

DISCUSSION PAPER SERIES

IZA DP No. 18090

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Infant Mortality Impacts of Industrial  
Lead Pollution**

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## ABSTRACT

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# The Hidden Toll of Airborne Lead: Infant Mortality Impacts of Industrial Lead Pollution\*

This paper uses U.S. Toxic Release Inventory data on industrial air lead emissions to provide IV estimates of the effects of air lead concentration on infant mortality. The causal effect of lead on infant mortality is identified by annual variation in air fugitive lead emissions interacted with wind speed near reporting plants, which together determine local ambient lead concentration. Unlike stack emissions, which occur routinely and may prompt avoidance behavior, fugitive emissions are intermittent and influenced by both historical and current factors, such as wind speed variation, making them difficult to avoid. The paper has two main findings. First, higher air lead concentration causes higher infant mortality in the first month and in the first year, suggesting that both in utero and environmental exposures matter. Second, higher lead concentration increases deaths from low birthweight, sudden unexplained infant death (SUID), and respiratory and nervous system causes, which is consistent with findings from animal studies, even when accounting for behavioral responses. Back of the envelope calculations indicate that declines in industrial air lead emissions prevented more than 300 infant deaths per year, generating benefits of at least \$3.5 billion annually in 2023 dollars.

**JEL Classification:** I12, Q53, Q58

**Keywords:** infant mortality, airborne lead pollution, Toxic Release Inventory (TRI)

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# 1 Introduction

Air lead concentrations remain substantial in both developed and developing countries in the early twenty-first century, largely due to industrial emissions. Emissions have been increasing in countries like China and Mexico (Li et al., 2012; Tanaka et al., 2022). Even after the phase-out of leaded gasoline, developed countries, including the United States, continue to experience notable levels of airborne lead pollution (EPA, 2018). These ongoing emissions contribute to persistently elevated child blood lead levels worldwide. A recent UNICEF report found that one in three children globally has blood lead levels above  $5\mu\text{g}/\text{dL}$ —a widely used threshold for high lead exposure (Burki, 2020; Rees and Fuller, 2020).

Despite significant levels of air lead emissions in the United States, the most recent revision of the National Ambient Air Quality Standards (NAAQS) for airborne lead in the U.S. in 2016 retained the 2008 standard (USEPA, 2016). This decision was largely due to a lack of robust new evidence on health harms at lower exposure levels. Two causal papers on lead and mortality exist, but neither is directly relevant to current policy. One paper, Clay et al. (2014), estimates the historical effects of lead on infant mortality between 1900 and 1920, but tells us very little about current relationships between lead and infant mortality. A second more recent paper, Hollingsworth and Rudik (2021), examines the relationship between lead and *elderly* mortality, but looks at intermittent high exposure associated with NASCAR races.<sup>1</sup> Those studies suggest a link between lead exposure and mortality, but new causal evidence on broader, lower-level population exposure in the modern era is still needed to inform current policy.

This paper draws on data from the Toxic Release Inventory (TRI), EPA monitors, and the confidential National Vital Statistics database to provide instrumental variable (IV) estimates of the effects of air lead concentration on infant mortality in the United States between 1988 and 2018. Air pollution studies often implicitly treat present-day pollution

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<sup>1</sup>In the absence of policy relevant evidence on lead and mortality, the focus has been on neurocognitive outcomes, which often have low benefits relative to the benefits associated with avoided mortality.

levels as accurate proxies for both short-term and long-term individual exposure. However, actual lifetime exposure is frequently unobserved. Because children tend to have shorter exposure windows and are less geographically mobile than adults, estimating the impact of pollution on children is typically more feasible (Currie et al., 2014). An additional advantage of focusing on early-life exposure is that preventing pollution-related deaths in infancy results in a greater number of life-years saved. While this does not diminish the value of protecting the health and well-being of the elderly, it highlights the particularly long-term benefits of reducing pollution exposure among the youngest and most vulnerable.

Beginning in 1987, firms were required to report emissions of a range of chemicals including lead to the TRI. Firms are allowed to use continuous, periodic, or random monitoring of emissions, or to follow EPA rules for calculations based on engineering models, emissions factors, and mass balance calculations. Notably, firms report both stack and fugitive emissions to TRI. Stack emissions are emissions that occur through confined air streams. Many plants use air pollution control devices to reduce stack emissions. Fugitive emissions include fugitive dust, which arises from traffic on paved and unpaved roads near plants and storage piles, and process fugitive emissions, which are uncaptured emissions that arise from handling and processing ore, furnace operation, hot metal transfer and processing, and casting.

Our estimation approach overcomes the challenges posed by avoidance behavior, omitted variable bias, and measurement error by instrumenting for EPA air lead concentration with TRI fugitive lead emissions *interacted* with wind speed near the emitting plants, conditional on a rich set of controls, including the fugitive emissions and wind speed themselves. While stack lead emissions occur routinely and may be subject to avoidance behavior, fugitive lead emissions are intermittent and depend on a variety of factors including: traffic; historical deposition of lead dust from fine ore, crushing, and other sources; production; and emission control efforts (EPA, 1994). Distances that emissions travel depend on wind speed. Unforeseeable annual variations in fugitive lead emissions and wind speed make it difficult to engage in avoidance behavior.

We provide evidence that fugitive lead emissions and wind speeds are strongly linked to air lead concentration readings of EPA monitors. Descriptive evidence also shows that fugitive lead emissions are positively and statistically significantly related to the percentage of children in a county who have blood lead levels above  $10\mu\text{g}/\text{dL}$ , while stack emissions are not. The effect is substantial – a one standard deviation increase in fugitive lead emissions is associated with a 13% increase in share of children with an elevated blood lead level.<sup>2</sup> Further, a higher proportion of fugitive emissions in total lead emissions is positively and statistically significantly associated with greater daily variability in ambient lead concentrations throughout the year, reflecting the intermittent nature of these emissions.

The paper has two main findings. First, higher air lead concentration causes higher infant mortality in the first postnatal month and in the first year, suggesting that both in utero and environmental exposures may contribute to this outcome. Our estimates increase as the timing of exposure is accounted for, highlighting the importance of measurement error. Estimates by race suggest proportionate effects on white and nonwhite infants, despite larger point estimates for nonwhites. Back of the envelope calculations indicate that declines in industrial lead emissions prevented 307 infant deaths per year, generating benefits of \$3.5 billion annually in 2023 dollars and that declines in air lead concentration prevented more than 412 infant deaths per year, generating benefits of \$4.7 billion per year. These estimates are based on sample counties that include 44% of the U.S. population and 34% of TRI air lead emissions and so represent lower bounds on the number of infant deaths attributable to air lead. Further, given that lead exposure causes morbidity and mortality in other populations beyond infants and has lasting impacts on child development, the societal benefits are likely substantially larger.

Second, higher lead concentration increases deaths from sudden unexplained infant death

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<sup>2</sup>In an urban sample with similar average blood lead levels that examines the effects of lead exposure from multiple sources, Lanphear et al. (1998) find an increase in water lead from  $0.0005$  to  $0.015\mu\text{g}/\text{L}$ —the current EPA water lead standard—is associated with a 13.7% increase in the share of children with blood lead levels above  $10\mu\text{g}/\text{dL}$ .

(SUID), respiratory and nervous system causes, and low birthweight.<sup>3</sup> This aligns with findings from animal studies, even when accounting for behavioral responses. Placebo tests show no link between lead and deaths from perinatal causes and deaths from congenital anomalies. The evidence on SUID is particularly interesting, because there had previously been speculation regarding the relationship between lead and sudden infant death syndrome (SIDS), which is a subset of SUID (Erickson et al., 1983; Lyngbye et al., 1985). The hypothesized mechanism was the adverse impact of lead on the parts of the brain that control sleep and arousal. Evidence from Kato et al. (2003) on infants studied for sleep apnea who did and did not later die of SIDS is consistent with this hypothesis, as is evidence on lead and sleep disruption in children (Jansen et al., 2019; Liu et al., 2015). Animal studies also support the possibility of a relationship between lead and SIDS. Lead causes neuroinflammation and sleep disruption in rats (Chibowska et al., 2020; Hsu et al., 2021). SUID and respiratory and nervous system illness may be the underlying cause of deaths of low birthweight infants, since deaths of these infants are often categorized as being from low birthweight rather than a specific cause.<sup>4</sup>

The paper contributes to two main literatures. The first is the literature on lead and infant health. Previous studies have shown impacts of air and water lead on intensive margin health outcomes such as birth weight and prematurity (Bui et al., 2022; Dave and Yang, 2022; Tanaka et al., 2022; Wang et al., 2022). There is also a sublitterature on historical *water* lead and infant mortality (Troesken, 2008; Clay et al., 2014), which provides causal effects of *water* lead on infant mortality for 1900-1920. See Clay et al. (2024) for a review on the effects of lead exposure on infant health. This paper provides the first causal estimates of the effects of airborne lead on infant mortality – an extreme adverse health outcome at the extensive margin – in a setting with modern medical care and at modern exposure levels.<sup>5</sup>

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<sup>3</sup>Sudden unexplained infant death includes sudden infant death syndrome (SIDS), accidental suffocation and strangulation on bed (ASSB), and other unexplained deaths.

<sup>4</sup>Air lead concentration shows a small and statistically insignificant positive effect on both prematurity and low birth weight. This suggests that higher lead exposure may worsen these infant health outcomes as well.

<sup>5</sup>Hollingsworth and Rudik (2021) find that air lead from leaded gasoline significantly impacts elderly mortality, but they find only weak evidence for its effects on infant mortality.

The second is the literature on the mechanisms through which lead exposure affects infant mortality. This topic has not been explored in economics, but has received some attention in animal studies and in the medical literature. Doctors in the medical literature had speculated that lead caused SIDS (now part of the the broader category of SUID) and this is supported by a range of other evidence discussed above (Erickson et al., 1983; Lyngbye et al., 1985; Kato et al., 2003; Liu et al., 2015; Jansen et al., 2019; Chibowska et al., 2020; Hsu et al., 2021). For respiratory causes, the animal literature provides evidence that lead weakens the immune system (Thind and Yusuf Khan, 1978; Dyatlov and Lawrence, 2002; Metryka et al., 2018). Weakening of the immune system may contribute to deaths from respiratory causes. This paper provides the first causal evidence linking lead exposure to individual causes of death, including sudden unexplained infant death (SUID), respiratory and nervous system causes, and low birthweight.

