when the state enacts ePDMP. Nearby enactment of ePDMP has a spillover effect on the mortality in the border counties of the local state.

Table B.3: Impact of ePDMP laws on opioid sales using any PDMP dates, e-access dates, and must-access dates.

| | Dependent variable: | | | | |
|---|---|---|---|---|---|
| | Sales per person | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| *(A) Any PDMP* | | | | | |
| $\beta_1$ - PDMP law | 0.006*** | 0.005*** | 0.005** | 0.004 | 0.003 |
| | (0.001) | (0.002) | (0.003) | (0.002) | (0.002) |
| $\beta_2$ - Border county | | | 0.002 | 0.003 | 0.002 |
| | | | (0.003) | (0.003) | (0.003) |
| $\beta_3$ - Law x border | | | 0.0001 | -0.004 | -0.004 |
| | | | (0.003) | (0.003) | (0.003) |
| $\beta_4$ - Nearby law x border | | | | | -0.001 |
| | | | | | (0.002) |
| *(B) Electronic access PDMP (main regression)* | | | | | |
| $\beta_1$ - PDMP law | -0.006*** | -0.005*** | -0.002 | -0.003 | -0.003 |
| | (0.001) | (0.002) | (0.002) | (0.002) | (0.002) |
| $\beta_2$ - Border county | | | 0.006*** | 0.004*** | 0.004*** |
| | | | (0.002) | (0.001) | (0.002) |
| $\beta_3$ - Law x border | | | -0.009*** | -0.008*** | -0.008*** |
| | | | (0.002) | (0.002) | (0.002) |
| $\beta_4$ - Nearby law x border | | | | | 0.0004 |
| | | | | | (0.002) |
| *(C) Must access PDMP* | | | | | |
| $\beta_1$ - PDMP law | 0.004*** | 0.003 | -0.001 | 0.001 | 0.001 |
| | (0.001) | (0.003) | (0.003) | (0.003) | (0.003) |
| $\beta_2$ - Border county | | | 0.001 | 0.0001 | -0.001 |
| | | | (0.001) | (0.001) | (0.001) |
| $\beta_3$ - Law x border | | | 0.012** | 0.006 | 0.006 |
| | | | (0.005) | (0.004) | (0.004) |
| $\beta_4$ - Nearby law x border | | | | | 0.010*** |
| | | | | | (0.003) |
| County FE | Yes | | | | |
| State FE | | Yes | Yes | Yes | Yes |
| Year FE | Yes | Yes | Yes | Yes | Yes |
| Controls | | | | Yes | Yes |
| Observations | 26,154 | 26,154 | 26,154 | 26,154 | 26,154 |

## Additional Figures




*Note:* The horizontal blue rectangle marks our sample period (2006 to 2014). For ePDMP, 9 states adopted before the start of our sample period, 16 states adopted in the first half of our sample, 18 states adopted in the second half, and 8 states had not adopted by the end of our sample period.

Figure B.1: PDMP implementation dates by state.