The paper also adds to the literature on air pollution and infant mortality (Chay and Greenstone, 2003; Currie and Neidell, 2005; Greenstone and Hanna, 2014; Luechinger, 2014; Gutierrez, 2015; Tanaka, 2015; Arceo et al., 2016; Knittel et al., 2016; Cesur et al., 2017; Alexander and Schwandt, 2022). Within this area, our study aligns with research on Toxic Release Inventory (TRI) emissions and infant mortality (Currie and Schmieder, 2009; Agarwal et al., 2010), which has identified a positive relationship between overall TRI emissions or broad chemical classes and infant mortality. While previous studies have examined pollutants such as particulate matter, carbon monoxide, and various other chemicals, this paper is the first to provide causal estimates of the effects of airborne lead on infant mortality.

The rest of the paper proceeds as follows. Section 2 discusses the background information on global use of lead, the Toxic Release Inventory, and the literature on lead and infant health. Section 3 describes our data. Section 4 presents our empirical strategy. Section 5 reports results, and Section 6 concludes.



## 2 Background

### 2.1 Toxic Release Inventory

The Toxic Release Inventory (TRI) was created by the Emergency Planning and Community Right-to-know Act (EPCRA) in 1986. The TRI was a response to chemical releases in Bhopal in 1984 and in West Virginia in 1985. The EPCRA required plants meeting certain criteria to annually report their emissions to the EPA for public disclosure through the TRI beginning in 1987. Lead was included in the original set of chemicals and so plants reported emissions beginning in 1987.<sup>6</sup> The EPA brought enforcement actions for non-reporting (Marchi and Hamilton, 2006). Analyses have found that TRI reporting is generally accurate (Brehm and Hamilton, 1996; Natan and Miller, 1998; Marchi and Hamilton, 2006).

Plants separately report stack and fugitive emissions for each chemical, including lead. Stack emissions are all releases “to the air that occur through confined vents, ducts, pipes, or other confined air stream” (EPA, 2023). Most plants use air pollution control devices to reduce stack emissions. These devices can include cooling towers, scrubbers, and bag houses that separate lead and other heavy metals from the exhaust. Lead collected by those devices may be recycled, transferred to offsite treatments, or emitted via wastewater or landfill, which generates substitution between air, water, and land emissions. The remaining lead in exhaust is emitted via stacks or other confined air streams.

Fugitive emissions are “all releases ... that are not released through confined vents, ducts, pipes, or other confined air stream” (EPA, 2023). Fugitive lead emissions include fugitive dust, which arises from traffic on paved and unpaved roads near plants and storage piles (EPA, 1994). Dust is deposited during ore arrival, transfer, and crushing, when fine ore dust falls to the ground and additional dust may be generated. Thus, fugitive dust reflects both current and historical deposition of lead. Fugitive process emissions are uncaptured

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<sup>6</sup>The TRI chemical list was expanded several times since 1987 to include more chemicals. The first reporting year of lead was 1987, but there were some concerns about completeness of reporting. We follow the literature and begin our analysis in 1988.

emissions that arise from handling and processing operations, furnaces, hot metal transfer and processing, and casting. These emissions are released directly, if the activity occurs outside, or via building openings such as roof vents and bays.

Firms are allowed to use continuous, periodic, or random monitoring of emissions or to follow EPA rules for calculations based on engineering models, emissions factors, and mass balance calculations. The EPA offers detailed guidance on the use of these methods and has a number of lengthy reports on estimation of fugitive lead emissions (EPA 1994; EPA 1998; EPA 2020b). Estimates will depend on what the lead is being used for, the specifics of the process including the types of furnace, and the types of emissions controls in place. Examples of processes that use lead include lead smelting, battery manufacturing, copper smelting, and solder and ammunition manufacturing. Examples of types of furnaces for lead smelting include blast, rotary, and kettle furnaces. Emissions controls include watering roads to reduce lead dust resuspension, changes in processes to reduce lead exposure to air, installation of hoods, and use of baghouses and scrubbers to reduce air lead emissions. For stack emissions, firms use the following approaches to calculate emissions: engineering calculations (45%), emission factors (24%), monitoring (22%), and mass balance (9%). For fugitive emissions, firms use: engineering calculations (61%), emission factors (22%), monitoring (9%), and mass balance (8%).

Figure 1 shows the downward trend in stack and fugitive air lead emissions, Appendix Figure A.1 displays emissions by industry, and Appendix Figure A.2 compares airborne with water and land emissions and recycled lead.<sup>7</sup> Fugitive emissions are around one-third of air lead emissions, with stack emissions making up two-thirds. The vertical lines indicate changes to the database in 1998 and 2001, when seven industries were added and the reporting threshold was lowered, respectively.<sup>8</sup> Four industry groups – lead manufacturing,

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<sup>7</sup>Pressure from environmental groups may have contributed to declines (Maxwell et al., 2000). Avoidance of nonattainment designation under NAAQS, which occurred in January 1992 for the 1978 lead standards, may have also contributed to the declines.

<sup>8</sup>In 1998, metal mining, coal mining, electric utilities, hazardous waste disposal, chemical wholesalers, petroleum terminals, and solvent recovery services were added to the list for reporting. They account for 14% of lead emissions after 1998. In 2001, the reporting threshold for lead was significantly lowered.

other metal manufacturing, ceramics manufacturing, and paint and pigment manufacturing – account for more than 90% of the total air lead emissions by all TRI-reporting facilities.<sup>9</sup>

## 2.2 Vectors of Lead Exposure

Although pregnant and nursing mothers and infants were exposed to lead through a number of vectors between 1988 and 2018, apart from industrial emissions these vectors did not vary or did so very slowly. For example, lead in soil and paint changed slowly or not at all. Lead in soil is a reflection of past deposition and local geology. Lead in paint is a function of the age of the housing stock. The federal government banned lead paint for housing in 1978, but some states had banned it earlier. Thus most housing with lead paint was built before 1960. Maternal and infant exposure tends to be through ingestion of soil, paint chips, or dust that includes lead from these sources or through breathing aerosolized dust.

Lead in water also changed very slowly. Lead in water is a function of the age of the housing stock and historical factors that drove the use of lead pipes for water in specific locations. The 1986 Safe Drinking Water Amendments required the use of lead free plumbing in public water systems. The 1991 Lead and Copper rule set limits on lead in tap water and required water systems to survey their use of corrosion control. Major changes to water systems such as those that occurred in Washington DC in 2001 and in Flint, Michigan, in 2014 can affect leaching of lead from lead pipes into water. These events have, however, been rare, and Washington DC and Genesee County, Michigan, are not in our sample.

By 1988, emissions due to lead in gasoline were already low. The dramatic decline in lead in gasoline over the 1970s and 1980s had been driven by two factors. The first was the requirement that new cars have catalytic converters beginning in 1974. Cars with these

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Appendix Figure A.3 shows that the number of reporting firms increased dramatically, but their contribution to the reported TRI lead emissions was small.

<sup>9</sup>The main activities of the lead manufacturing plants include extracting lead from lead ore or lead-bearing scrap materials (e.g., used lead-acid batteries) through high-temperature smelting and refining work. Iron, copper, and other metal manufacturing plants passively process lead contained in raw materials or in the coke and oil for combustion. Ceramics manufacturing uses lead compounds in glazing, and paint and pigment foundries use lead as quick driers (EPA, 2020a).

converters required unleaded gasoline. Over time leaded gasoline as a share of all gasoline fell. The second was regulatory decreases in allowable lead levels in leaded gasoline, which began in 1979 and reached its final level of 0.1 g/gallon in 1988.<sup>10</sup> As a result of these changes, air emissions from on-road vehicles fell from 171.96 in 1970 to 0.42 thousand tons in 1990. In 1990, air emissions from metals industrial processing was 2.17 thousand tons.

Although it may be possible to partially or fully avoid some vectors of exposure, avoidance is likely to be particularly difficult for fugitive lead. Lead exposure from water, soil, and paint can to varying degrees be avoided by not living in older housing stock, through testing of water, soil, and paint, and by remediation if levels are high. Stack lead may be partially avoidable if residents are aware of general smokestack emissions and stay inside during certain times to avoid them. Residents are, however, unlikely to be aware of fugitive emissions, which are intermittent and not stack based, and the role that wind speed plays in the dispersion of lead.

To provide descriptive evidence that pregnant women and infants are likely to have been impacted by industrial air lead emissions, we investigate the effect of fugitive and stack lead emissions on the percentage of children who have blood lead levels above  $10\mu\text{g}/\text{dL}$ . Our main sample includes 127 counties, representing 31% of the US population in 1990, as discussed in the data section. The CDC funds 35 state and local health departments for lead surveillance.<sup>11</sup> Because only a subset of states report child blood lead levels to the CDC, and child blood lead level data are only available for 2005-2015, the analysis is for a subset of the counties and years that we examine our main analysis. It includes the same control variables used in our main specification.

Table 1 highlights the differential effect of fugitive and stack emissions on the percentage of children who have blood lead levels above  $10\mu\text{g}/\text{dL}$ . The coefficient on air fugitive lead emissions is positive, statistically significant, and substantial. In contrast, the coefficient

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<sup>10</sup>In 1996, the use of lead in gasoline for on-road vehicles was banned entirely in the United States.

<sup>11</sup>The funding supports collection in 29 states, the District of Columbia, and five cities – Chicago, Houston, Los Angeles, New York City, and Philadelphia

on air stack lead emissions is positive, but not statistically significant and small. In our preferred estimate in column 4, a one standard deviation increase in fugitive lead emissions is associated with a 0.805 percentage point increase in the percentage of children with elevated levels. Given that 6.57% of children had elevated blood lead levels in these counties, this translates into a 13.4% increase in share of children with an elevated blood lead level.<sup>12</sup> For comparison, in an urban sample with similar average child blood lead levels, Lanphear et al. (1998) investigate the effects of increases in lead in household dust, soil, paint, and water on child blood lead levels. They find an increase in water lead from 0.0005 to 0.015 $\mu\text{g}/\text{L}$ —the current EPA water lead standard—is associated with a 13.7% increase in the share of children with blood lead levels above 10 $\mu\text{g}/\text{dL}$ .

## 2.3 Lead and Infant Mortality

Lead is known to cause adverse health effects in humans across a range of exposures. At very high levels, exposure to lead can cause lead poisoning. Lead exposure is also harmful at lower levels. The large epidemiological literature on the health effects of low level lead exposure is comprehensively reviewed by the National Toxicology Program in NTP (2012). Lead adversely impacts the neurological, immune, cardiovascular, and renal systems. Mason et al. (2014) reviews the neuropsychological effects of lead toxicity. Adverse effects of lead come through at least three channels – morphological, pharmacological, and indirect effects. Morphologically, lead disrupts or alters development of the nervous system both prenatally and after birth. Pharmacologically, lead substitutes for calcium and zinc, disrupting or altering operation of the nervous system. Indirect effects come from lead’s effects on other bodily systems.

One causal study and numerous epidemiological studies show relationships between air lead and adult mortality. Hollingsworth and Rudik (2021) use the switch in racing fuel from leaded to unleaded in NASCAR and ARCA races to examine the causal effect of lead on

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<sup>12</sup> $0.805 \times 1.094 / 6.572 = 0.134$

elderly mortality. They find that having a leaded race in a county in a given year increased the elderly all-cause mortality rate, with much of the change coming from cardiovascular mortality and ischemic heart disease. Epidemiological evidence supports a link between lead and cardiovascular mortality that appears to be driven at least in part by lead’s impact on blood pressure.<sup>13</sup>

To our knowledge, there are no studies that show a causal relationship between air lead and infant mortality.<sup>14</sup> This reflects a lack of studies, as opposed to studies finding a lack of harm. Animal studies support the link between water lead and infant mortality (Aprioku and Siminialayi, 2013). Studies that examine waterborne lead in the early twentieth century also provide evidence that in utero and infant exposure caused infant mortality (Troesken, 2008; Clay et al., 2014).<sup>15</sup> Employing data from Massachusetts towns in 1900, Troesken (2008) compares infant death rates in cities that used lead water pipes to rates in cities that used nonlead pipes. In the average town in 1900, the use of lead pipes increased infant mortality by 25 to 50%. Using data from 1900-1920, Clay et al. (2014) provide causal evidence on water lead and infant mortality, leveraging differences in use of lead pipes for water and differences in the acidity of water sources.

The mechanisms through which contemporary levels of lead exposure might cause infant mortality are not well understood. Part of the issue is that infant mortality rates are low, and lead has not been linked to major causes of death such as conditions originating in the perinatal period and congenital anomalies. One potential pathway is damage to neurological development, possibly including damage to the parts of the brain that control sleep and

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<sup>13</sup>Recent work by Lanphear et al. (2018) using NHANES data suggests that lead may account for as much as 18% of all-cause mortality and larger shares of cardiovascular mortality. While not a causal analysis, the estimates are worth noting because of their large size. Earlier papers including Pirkle et al. (1985), Lustberg and Silbergeld (2002), Menke et al. (2006) also found a relationship between lead and mortality.

<sup>14</sup>Two observational studies examine the link between TRI emissions and infant mortality. Currie and Schmieder (2009) uses TRI data, leveraging the difference between fugitive and stack emissions. As part of a larger analysis of TRI emissions and their impact on birth outcomes, they find wrong signed estimates of lead on infant mortality. A related economics literature uses aggregate TRI data to examine infant mortality. Agarwal et al. (2010) uses national data from 1989-2002 and finds that the elasticity of infant deaths with respect to TRI air emissions is 0.03.

<sup>15</sup>In a recent observational study, Edwards (2014) shows that spikes in water lead in Washington DC due to the switch from chlorine to chloramine are associated with higher fetal death rates.

arousal. As mentioned earlier, the medical literature has speculated that this might be an important pathway for sudden infant death syndrome (SIDS), which is a subset of SUID (Erickson et al., 1983; Lyngbye et al., 1985). Another potential pathway is through effects on the immune system. In a study that exposed lab mice to various dosages of lead and then to bacteria, mice with higher exposure to lead were more likely to die (Dyatlov and Lawrence, 2002). We know little about the importance of these or other mechanisms in humans at modern levels of exposure.

### 3 Data

Data on industrial fugitive and stack emissions of lead and other chemicals are from the U.S. Toxic Release Inventory (TRI). The TRI covers a large number of chemicals – 650 chemicals in 2018. TRI chemicals are classified into different and partially overlapping categories. For example, 189 chemicals are classified as hazardous air pollutants (HAP). The EPA states that “[h]azardous air pollutants (HAPs) are those pollutants that are known or suspected to cause cancer or other serious health effects, such as reproductive effects or birth defects, or adverse environmental effects.” One hundred chemicals are classified as developmental toxins, which are thought to affect reproductive success or to affect fetal, infant, or child development. Lead is listed as both HAP and a developmental toxin.<sup>16</sup>

Lead monitor data are from the EPA’s Air Quality System (AQS). The AQS provides daily-level monitoring data on air lead levels measured in micrograms per cubic meter of air ( $\mu\text{g}/\text{m}^3$ ). The number of lead monitors varies over time from 2.1 to 4.1 monitors per county.<sup>17</sup> AQS monitor data on particulate matter (PM) and carbon monoxide (CO) are also used in some specifications.

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<sup>16</sup>We use information from the TRI-chemical Hazard Information Profiles to identify developmental toxins. See <https://www.epa.gov/toxics-release-inventory-tri-program/tri-supplemental-documentation> for more information on developmental toxins. See <https://www.epa.gov/haps/what-are-hazardous-air-pollutants> for more details on HAP chemicals.

<sup>17</sup>Alternatively, 60 to 261 monitors in the 127 sample counties. Notice, however, that the number of counties also varies over time in the sample.

Wind data are from the Global Summary of the Day (GSOD) provided by the National Centers for Environmental Information (NCEI). The GSOD provides daily-level monitoring data on wind speed and the data on locations of weather stations. Because wind is highly local, we restrict the sample to plants within 10 miles of a wind monitoring weather station.<sup>18</sup> County-average climate data on precipitation and temperature are from the Parameter-elevation Regressions on Independent Slopes Model (PRISM) Climate Data.

Data on infant health are from the National Vital Statistics system of the National Center for Health Statistics (NCHS). Our analysis uses the restricted sample, which covers reported births and infant deaths in all counties. The NCHS provides information on infant birth and death, birth weight (in grams), and gestation weeks. It also provides data on mothers' characteristics including age, race, ethnicity, and education. County-level characteristics are from the census. County socio-economic data on employment, income, race, and other characteristics are from the decennial censuses and the 2018 American Community Survey (5-year data).

Our main sample includes 127 counties that have plants with lead emissions that are within 2 miles of EPA lead monitors and within 10 miles of a wind monitor.<sup>19</sup> In 1990, 31% of the US population lived in these counties. This sample accounts for 21% of lead emissions and 1.6% of TRI total emissions. Appendix Figure A.4 shows the geographic distribution of the counties used for the IV sample. Appendix Figure A.5 shows the distribution of wind speeds for the IV sample. Appendix Table A.1 presents birth-weighted county-level summary statistics for the IV sample.

We also calculate effects for an extended sample of 227 counties. These counties that have plants with lead emissions that are within 2 miles of EPA lead monitors but are not within 10 miles of a wind monitor. In 1990, 44% of the US population lived in these counties. This sample accounts for 34% of lead emissions and 2.6% of TRI total emissions.

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<sup>18</sup>Expanding the radius does not include many more plants in the sample, but makes the estimates noisier.

<sup>19</sup>Analysis below shows that plant lead emissions are only detectable at EPA lead monitors within 2 miles.



## 4 Empirical Strategy

As discussed earlier, there are three challenges to identify the impact of air lead concentrations on infant mortality. First, downward bias may arise if parents engage in avoidance behavior based on observed pollution. Second, omitted variable bias may occur if local economic activity impacts infant health through factors like local demand shocks, which can influence employment and income levels. This is because the level of pollution emitted is partially determined by the production scale of the plants, which is correlated with the economic impacts. Third, measurement error arising from the usual disconnection between where air lead is measured and where individuals live, as well as from the timing of exposure, may generate attenuation bias.

Our empirical strategy overcomes these challenges by instrumenting air lead concentration with the *interaction* between fugitive lead emissions and wind speed, conditional on these variables themselves and other controls. Basically, our strategy compares the effect on infant mortality of air lead concentrations caused by high versus low fugitive lead emissions during years with stronger versus weaker winds. Stack emissions are predictable, are often emitted from high smokestacks, may be subject to avoidance behavior, and are correlated with production scale and economic impact. In contrast, fugitive emissions are intermittent, often occur closer to the ground, may be less observable to people than the smoke from tall stacks, and are relatively uncorrelated (0.5) with stack emissions. Wind speed affects how far lead emissions disperse and year-to-year variations in wind speed are plausibly exogenous.

Regarding the pattern of fugitive emissions, Table 2 documents that a greater share of fugitive emissions in overall lead emissions is positively and statistically significantly associated with the daily standard deviation in ambient lead concentration. This evidence seems to indeed support the intermittency and higher unpredictability of fugitive emissions.

## 4.1 Air Lead Emissions and Concentrations

Our first stage estimating equation can be expressed as:

$$\begin{aligned}
 AirLead_{ct} = & \delta^F F_{ct} + Wind_{ct} \delta_w + (Wind_{ct} \times F_{ct}) \delta_w^F \\
 & + \delta^S S_{ct} + (Wind_{ct} \times S_{ct}) \delta_w^S + Chem_{ct} \delta_c + (Wind_{ct} \times Chem_{ct}) \delta_{wc} \\
 & + \eta_c + \lambda_{rt} + Media_{ct} \psi + Z_{ct} \pi + \omega_{ct}
 \end{aligned} \tag{1}$$

where  $AirLead_{ct}$  is air lead concentration in county  $c$  in year  $t$ , measured as the average across all monitors within two miles from any industrial plants with air lead emissions.<sup>20</sup> The key explanatory variables are  $F_{ct}$ , denoting the aggregated fugitive lead emissions from plants in county  $c$  in year  $t$ , and its interaction with  $Wind_{ct}$ , a fourth order polynomial for wind speed.<sup>21</sup> We control for the stack lead emissions ( $S_{ct}$ ), fugitive and stack emissions of other TRI-reported chemicals ( $Chem_{ct}$ ), and their interactions with wind.<sup>22</sup> The regression includes county fixed effects ( $\eta_c$ ) and region-by-year fixed effects ( $\lambda_{rt}$ ) to control for time-invariant determinants of and region-specific trends in infant mortality over time. We also control for waterborne and landborne lead emissions ( $Media_{ct}$ ).<sup>23</sup>

We allow for higher orders of the polynomial in the *interaction* between fugitive lead emissions and wind speed because of (i) potentially different heights at which fugitive emissions happen, and (ii) the established nonlinear relationship between wind speed and emission dispersion. Unlike stack emissions, fugitive lead emissions can occur at different heights,

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<sup>20</sup>To improve precision in the measurement of this variable, we have examined the relationship between lead emissions, wind, and distance between emitting plants and EPA monitors. See Appendix A.1. We have provided evidence that the EPA monitors detect air lead emissions up to two miles from emitting plants. We have also explored the impact of wind direction by splitting the lead emissions into those from plants upwind from the monitors and from plants downwind from the monitor. We did not find improvements in the F-stats when adding wind direction in the analysis. This is perhaps because the North American Regional Reanalysis (NARR) wind direction data that we and many other papers use (e.g., Deryugina et al., 2019) are noisy at a 2-mile scale.