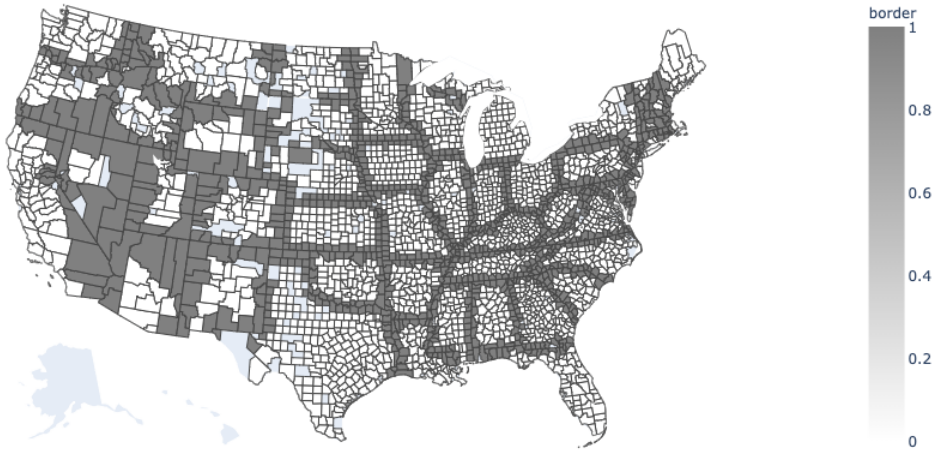
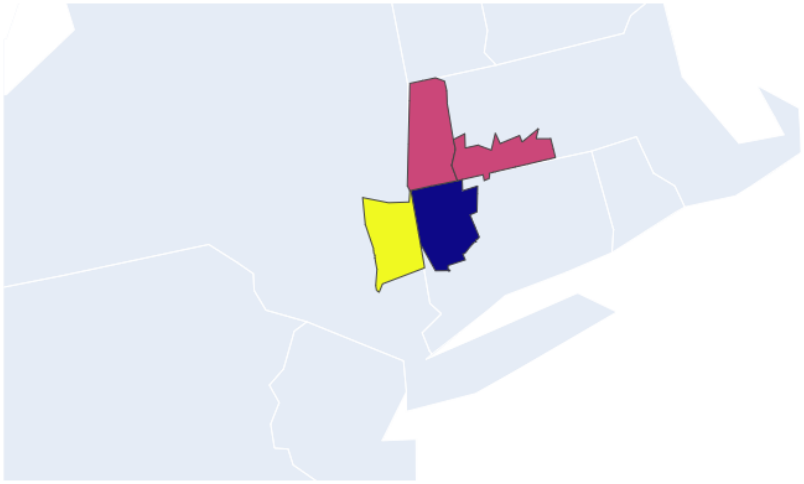



Figure B.2: Map of border vs. inland counties.



*Note:* This picture illustrates how we calculate nearby law for Litchfield County, Connecticut (blue) in 2012 . The Litchfield County borders three counties from nearby states: Dutchess County from New York (yellow), and Birkshire County and Hampden County from Massachusetts (pink). In 2012, the state of New York has not adopted ePDMP and the state of Massachusetts has. To calculate nearby law for Litchfield County, we sum up the population nearby with no ePDMP (294, 000) and the population nearby with ePDMPs (125, 000 + 466, 000 = 591, 000). Since more people nearby live in counties with ePDMP, nearby law for Litchfield County in 2012 is 1.

Figure B.3: Calculating nearby ePMDP status for counties bordering several states.

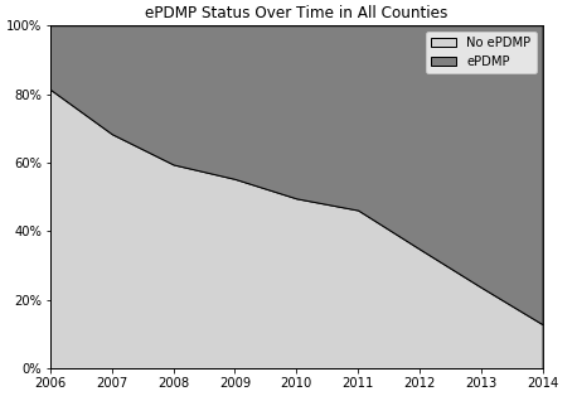
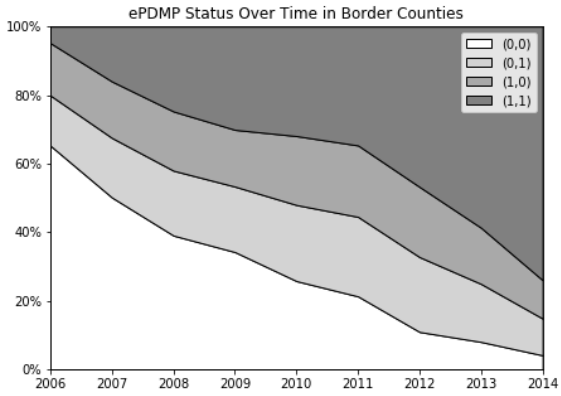



Figure B.4: ePDMP adoption over time in all counties.




*Note:* For the ease of reference, we use the (my law, nearby law) syntax to denote the ePDMP status of a border county. A border county of $(0, 0)$ has no ePDMP law and no nearby ePDMP law and its cross-state neighbors also do not have one; a border county of $(1, 0)$ has an ePDMP law himself but not nearby; a border county of $(0, 1)$ does not have a law himself it's nearby state does; and a border county of $(1, 1)$ has an ePDMP law itself and so do its out-of-state neighbors.