<sup>21</sup>We will compare first stage and IV results with third, fifth and sixth order polynomials as well.

<sup>22</sup>Appendix Table A.2 presents the empirical variation in key explanatory variables.

<sup>23</sup>We only include onsite emissions. The offsite emissions via water and landfill mainly happened on waste treatment facilities that locate far from the original neighborhood of the manufacturing facilities. But controlling for offsite emissions does not affect our results.

including on the ground level. This unknown pattern can generate uneven distribution of pollution across space.

Regarding the relationship between wind speed and emission dispersion, it has been established that the nonlinearity depends on two key factors: air buoyancy and air turbulence. Air buoyancy refers to the upward force exerted by the air on objects placed within it. This force occurs due to the difference in density between the object and the surrounding air. Objects that are less dense than the air around them (e.g., hot air balloons) experience a buoyant force that enables them to float or rise upward. Air turbulence refers to irregular or disturbed flow of air in the atmosphere. It often manifests as rapid and unpredictable changes in wind speed and direction, causing fluctuations in air pressure. Turbulence can occur at various altitudes and can arise from uneven heating of the Earth’s surface or interruption of air flow due to natural or man-made structures such as mountains, buildings, or other terrain features.

Appendix Figure A.6 displays plume patterns based on laboratory experiments under different air buoyancy and turbulence conditions. Appendix Figure A.7 displays several theoretical plume patterns based on meteorological conditions in the United States. On an annual basis, looping (Panel A) is one of the most frequent situations experienced in the country, having a frequency of about 30% or more. Thus, it is evident that we need a higher-order polynomial for the interaction between fugitive lead emissions and wind speed to capture this fluid dynamics.

To address concerns on omitted variable bias in our main equation below on infant mortality and air lead concentration, our first stage equation also controls for a rich set of factors that have been linked to infant health ( $Z_{ct}$ ): county socioeconomic characteristics (population density, percent white, percent age 25 and older with high school degree, median household income, percent manufacturing employment, and employment rate); climate variables (county-average annual precipitation and temperature); mothers’ characteristics (percent white, percent Hispanic, percent high school degree, and percent aged over 35); and

linear trends of baseline mortality rate from 1980 to 1986.

All regressions are weighted by the number of live births. Robust standard errors are clustered at the county-level to adjust for arbitrary heteroskedasticity and within-county serial correlation. From the point of view of the design-based approach for standard errors (Abadie et al., 2023), the assignment to treatment is also at the county level. In robustness checks, we also provide results using spatial standard errors.

## 4.2 Air Lead Concentrations and Infant Mortality

To measure the effect of air lead concentrations on infant mortality, we estimate the following equation:

$$\begin{aligned} InfMort_{ct} = & \beta^{AL} AirLead_{ct} + \beta^F F_{ct} + Wind_{ct}\beta_w \\ & + \beta^S S_{ct} + (Wind_{ct} \times S_{ct})\beta_w^S + Chem_{ct}\beta_c + (Wind_{ct} \times Chem_{ct})\beta_{wc} \\ & + \eta_c + \lambda_{rt} + Media_{ct}\theta + Z_{ct}\gamma + \epsilon_{ct} \end{aligned} \quad (2)$$

where  $InfMort_{ct}$  is infant mortality in county  $c$  in year  $t$ .<sup>24</sup> We instrument  $AirLead_{ct}$  with the *interactions* between fugitive lead emissions and the fourth order polynomial in wind speed ( $Wind_{ct} \times F_{ct}$ ). Fugitive lead, wind speed, stack lead, and the other control variables are the same as in the first stage equation.<sup>25</sup>

The exclusion restriction assumes that the *interactions* between fugitive lead emissions and wind speed affects infant mortality solely through its impact on air lead concentrations. We account for the direct effects of both fugitive lead emissions and wind speed on infant

<sup>24</sup>We also examine premature birth (< 37 weeks) and low birthweight (< 2500 grams).

<sup>25</sup>We considered a two-sample IV approach, using the more restricted sample for the first stage and the broader sample for the second stage. However, lead emission levels in the regular IV sample counties are substantially higher—about four times greater—than in other TRI counties with lead emissions. This discrepancy raised concerns about the validity of extrapolating the first-stage relationship to the broader sample. Moreover, given evidence in the literature of a nonlinear (concave) relationship between pollution and health outcomes (Pope III et al., 2009,0; Heft-Neal et al., 2023; Miller et al., 2025), combining a higher-exposure sample with a lower-exposure one could lead to biased estimates. To avoid this risk, we ultimately chose not to pursue this strategy.

mortality, as well as the direct effects of stack lead emissions and their interactions with wind, alongside other control variables. Because only the combination of intermittent fugitive lead emissions and unexpected local wind strength is excluded from the main equation, this assumption should be reasonable.

## 5 Results

### 5.1 First Stage: Air Lead Emissions and Concentrations

In this section, we show that fugitive lead emissions have predictive power for air lead concentration even when we include a range of controls. Furthermore, we present evidence that a fourth order polynomial provides a better and a more parsimonious fit than lower or higher order polynomials, consistent with the nonlinear relationship between wind speed and emission dispersion discussed in the empirical strategy section. Lastly, we report that fugitive lead interacted with wind does not predict either county or maternal characteristics, which supports the exclusion restriction assumption.

Figure 2 highlights that fugitive lead emissions have predictive power for air lead concentration even when we include a rich set of controls. The figure plots the highly nonlinear relationship between fugitive lead emissions ( $\hat{\delta}_w^F$ ) and air lead concentration as a function of wind speed for counties with plants within 2 miles of an EPA air lead monitor. Appendix Figure A.8 shows the graphs as we move from the most parsimonious specification to specifications with richer sets of controls. The F-statistics are all at 38.0 or higher, and the graphs are remarkably similar.

Appendix Figure A.9 demonstrates that a fourth order polynomial is a parsimonious model to capture the nonlinear relationship between fugitive lead emissions ( $\hat{\delta}_w^F$ ) and air lead concentration. It plots the marginal effects of a specification that includes a full set of controls as we move from a first order polynomial to a sixth order polynomial.<sup>26</sup> The F-

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<sup>26</sup>Again, this is not surprising given the complexity in modeling air pollution due to atmospheric turbulence

statistic is much higher for the fourth order polynomial (41.2) than for the third order (17.6), which suggests that the addition of a fourth order term provides benefits. It is worth noting that the fifth and sixth order polynomials have lower F-statistics (31.4, 26.3), suggesting that there are no benefits to adding more terms beyond the fourth order term.

Appendix Tables A.3 and A.4 show that fugitive lead interacted with wind does not predict either county or maternal characteristics. The F-statistics are all below 4, with the exception of population density, where the F statistic is below 6. This evidence seems to provide support for our exclusion restriction assumption. Among the variables considered, there does not appear to be any other channel through which the interaction between fugitive lead and wind meaningfully affects our outcome variables. We provide a range of additional robustness checks in Section 5.4.

## 5.2 IV Effects of Air Lead Concentrations on Infant Mortality

Table 3 shows that higher levels of air lead concentrations cause higher infant mortality.<sup>27</sup> The coefficient on air lead concentration in the IV specification is positive and statistically significant across all five specifications. The decline in air lead concentration in the sample is  $0.144 \mu g/m^3$  from 1988 to 2018.<sup>28</sup> This decrease in air lead concentration would decrease infant mortality by 0.241 per thousand live births or about 3.1% of mean infant mortality.<sup>29</sup> We discuss deaths averted due to changes in lead emissions and deaths averted due to declines in air lead concentration further in Section 5.5.

Table 4 shows that the estimates in Table 3 are robust to different ages at death and to adjusting for the timing of exposure. Two-thirds of infant deaths occur within the first

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(e.g., Nieustadt and van Dop, 1982; Raputa and Lezhenin, 2020).

<sup>27</sup>Appendix Figure A.10 displays the reduced form relationship between fugitive lead emissions and infant mortality as a function of wind speed for the IV sample. The pattern is generally consistent with the first stage depicted in Figure 2. The reduced form shows a positive effect at higher wind speeds, suggesting that populations beyond the vicinity of the plants may be affected.

<sup>28</sup>0.144 is the difference between the average county-specific air lead concentration over 1988 to 1991 and 2015 to 2018. We take the mean over years to reduce reliance on any specific year.

<sup>29</sup>0.241 is calculated by multiplying the coefficient of air lead concentration, 1.676, in column 5 Table 3 by 0.144, the change in air lead concentration.  $3.1\% = 0.241 / 7.718$ , where 7.718 is the mean infant mortality rate across the sampling county-years.

month. For these infants, a large share of lead exposure will have come in utero. Column 1 presents the results for deaths within the first month, and column 2 replicates our preferred results from column 5 of Table 3 for deaths within the first year. The coefficient on air lead concentration for deaths within the first month in column 1 is positive and statistically significant, and the magnitude of the coefficient is smaller than in column 2.

One concern with the analysis in columns 1 and 2 is that many infants may have been exposed to lead in the previous year. For example, an infant that is born in January and dies in January experienced nearly all of its in utero exposure in the previous year. Columns 3 and 4 restrict attention to infant deaths from April to December. For mortality in the first month (April) in column 3, all of the infants spent at least the third trimester in the current year. The median infant in this sample was born in mid-August and so spent part of the first trimester and all of the second and third trimesters in the current year. Columns 5 and 6 restrict attention to infant deaths from July to December. For mortality in the first month (July) in column 5, all of the infants spent at least the second and third trimester in the current year. The median infant in this sample was born at the very beginning of October and so spent all three trimesters in the current year.

Compared to the results in columns 1 and 2, the point estimates in columns 3-6 are substantially larger, suggesting that using current year exposure for infants with significant exposure in the previous year is causing some attenuation bias. For infant mortality within the first month, the point estimates increase from 0.728 for January-December to 1.155 for April-December and 1.143 for July-December. For infant mortality within the first year, the point estimates increase from 1.676 for January-December to 2.128 for April-December and 2.660 for July-December.

### 5.3 Mechanisms and Heterogeneity Analysis

Table 5 draws on data on cause of death to better understand the mechanisms through which lead is causing mortality. In both panels, air lead concentration is statistically significantly

related to five causes: low birthweight; sudden infant death syndrome (SIDS) and accidental suffocation and strangulation on bed (ASSB); respiratory; nervous; and other causes of death. The coefficients on air lead concentration are small and not statistically significant for the other two causes: congenital anomalies; and other conditions originating in the perinatal period.

Before discussing the low birthweight mortality results, it is useful to examine the average effect of lead exposure on low birthweight and prematurity. Appendix Table A.5 shows the IV effects of lead on the number of low birthweight and prematurity per thousand live births are positive but imprecisely estimated. Taken at face value, the implied decreases in these outcomes from the  $0.144 \mu\text{g}/\text{m}^3$  decline in air lead concentration over the study period are 0.305 for low birthweight and 0.390 for prematurity, per thousand live births, which represent 0.4% and 0.5% of their means, respectively.

Taken together, Tables 5 and A.5 suggest that in places with higher air lead concentration, not only the incidence of low birthweight is significantly higher, but also low birthweight infants are more likely to die. In column 1 of Table 5, higher air lead concentration is causally related to deaths associated with low birthweight. The coefficients are nearly identical in magnitude at one month (0.363) and at one year (0.349). We would expect the magnitudes to be similar, since low birthweight infants generally die in the first month.

Because of inconsistent classification, sudden infant death syndrome (SIDS) and accidental suffocation and strangulation on bed (ASSB) are often combined with other unexplained infant deaths into a single category called SUID, or sudden unexpected infant death. SUID deaths tend to peak in months 1-4 (Moon et al., 2016). As we noted earlier, there has been speculation in the medical literature about the link between lead and SIDS (Lyngbye et al., 1985; Erickson et al., 1983). In 1994, a number of organizations including the American Academy of Pediatrics launched the Back-to-Sleep Campaign to address SIDS. The inclusion of region-by-year fixed effects in our preferred specification should control for introduction of this campaign.



Table 5 provides evidence of a causal link between air lead concentration and incidence of SUID. Consistent with the evidence on the timing of SUID, the coefficient is much smaller for mortality in the first month (0.020) than it is for mortality in the first year (0.407).

Regarding the impacts on deaths caused by diseases of the respiratory and nervous systems, the results are not surprising because lead is a component of particulate matter. Previous studies have estimated the causal impact of exposure to particle pollution on infant mortality, mostly associated with respiratory illnesses (e.g., Chay and Greenstone, 2003; Currie and Neidell, 2005). The novelty of our results is that we can pinpoint the impacts of lead, controlling for other pollutants. Recall that the reduction in air lead concentration in the sample is  $0.144 \mu\text{g}/\text{m}^3$ . The implied respiratory effects are 0.035 (at one month) and 0.060 (at one year) per thousand live births, which represent 5.6% and 6.7% of the mean. The implied nervous system effects are smaller in magnitude, but much larger relative to the basis: 0.008 (at one month) and 0.017 (at one year) per thousand live births, which represent 47.1% and 15.2% of the mean. Although no prior study finds impacts on infant mortality, our results corroborate a large literature documenting the relationship between lead exposure and cognitive and behavioral issues (e.g., Needleman and Bellinger, 1991; Needleman, 2004; Reyes, 2007; Aizer et al., 2018; Aizer and Currie, 2019; Reyes, 2015).

Estimates by race in Table 6 suggest proportionate effects on white and nonwhite infants, despite larger point estimates for nonwhites. It is worth noting that because of the large standard errors, the two point estimates in columns 1 and 2 are not statistically significantly different from each other. The implied effects for the  $0.144 \mu\text{g}/\text{m}^3$  decrease in air lead concentration are 0.44 per thousand live births for nonwhite infants and 0.20 for white infants. Despite the differences in the coefficients, the effects are proportionate at 3.7% and 3.4% of nonwhite and white mortality. Columns 3 and 4 suggest that these differences are also present in the first month.

## 5.4 Further Robustness Checks

Appendix Table A.6 shows that the results are robust to adding individual groups of chemicals and particulate matter (PM10) and carbon monoxide (CO) as controls. Recall that our estimates in Table 3 shifted very little from column 4 to column 5 with the addition of three groups of chemicals – developmental chemicals, nondevelopmental chemicals, and HAPs – all interacted with wind as well. Consistent with this in Appendix Table A.6, the F-stat is quite similar across the first three columns as we include controls for developmental chemicals in column 1, add nondevelopmental chemicals in column 2, and add HAPs in column 3. The third column is our base model from Table 3. Column 4 adds controls for PM10 and column 5 adds controls for CO. Unfortunately, not all monitoring stations have data for PM10 and CO, so the sample sizes are smaller in these columns. Despite the fact that the sample in column 5 is about one-third smaller than in column 3, the coefficient on air lead concentration is nearly identical – 1.725 vs. 1.676.

One possible concern is that lead is co-emitted with other chemicals and so the coefficient on lead captures the effect of lead and other chemicals. Column 6 adds metals emissions (excluding zinc), which partially overlap with HAPs but are particularly likely to be co-emitted with lead given the nature of the industries in our sample. Compared to the previous columns, the F-stat is somewhat lower but still above 10. The coefficient on air lead concentration is also higher, though not statistically significantly different from previous estimates.

In column 7, we see the estimate is still positive but noisier with the inclusion of zinc. We cannot rule out that it is statistically similar to our main estimate. Zinc tends to be co-emitted with lead – ores tend to have both – and air fugitive lead and air fugitive zinc are relatively highly correlated (0.58). The imprecise estimate is likely due to multicollinearity.

Although our estimates for lead may capture the effect of zinc and lead, the adverse effects are very likely driven by lead. Lead is a toxin and has adverse effects on a range of bodily functions. In contrast, zinc is essential for a wide range of enzymatic and structural

functions. The EPA’s RSEI model assigns a high toxicity score to lead inhalation (23,000) and a low toxicity score to zinc (100). Consistent with this the CDC has extensive guidance on reducing lead exposure, while guidance for zinc primarily involves ensuring that there is adequate nutritional intake.<sup>30</sup>

Appendix Table A.7 presents a series of additional robustness checks. Column 1 replicates our preferred specification from column 5 of Table 3. Column 2 drops counties with plants that always report zero fugitive lead emissions. Column 3 controls for emissions from other non-lead emitting plants in the county. Column 4 requires that all counties have at least 10 monitor-years of data. It is well-known that EPA monitoring data are quite unbalanced due to the entry and exit of pollution monitors over time. In columns 5 and 6, we shorten the sample period by five and ten years. Much of the variation occurs in the early part of the sample period, so the question is whether the coefficients differ with a shorter sample period. The coefficients on air lead concentration in columns 2-6 are similar to the baseline estimate in column 1 in sign, magnitude, and significance.

## 5.5 Infant Deaths Averted

In Table 7, we use our estimates from Table 3 to do back of the envelope calculations of the number of infant deaths averted. We use county-specific realized declines in fugitive lead emissions to estimate the effects on air lead concentration and on infant mortality. To reduce the reliance on any one year, the comparison is between average county-specific fugitive emissions over 1988-1991 and 2015-2018.

In addition to using our main sample of 127 counties, we also use a sample of 227 counties to calculate the infant deaths averted. As we mentioned in Section 4, one could in principle use a two-sample IV approach to calculate the effects for all 1,227 counties with TRI lead plants. Our concern is that the emissions in most of these counties are low – the average annual emissions for the 1,227 counties are less than 25% of average emissions in the 127

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<sup>30</sup>The CDC has guidance on occupational exposure to zinc only to very high levels of zinc fumes.

counties in our IV sample. Thus, many of the counties in the larger sample are at a very different point on the dose-response curve, which may or may not be linear. There are, however, an additional 100 counties that have TRI plants and lead monitors within two miles of the plant but do not have wind monitors within 10 miles of the plants. Average fugitive and total emissions in the sample of 227 (=127+100) counties are 1,250 pounds and 4,280 pounds. These values are very similar to average emissions in the IV sample of 127 counties, which are 1,410 and 4,420 pounds. Further, estimates of the effect air fugitive emissions on child blood level in the sample of 227 counties (0.748\*) are very similar to the estimates in column 4 of Table 1 (0.805\*\*). This allows us to calculate the effects in counties representing 44% of the U.S. population.

Table 7 summarizes the infant deaths averted and the value of these lives saved. The annual number of births in the 227 counties is 1.72 million. Declines in lead emissions prevented 307 infant deaths in these counties and all declines in air lead prevented 412 infant deaths. At the EPA valuation of \$11.3 million per death averted in 2023 USD, the benefits associated with these infant lives saved are \$3.5 billion per year for lead emissions and \$4.7 billion per year for all airborne lead.<sup>31</sup>

## 6 Conclusion

This paper provides national IV estimates of the effects of air lead concentration on infant mortality in the United States over the period 1988-2018. Our identification overcomes the challenges associated with avoidance behavior, omitted variable bias, and measurement error by instrumenting for EPA air lead concentration with TRI fugitive lead emissions *interacted* with wind speed near the emitting plants and including a rich set of controls. We provide evidence that fugitive lead emissions and wind speeds are strongly linked to air lead concentration readings of EPA monitors.

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<sup>31</sup>The value of a statistical life that the EPA uses is \$7.4 million (2006 USD). This is \$11.3 million (2023 USD).

The paper has two main findings. First, we find a positive and statistically significant relationship between air lead concentration and infant mortality. Accounting for the timing of lead exposure in utero increases the magnitude of the estimates. Second, cause of death data show that lead increases deaths from low birthweight and sudden unexplained infant death.

Back of the envelope calculations indicate that declines in lead emissions avoided 307 infant deaths annually generating benefits of \$3.5 billion annually and the full decline in air lead concentration from all sources avoided 412 infant deaths annually generating benefits of \$4.7 billion per year. These estimates are based on a sample of 227 counties representing 44% of the U.S. population and so are a lower bound on avoided infant deaths. In addition, the societal benefits are likely substantially larger given that lead exposure causes morbidity and mortality in other populations beyond infants and has lasting impacts on child development.

Returning to the broader picture, air emissions of lead from industry and some other sectors such as aviation (Zahran et al., 2017; Zahran et al., 2023) and legacy lead in soil (Mielke and Reagan, 1998; Mielke et al., 2019; Klemick et al., 2020), continue to impact millions in the United States and globally. In the U.S., industrial firms and the aviation industry still emit hundreds of thousands of pounds of lead into the air. These new estimates can inform investments in reductions in air lead emissions and soil cleanups.

For developing countries where lead emissions remain high or are increasing, the U.S. experience indicates that early action can bring substantial and relatively rapid public health gains. The strong association we find between industrial emissions and infant mortality in the U.S. context suggests that similar patterns may arise elsewhere, particularly in rapidly industrializing regions. The role of monitoring systems in supporting U.S. emission reductions points to the potential importance of building capacity to track emissions and air lead concentrations. Finally, the persistence of legacy contamination in the U.S. shows that historical emissions can continue to affect populations for decades, implying that the timing of interventions may shape both the magnitude of health impacts and the eventual costs of

remediation.