Figure B.5: ePDMP adoption over time in border counties.

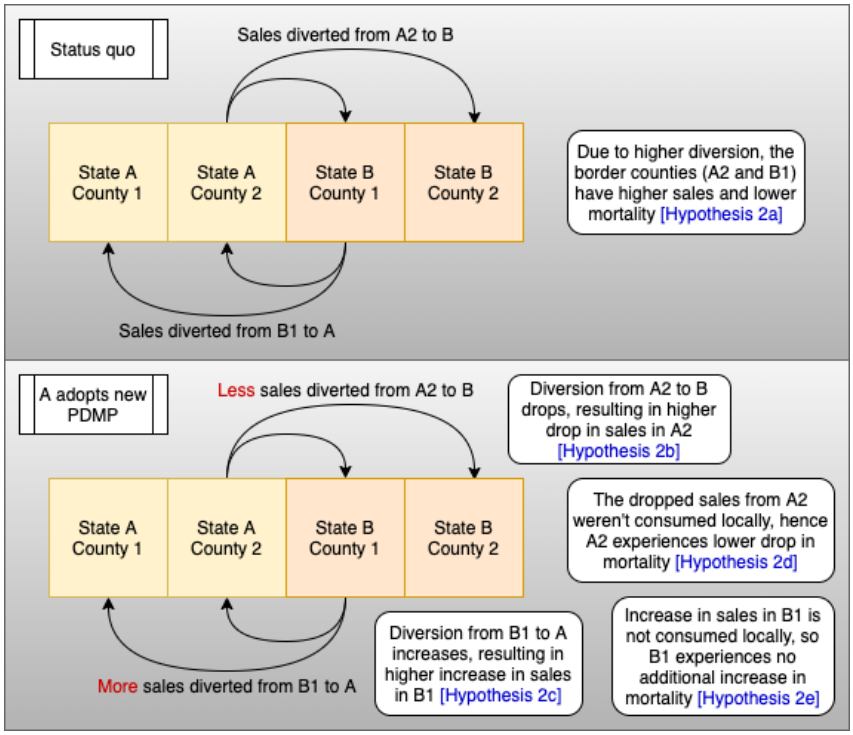



Figure B.6: Visual presentation of the spillover framework.

*Appendix C*

# CHAPTER 3 APPENDIX

**OLS Results Other Pollutants**

Table C.1: OLS regression of same-day CO pollution on daily absences for months without snow (April through November).

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_3$ - PPB of CO | 1.381*** | -4.260*** | 7.466*** | -11.54*** | 2.792*** |
| | (0.22) | (0.16) | (0.15) | (0.18) | (0.17) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.010 | 0.506 | 0.596 | 0.511 | 0.60 |
| $R^2$ Adj. | 0.010 | 0.506 | 0.596 | 0.511 | 0.600 |

Table C.2: OLS regression of same-day SO2 pollution on daily absences for months without snow (April through November).

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_4$ - PPB of SO2 | 50.01*** | -2.44** | 35.95*** | -56.94*** | 1.35 |
| | (1.17) | (0.78) | (0.76) | (1.01) | (0.98) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.012 | 0.506 | 0.597 | 0.511 | 0.600 |
| $R^2$ Adj. | 0.012 | 0.505 | 0.596 | 0.511 | 0.600 |

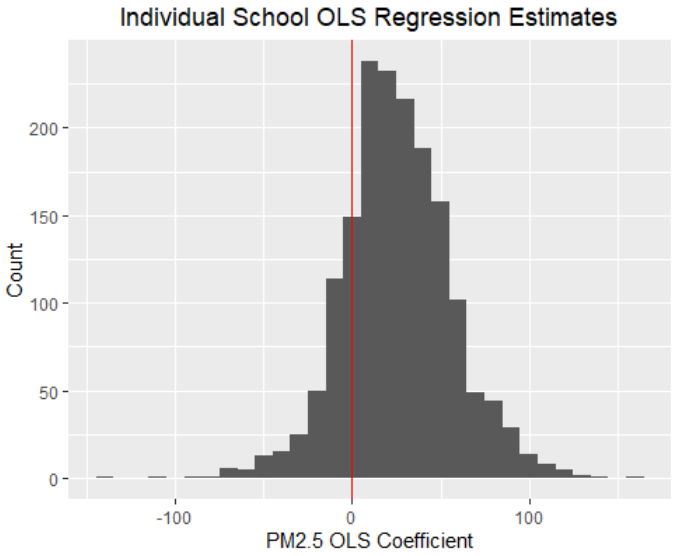



Figure C.1: PM2.5 OLS regression with every school run individually. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