## References

- Abadie, Alberto, Susan Athey, Guido W. Imbens, and Jeffrey M. Wooldridge, “When Should You Adjust Standard Errors for Clustering?,” *Quarterly Journal of Economics*, 2023, 138 (1), 1–35.
- Agarwal, Nikhil, Chanont Banternghansa, and Linda T.M. Bui, “Toxic exposure in America: Estimating fetal and infant health outcomes from 14 years of TRI reporting,” *Journal of Health Economics*, 2010, 29 (4), 557–574.
- Aizer, Anna and Janet Currie, “Lead and Juvenile Delinquency: New Evidence from Linked Birth, School, and Juvenile Detention Records,” *The Review of Economics and Statistics*, 2019, 101 (4), 575–587.
- , —, Peter Simon, and Patrick Vivier, “Do Low Levels of Blood Lead Reduce Children’s Future Test Scores?,” *American Economic Journal: Applied Economics*, 2018, 10 (1), 307–41.
- Alexander, Diane and Hannes Schwandt, “The Impact of Car Pollution on Infant and Child Health: Evidence from Emissions Cheating,” *The Review of Economic Studies*, 2022, 89 (6), 2872–2910.
- Aprioku, JS and IM Siminialayi, “Maternal lead exposure and pregnancy outcome in Wistar albino rats,” *Journal of Toxicology and Environmental Health Sciences*, 2013, 5 (10), 185–193.
- Arceo, Eva, Rema Hanna, and Paulina Oliva, “Does the effect of pollution on infant mortality differ between developing and developed countries? Evidence from Mexico City,” *The Economic Journal*, 2016, 126 (591), 257–280.
- Brehm, John and James T Hamilton, “Noncompliance in Environmental Reporting : Are Violators Ignorant, or Evasive, of the Law?,” *Journal of Political Science*, 1996, 40 (2), 444–477.
- Bui, Linda T.M., Ron Shadbegian, Alicia Marquez, Heather Klemick, and Dennis Guignet, “Does short-term, airborne lead exposure during pregnancy affect birth outcomes? Quasi-experimental evidence from NASCAR’s deleading policy,” *Environment International*, 2022, 166, 107354.
- Burki, Talha, “Report says 815 million children have high blood lead levels,” *The Lancet*, 2020, 396 (10248), 370.
- Cesur, Resul, Erdal Tekin, and Aydogan Ulker, “Air pollution and infant mortality: evidence from the expansion of natural gas infrastructure,” *The economic journal*, 2017, 127 (600), 330–362.
- Chay, Kenneth Y. and Michael Greenstone, “The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession,” *Quarterly Journal of Economics*, 2003, 118 (3), 1121–1167.

- Chibowska, Karina, Jan Korbecki, Izabela Gutowska, Emilia Metryka, Maciej Tarnowski, Marta Goschorska, Katarzyna Barczak, Dariusz Chlubek, and Irena Baranowska-Bosiacka**, “Pre-and neonatal exposure to lead (Pb) induces neuroinflammation in the forebrain cortex, hippocampus and cerebellum of rat pups,” *International Journal of Molecular Sciences*, 2020, *21* (3), 1083.
- Clay, Karen, Alex Hollingsworth, and Edson Severnini**, “The Impact of Lead Exposure on Fertility, Infant Mortality, and Infant Birth Outcomes,” *Review of Environmental Economics and Policy*, 2024, *18* (2), 301–320.
- , **Werner Troesken, and Michael Haines**, “Lead and Mortality,” *Review of Economics and Statistics*, 2014, *96* (3), 458–470.
- Currie, Janet and Johannes F. Schmieder**, “Fetal exposures to toxic releases and infant health,” *American Economic Review: Papers & Proceedings*, 2009, *99* (2), 177–183.
- **and Matthew Neidell**, “Air pollution and infant health: What can we learn from California’s recent experience?,” *Quarterly Journal of Economics*, 2005, *120* (3), 1003–1030.
- , **Joshua Graff Zivin, Jamie Mullins, and Matthew Neidell**, “What Do We Know About Short- and Long-term Effects of Early-life Exposure to Pollution?,” *Annual Review of Resource Economics*, 2014, *6* (1), 217–247.
- , **Lucas Davis, Michael Greenstone, and Reed Walker**, “Environmental Health Risks and Housing Values : Evidence from 1600 Toxic Plant Openings and Closings,” *American Economic Review*, 2015, *105* (2), 678–709.
- Dave, Dhaval M. and Muzhe Yang**, “Lead in Drinking Water and Birth Outcomes: A Tale of Two Water Treatment Plants,” *Journal of Health Economics*, 2022, *84*, 102644.
- Deryugina, Tatyana, Garth Heutel, Nolan H. Miller, David Molitor, and Julian Reif**, “The mortality and medical costs of air pollution: Evidence from changes in wind direction,” *American Economic Review*, 2019, *109* (12), 4178–4219.
- Dyatlov, Vladimir A and David A Lawrence**, “Neonatal Lead Exposure Potentiates Sickness Behavior Induced by *Listeria monocytogenes* Infection of Mice,” *Brain, Behavior, and Immunity*, 2002, *16* (4), 477–492.
- Edwards, Marc**, “Fetal death and reduced birth rates associated with exposure to lead-contaminated drinking water,” *Environmental science & technology*, 2014, *48* (1), 739–746.
- EPA**, “Estimating and Controlling Fugitive Lead Emissions from Industrial Sources,” Technical Report 1994.
- , “Locating and Estimating Air Emissions From Lead and Lead Compounds,” Technical Report EPA-454/R-98-006, US Environmental Protection Agency 1998.
- , “Lead Emissions,” Technical Report, US Environmental Protection Agency 2018.



- , “TOXICS RELEASE INVENTORY Guidance for Reporting the Dioxin and Dioxin-like Compounds Category,” Technical Report 2020.
  - , “Toxics Release Inventory: Guidance for Reporting the Lead and Lead Compounds Category,” [https://ordspub.epa.gov/ords/guideme\\_ext/f?p=guideme:gd::::gd:lead\\_guidance](https://ordspub.epa.gov/ords/guideme_ext/f?p=guideme:gd::::gd:lead_guidance) 2020. Accessed: 2023-02-06.
  - , “Air Releases,” 2023. <https://www.epa.gov/trinationalanalysis/air-releases> [Accessed: 2023-09-30].
- Erickson, Marilyn M, Alphonse Poklis, George E Gantner, Allan W Dickinson, and Laura S Hillman**, “Tissue mineral levels in victims of sudden infant death syndrome I. Toxic metals—lead and cadmium,” *Pediatric research*, 1983, *17* (10), 779–784.
- Greenstone, Michael and Rema Hanna**, “Environmental regulations, air and water pollution, and infant mortality in India,” *American Economic Review*, 2014, *104* (10), 3038–3072.
- Gutierrez, Emilio**, “Air quality and infant mortality in Mexico: evidence from variation in pollution concentrations caused by the usage of small-scale power plants,” *Journal of Population Economics*, 2015, *28* (4), 1181–1207.
- Heft-Neal, Sam, Carlos F. Gould, Marissa L. Childs, Mathew V. Kiang, Kari C. Nadeau, Mark Duggan, Eran Bendavid, and Marshall Burke**, “Emergency Department Visits Respond Nonlinearly to Wildfire Smoke,” *Proceedings of the National Academy of Sciences*, 2023, *120* (39), e2302409120.
- Hollingsworth, Alex and Ivan Rudik**, “The effect of leaded gasoline on elderly mortality: Evidence from regulatory exemptions,” *American Economic Journal: Economic Policy*, 2021, *13* (3), 345–73.
- Hsu, Chung-Yao, Yao-Chung Chuang, Fang-Chia Chang, Hung-Yi Chuang, Terry Ting-Yu Chiou, and Chien-Te Lee**, “Disrupted sleep homeostasis and altered expressions of clock genes in rats with chronic lead exposure,” *Toxics*, 2021, *9* (9), 217.
- Jansen, Erica C, Galit Levi Dunietz, Aleena Dababneh, Karen E Peterson, Ronald D Chervin, Jonggyu Baek, Louise O’Brien, Peter XK Song, Alejandra Cantoral, Howard Hu et al.**, “Cumulative childhood lead levels in relation to sleep during adolescence,” *Journal of Clinical Sleep Medicine*, 2019, *15* (10), 1443–1449.
- Kato, Ineko, Patricia Franco, Jose Groswasser, Sonia Scaillet, Igor Kelmanson, Hajime Togari, and Andre Kahn**, “Incomplete arousal processes in infants who were victims of sudden death,” *American journal of respiratory and critical care medicine*, 2003, *168* (11), 1298–1303.
- Klemick, Heather, Henry Mason, and Karen Sullivan**, “Superfund cleanups and children’s lead exposure,” *Journal of Environmental Economics and Management*, 2020, *100*, 102289.

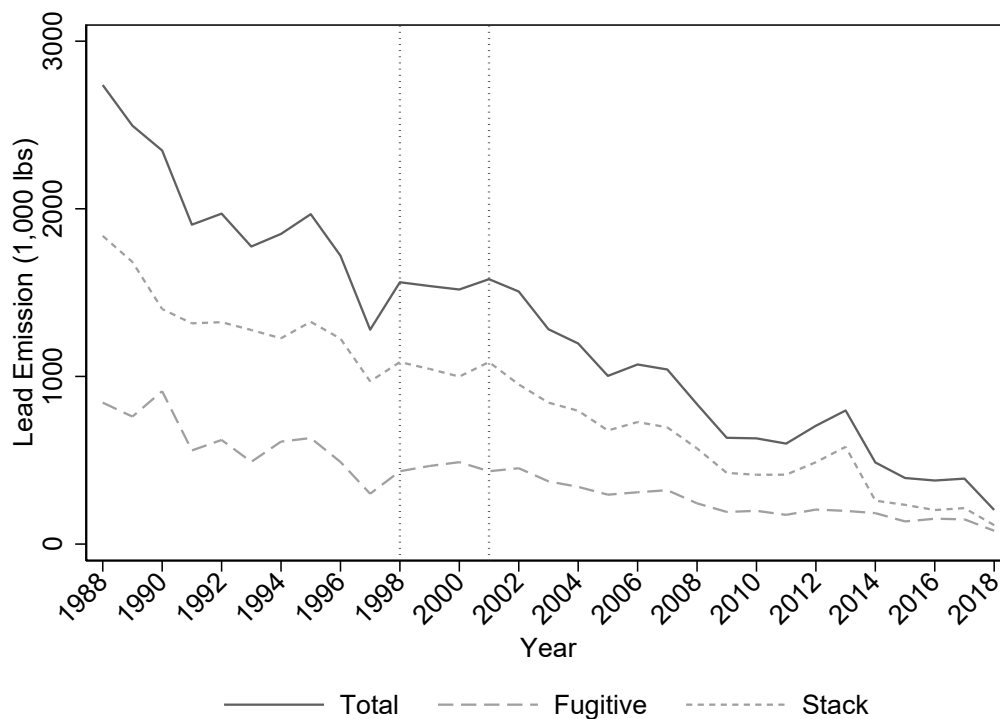
- Knittel, Christopher R., Douglas L. Miller, and Nicholas J. Sanders**, “Caution, Drivers! Children Present: Traffic, Pollution, and Infant Health,” *Review of Economics and Statistics*, 2016, 98 (2), 350–366.
- Lanphear, Bruce P, David A Burgoon, Steven W Rust, Shirley Eberly, and Warren Galke**, “Environmental exposures to lead and urban children’s blood lead levels,” *Environmental Research*, 1998, 76 (2), 120–130.
- , **Stephen Rauch, Peggy Auinger, Ryan W Allen, and Richard W Hornung**, “Low-level lead exposure and mortality in US adults: a population-based cohort study,” *The Lancet Public Health*, 2018, 3 (4), e177–e184.
- Li, Qian, Hongguang Cheng, Tan Zhou, Chunye Lin, and Shu Guo**, “The estimated atmospheric lead emissions in China, 1990–2009,” *Atmospheric Environment*, 2012, 60, 1–8.
- Liu, Jianghong, Xianchen Liu, Victoria Pak, Yingjie Wang, Chonghuai Yan, Jennifer Pinto-Martin, and David Dinges**, “Early blood lead levels and sleep disturbance in preadolescence,” *Sleep*, 2015, 38 (12), 1869–1874.
- Luechinger, Simon**, “Air pollution and infant mortality: A natural experiment from power plant desulfurization,” *Journal of Health Economics*, 2014, 37, 219–231.
- Lustberg, Mark and Ellen Silbergeld**, “Blood lead levels and mortality,” *Archives of internal medicine*, 2002, 162 (21), 2443–2449.
- Lyngbye, Troels, Ole Noerby Hansen, Lilian Vangbert, and Philippe Grandjean**, “Lead as a cause of SIDS,” *New England Journal of Medicine*, 1985.
- Marchi, Scott De and James T. Hamilton**, “Assessing the accuracy of self-reported data: An evaluation of the toxics release inventory,” *Journal of Risk and Uncertainty*, 2006, 32 (1), 57–76.
- Mason, Lisa H, Jordan P Harp, and Dong Y Han**, “Pb neurotoxicity: neuropsychological effects of lead toxicity,” *BioMed research international*, 2014, 2014.
- Maxwell, John W, Thomas P Lyon, and Steven C Hackett**, “Self-regulation and social welfare: The political economy of corporate environmentalism,” *The Journal of Law and Economics*, 2000, 43 (2), 583–618.
- Menke, Andy, Paul Muntner, Vecihi Batuman, Ellen K Silbergeld, and Eliseo Guallar**, “Blood lead below 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dL}$ ) and mortality among US adults,” *Circulation*, 2006, 114 (13), 1388–1394.
- Metryka, Emilia, Karina Chibowska, Izabela Gutowska, Anna Falkowska, Patrycja Kupnicka, Katarzyna Barczak, Dariusz Chlubek, and Irena Baranowska-Bosiacka**, “Lead (Pb) exposure enhances expression of factors associated with inflammation,” *International journal of molecular sciences*, 2018, 19 (6), 1813.

- Mielke, H W and P L Reagan**, “Soil is an important pathway of human lead exposure.,” *Environmental Health Perspectives*, 1998, *106* (suppl 1), 217–229.
- Mielke, Howard W., Christopher R. Gonzales, Eric T. Powell, Mark A. S. Laidlaw, Kenneth J. Berry, Paul W. Mielke, and Sara Perl Egendorf**, “The concurrent decline of soil lead and children’s blood lead in New Orleans,” *Proceedings of the National Academy of Sciences*, 2019, *116* (44), 22058–22064.
- Miller, Nolan H., David Molitor, and Eric Zou**, “The Nonlinear Effects of Air Pollution on Health: Evidence from Wildfire Smoke,” *American Economic Review: Insights*, 2025, *forthcoming*.
- Moon, Rachel Y, Robert A Darnall, Lori Feldman-Winter, Michael H Goodstein, Fern R Hauck, Task Force on Sudden Infant Death Syndrome et al.**, “SIDS and other sleep-related infant deaths: evidence base for 2016 updated recommendations for a safe infant sleeping environment,” *Pediatrics*, 2016, *138* (5).
- Natan, Thomas E. and Catherine G. Miller**, “Are toxics release inventory reductions real?,” *Environmental Science and Technology*, 1998, *32* (15).
- Needleman, H. L. and D. Bellinger**, “The health effects of low level exposure to lead,” *Annual Review of Public Health*, 1991, *12*, 111–140.
- Needleman, Herbert**, “Lead Poisoning,” *Annual Review of Medicine*, 2004, *55* (1), 209–222.
- Nieustadt, Frans T.M. and Han van Dop**, *Atmospheric Turbulence and Air Pollution Modelling*, Dordrecht, Holland: D. Reidel Publishing Company, 1982.
- NTP**, “NTP Monograph on health effects of low-level lead. US Department of Health and Human Services,” 2012.
- Pirkle, James L, Joel Schwartz, J Richard Landis, and William R Harlan**, “The relationship between blood lead levels and blood pressure and its cardiovascular risk implications,” *American journal of epidemiology*, 1985, *121* (2), 246–258.
- Pope III, C. Arden, Maureen Cropper, Jay Coggins, and Aaron Cohen**, “Health Benefits of Air Pollution Abatement Policy: Role of the Shape of the Concentration–Response Function,” *Journal of the Air & Waste Management Association*, 2015, *65* (5), 516–522.
- , **Richard T. Burnett, Daniel Krewski, Michael Jerrett, Yuanli Shi, Eugenia E. Calle, and Michael J. Thun**, “Cardiovascular Mortality and Exposure to Airborne Fine Particulate Matter and Cigarette Smoke: Shape of the Exposure–Response Relationships,” *Circulation*, 2009, *120* (11), 941–948.
- Raputa, V.F. and A.A. Lezhenin**, “Estimation of the Altitude of Smoke Plumes from Satellite Images,” *Atmospheric and Oceanic Optics*, 2020, *33*, 539–544.

- Rees, Nicholas and Richard Fuller**, *The toxic truth: children’s exposure to lead pollution undermines a generation of future potential*, UNICEF, 2020.
- Reyes, Jessica Wolpaw**, “Environmental policy as social policy? the impact of childhood lead exposure on crime,” *B.E. Journal of Economic Analysis and Policy*, 2007, 7 (1).
- , “Lead exposure and behavior: Effects on antisocial and risky behavior among children and adolescents,” *Economic Inquiry*, 2015, 53 (3), 1580–1605.
- Tanaka, Shinsuke**, “Environmental regulations on air pollution in China and their impact on infant mortality,” *Journal of health economics*, 2015, 42, 90–103.
- , **Kensuke Teshima**, and **Eric Verhoogen**, “North-South Displacement Effects of Environmental Regulation: The Case of Battery Recycling,” *American Economic Review: Insights*, 2022, 4 (3), 271–88.
- Thind, Inderjit S. and M. Yusuf Khan**, “Potentiation of the neurovirulence of langat virus infection by lead intoxication in mice,” *Experimental and Molecular Pathology*, 1978, 29 (3), 342–347.
- Troesken, Werner**, “Lead Water Pipes and Infant Mortality at the Turn of the Twentieth Century,” *Journal of Human Resources*, 2008, 43 (3), 553–575.
- USEPA**, “Tall stacks, various atmospheric phenomena and related aspects,” *U.S. Department of Health, Education, and Welfare Public Health Service: Consumer Protection and Environmental Health Service*, 1968.
- USEPA, U.S. Environmental Protection Agency**, “Review of the National Ambient Air Quality Standards for Lead,” <https://www.govinfo.gov/content/pkg/FR-2016-10-18/pdf/2016-24270.pdf> 2016. Federal Register, Vol. 81, No. 201, pp. 71906–71952, October 18, 2016.
- Wang, Rui, Xi Chen, and Xun Li**, “Something in the Pipe: The Flint Water Crisis and Health at Birth,” *Journal of Population Economics*, 2022, 35 (4), 1723–1749.
- Willis, G.E. and J.W. Deardorff**, “On plume rise within a convective boundary layer,” *Atmospheric Environment (1967)*, 1983, 17 (12), 2435–2447.
- Zahran, Sammy, Christopher Keyes, and Bruce Lanphear**, “Leaded aviation gasoline exposure risk and child blood lead levels,” *PNAS Nexus*, 2023, 2 (1), pgac285.
- , **Terrence Iverson, Shawn P. McElmurry, and Stephan Weiler**, “The effect of leaded aviation gasoline on blood lead in children,” *Journal of the Association of Environmental and Resource Economists*, 2017, 4 (2), 575–610.