Table C.3: OLS regression of same-day NO2 pollution on daily absences for months without snow (April through November).

| | Dependent Variable: | | | | |
|---|---|---|---|---|---|
| | Daily Absences Per 100,000 Students | | | | |
| | (1) | (2) | (3) | (4) | (5) |
| $\beta_4$ - PPB of NO2 | 9.08*** | -1.56** | 5.68*** | -10.47*** | -2.71*** |
| | (0.48) | (0.32) | (0.30) | (0.33) | (0.31) |
| School FE | | Yes | Yes | Yes | Yes |
| School × Month FE | | | Yes | | Yes |
| School × Year FE | | | | Yes | Yes |
| Observations | 2103540 | 2103540 | 2103540 | 2103540 | 2103540 |
| $R^2$ | 0.010 | 0.506 | 0.596 | 0.510 | 0.600 |
| $R^2$ Adj. | 0.010 | 0.505 | 0.595 | 0.510 | 0.600 |

**Additional PM2.5 OLS Regressions**

**Additional Ozone OLS Regressions**

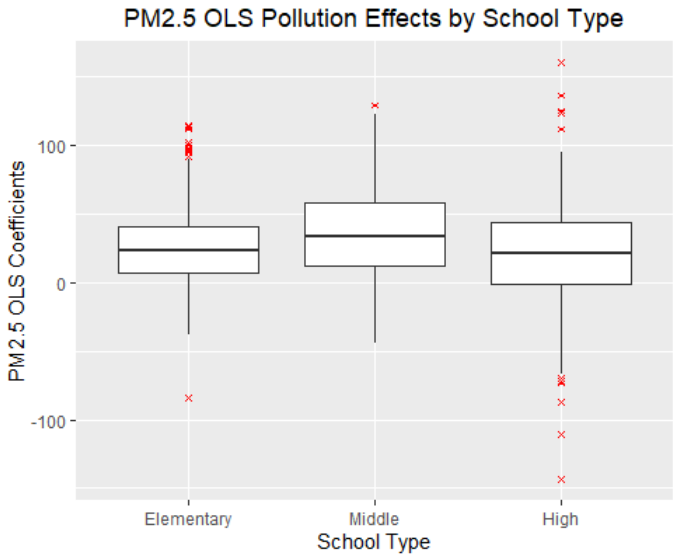



Figure C.2: PM2.5 OLS regression with every school run individually and grouped into school type. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

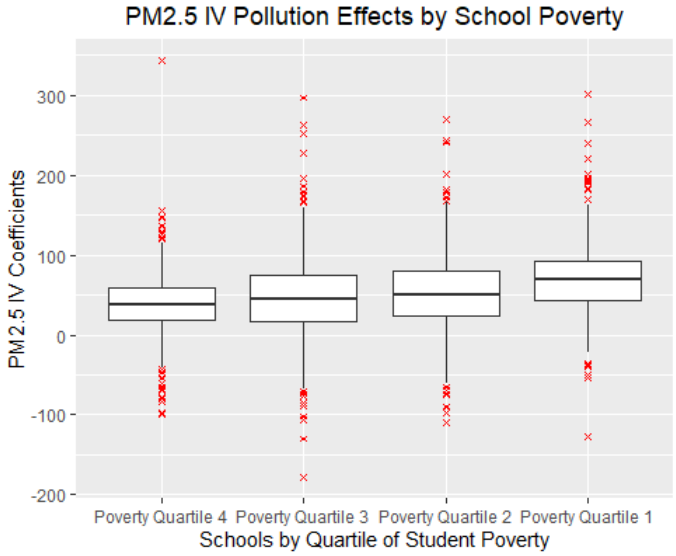



Figure C.3: PM2.5 OLS regression with every school run individually and grouped by percentage of students considered economically disadvantaged, where Quartile 4 is richest and Quartile 1 is poorest. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

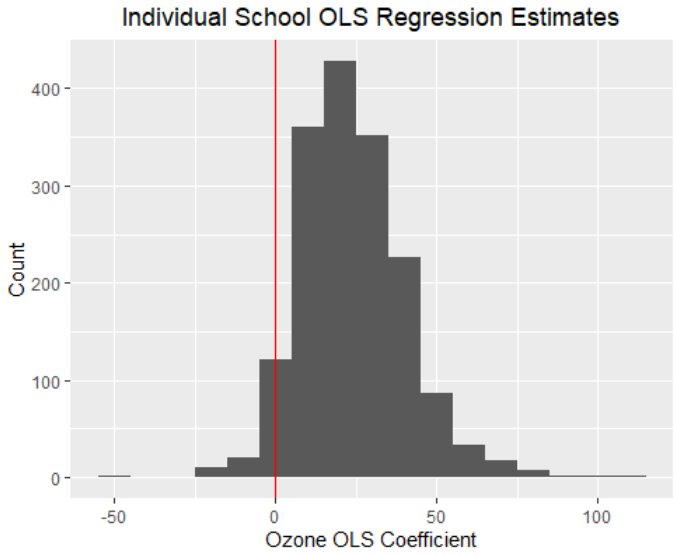



Figure C.4: Ozone OLS regression with every school run individually. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

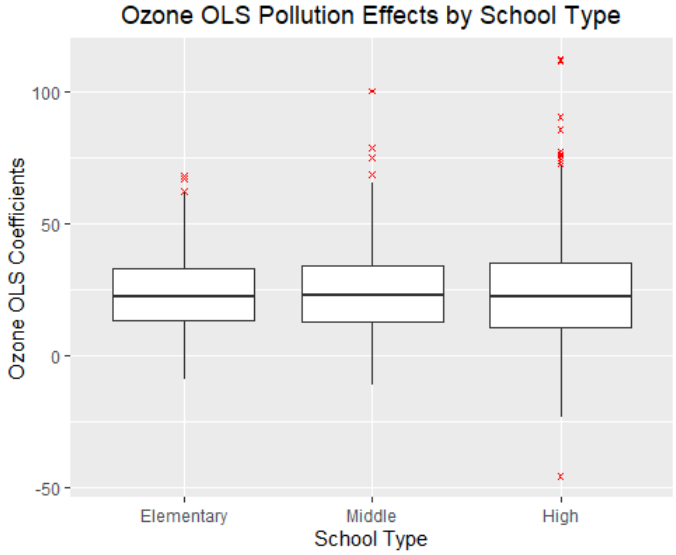



Figure C.5: Ozone OLS regression with every school run individually and grouped into school type. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.

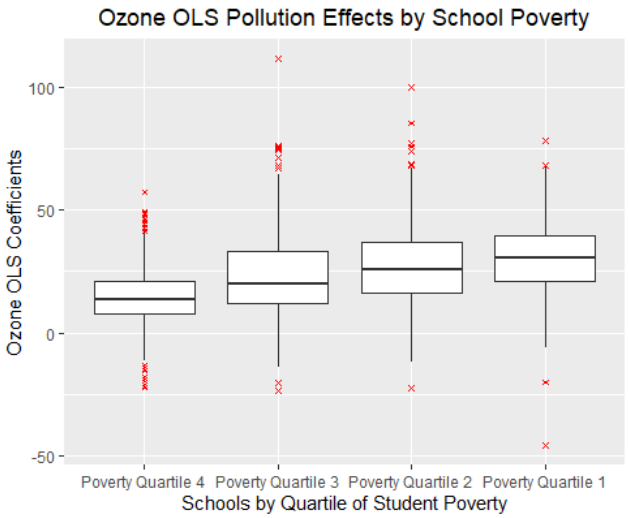



Figure C.6: Ozone OLS regression with every school run individually and grouped by percentage of students considered economically disadvantaged, where Quartile 4 is richest and Quartile 1 is poorest. These models include day of week, month, and year fixed effects, and the coefficient is reported in bins for every school.