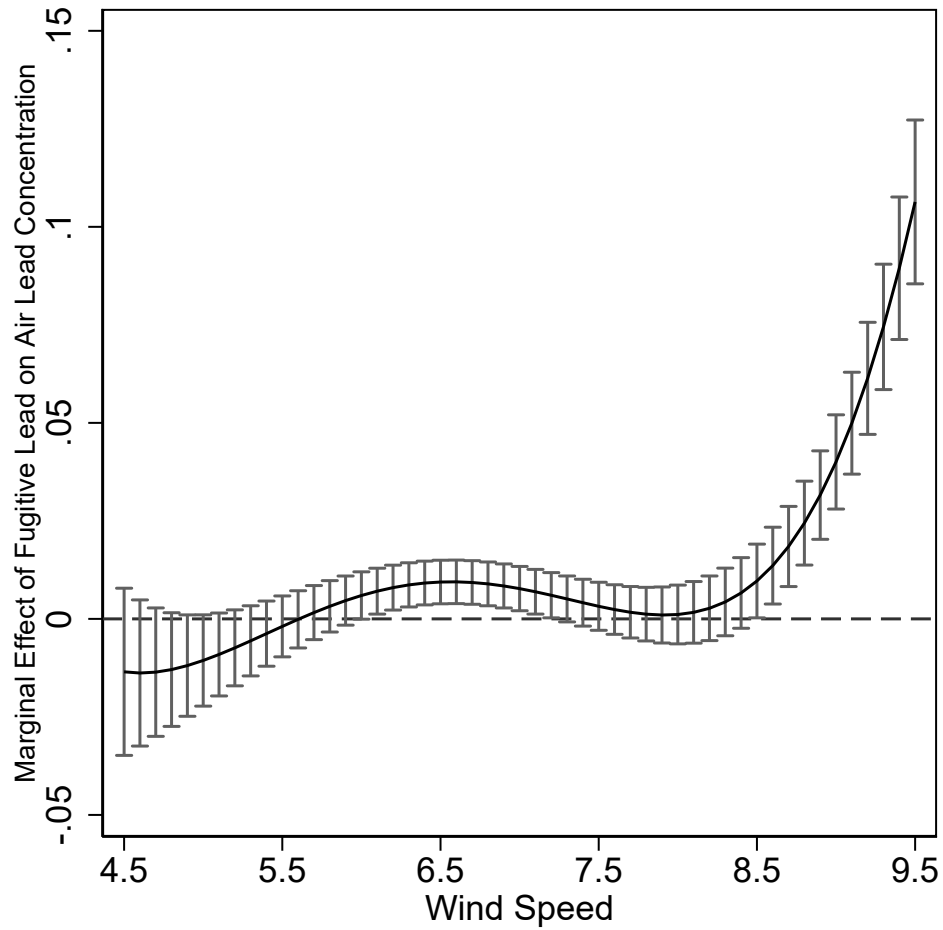
## Tables and Figures

Figure 1: Trends in Fugitive and Stack Emissions



*Notes:* This figure shows the trend of fugitive, stack, and total air lead emissions reported by TRI plants during 1988 to 2018. The vertical lines mark year 1998 when seven additional industries were added to TRI and year 2001 when the threshold for lead reporting was significantly lowered. Appendix Figure A.3 shows the number of reporting plants and changes that their inclusion have on reported totals. Appendix Figure A.2 shows trends in airborne, waterborne, landborne, and recycled lead.

Figure 2: Air Fugitive Lead and Air Lead Concentration with Controls



*Notes:* The figure plots the marginal effect of fugitive lead emissions ( $F_{ct}$ ) of plants on the air lead concentration readings at lead monitors under different wind speed. The figure is obtained from the first-stage regression of the IV estimation specified in equation 1.

Table 1: Child Blood Lead Level and Industrial Lead Emission

	Pct Elevated BLL			
	(1)	(2)	(3)	(4)
Air Fugitive Lead	0.763** (0.363)	0.837* (0.485)	0.898* (0.521)	0.805** (0.399)
Air Stack Lead	0.057 (0.062)	0.049 (0.044)	0.049 (0.047)	0.048 (0.050)
Adjusted $R^2$	0.989	0.989	0.989	0.991
Dep Var Mean	6.572			
Fug Mean[S.D.]	0.520[1.094]			
CountyYear	365	365	365	365
Counties	57	57	57	57
County,Region-Year FE	Y	Y	Y	Y
Other Chem	Y	Y	Y	Y
Base IMR		Y	Y	Y
Socioeconomic,Mother		Y	Y	Y
Climate Var			Y	Y
Water,Land Lead				Y

*Notes:* This table reports the regressions on child blood lead level. The dependent variable is the percentage of children in a county with elevated blood lead level ( $\geq 10\mu\text{g}/\text{dL}$ ) conditional on being tested. The key explanatory variables are county aggregated air fugitive and air stack lead emissions from industrial plants. Control variables are the same as in Table 3. The regression is weighted by county population. The sample is a subset of the IV sample with the county-years having child blood lead level data being included. The data on child blood lead level run from year 2005 to 2015. Data are obtained from Hollingsworth and Rudik (2021).

Table 2: Fugitive Lead Emissions and Daily Variation of Ambient Lead Concentration

	Dep Var: S.D. Daily Ambient Lead Concentration				
	(1)	(2)	(3)	(4)	(5)
High Frac Fugitive Lead	0.144*** (0.046)	0.140*** (0.042)	0.139*** (0.043)	0.138*** (0.042)	0.138*** (0.042)
Mean Dep Var	0.227	0.227	0.227	0.227	0.227
Adjusted $R^2$	0.692	0.694	0.696	0.696	0.696
Monitor-Year	3015	3015	3015	3015	3015
Monitors	352	352	352	352	352
Counties	127	127	127	127	127
Monitor, Region-Year FE	Y	Y	Y	Y	Y
Air, Water, Land Lead		Y	Y	Y	Y
Socio-economic			Y	Y	Y
Climate Var				Y	Y
Other Emissions					Y

*Notes:* This table reports the results for regressing standard deviation of daily ambient lead concentration within 2 miles of lead plants on the fraction of fugitive over total air lead emissions of the plants. Variable *High Frac Fugitive Lead* is an indicator for monitor-years that have above-median fraction of fugitive over total air lead. Mean number of days for calculating the standard deviation of monitoring data in a year is 213. We control for monitor fixed effects, region-by-year fixed effects in all specifications. We test for robustness including the level of air, water, land-borne lead emissions from plants, county socio-economic characteristics (population density, percent white, percent high school degree, median household income, percent manufacturing employment, and employment rate), county climate (annual total precipitation, annual average temperature, and wind speed), and other toxic emissions from lead and non-lead plants in the county. Monitor-years with positive air lead emissions and ambient lead concentration are included for the regressions. Standard errors are clusters at county level.



Table 3: IV Estimates of Air Lead Concentration and Infant Mortality

	(1)	(2)	(3)	(4)	(5)
	IMR	IMR	IMR	IMR	IMR
Air Lead Concentration	1.998*** (0.429)	1.753*** (0.453)	1.717*** (0.447)	1.685*** (0.395)	1.676*** (0.435)
KPFstat	41.983	40.252	38.029	45.036	41.232
DepMean	7.718	7.718	7.718	7.718	7.718
County-Year	1553	1553	1553	1553	1553
Counties	127	127	127	127	127
County,Region-by-Year FE	Y	Y	Y	Y	Y
Base IMR		Y	Y	Y	Y
Socioeconomic,Mother		Y	Y	Y	Y
Climate Var			Y	Y	Y
Water,Land Lead				Y	Y
Other Chem					Y

*Notes:* Baseline IMR is the county infant mortality rate averaged over 1980 to 1986 (the year prior to the start of TRI). Controls on other chemicals include air fugitive and stack emissions of developmental toxins and other TRI reported chemicals. Controls on socio-economic characteristics include population density, percent white, percent high school degree, median household income, percent manufacturing employment, and employment rate at county level. Data on these characteristics are from the 1990, 2000, 2010 Census and the 2018 ACS 5-year data and are interpolated to an annual panel. Controls on mothers' characteristics include county-average percent white, percent Hispanic, percent of high school degree, and percent of mothers aged over 35. Controls on climate variables include annual total precipitation and annual average temperature at the county level. Regressions are weighted by the number of births. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

Table 4: IV Estimates By Age at Death and Timing of Birth

	(1)	(2)	(3)	(4)	(5)	(6)
	IMR 1m	IMR 1y	IMR AD1m	IMR AD1y	IMR JD1m	IMR JD1y
Air Lead Concentration	0.728** (0.323)	1.676*** (0.435)	1.155*** (0.336)	2.128*** (0.430)	1.143*** (0.387)	2.660*** (0.530)
KPFstat	41.232	41.232	40.884	40.884	40.337	40.337
DepMean	5.104	7.718	5.09	7.565	4.988	7.453
County-Year	1553	1553	1553	1553	1553	1553
Counties	127	127	127	127	127	127
All Controls	Y	Y	Y	Y	Y	Y

*Notes:* The dependent variable is infant mortality in the first month or year for infants in columns 1-2. The dependent variable is infant mortality in the first month or year for infants born April to December in columns 3-4. The dependent variable is infant mortality in the first month or year for infants born July to December in columns 5-6. All controls are the controls from column 5 of Table 3. Regressions are weighted by the number of births. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

Table 5: IV Estimates By Cause

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	LowBw	SUID	Resp.	Nerv.	Cong.	Peri.	Others
<i>Panel A. IMR in the first year</i>							
Air Lead	0.349** (0.163)	0.407* (0.208)	0.419*** (0.149)	0.121** (0.051)	0.110 (0.144)	-0.238 (0.281)	0.544*** (0.120)
Dep Mean	1.172	1.113	0.890	0.112	1.541	1.880	0.834
<i>Panel B. IMR in the first month</i>							
Air Lead	0.363** (0.162)	0.020 (0.034)	0.242** (0.117)	0.059** (0.023)	0.080 (0.136)	-0.225 (0.295)	0.262*** (0.065)
Dep Mean	1.152	0.109	0.624	0.017	1.098	1.782	0.366
KP F-Stat	41.069	41.069	41.069	41.069	41.069	41.069	41.069
County-Year	1548	1548	1548	1548	1548	1548	1548
Counties	126	126	126	126	126	126	126
All Controls	Y	Y	Y	Y	Y	Y	Y

*Notes:* This table reports the IV estimates on infant mortality rate by cause of death. Column 1 reports results on infant deaths due to low birth weight, column 2 reports results on infant deaths from SUID, column 3 and 4 are for infant deaths from diseases of the respiratory system and the nervous system, respectively, column 5 is for infant deaths from congenital anomalies, column 6 is for other conditions originating in the perinatal period, and column 7 is for deaths from all other causes. All controls are the controls from column 5 of Table 3. Regressions are weighted by the number of births. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

Table 6: IV Estimates By Race of Mother

	(1)	(2)	(3)	(4)
	IMR1yrnwh	IMR1yrwh	IMR1mnwh	IMR1mwh
Air Lead Concentration	3.068** (1.212)	1.415*** (0.361)	1.977** (0.998)	0.478* (0.256)
KPFstat	39.646	41.711	39.646	41.711
DepMean	11.778	6.067	8.346	4.044
CountyYear	1552	1552	1552	1552
Counties	127	127	127	127
AllControls	Y	Y	Y	Y

*Notes:* The dependent variable is infant mortality in the first year for infants born to nonwhite (IMR1yrnwh) and white (IMR1yrwh) in columns 1 and 2. The dependent variable is infant mortality in the first month for infants born to nonwhite (IMR1mnwh) and white (IMR1mwh) in columns 3 and 4. The mean of the dependent variables differs from the summary statistics in Appendix Table A.1. In the summary statistics, the observations are weighted by all births, while here they are weighted by race-specific births. All controls are the controls from column 5 of Table 3. Regressions are weighted by the number of births to nonwhite and white mothers. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

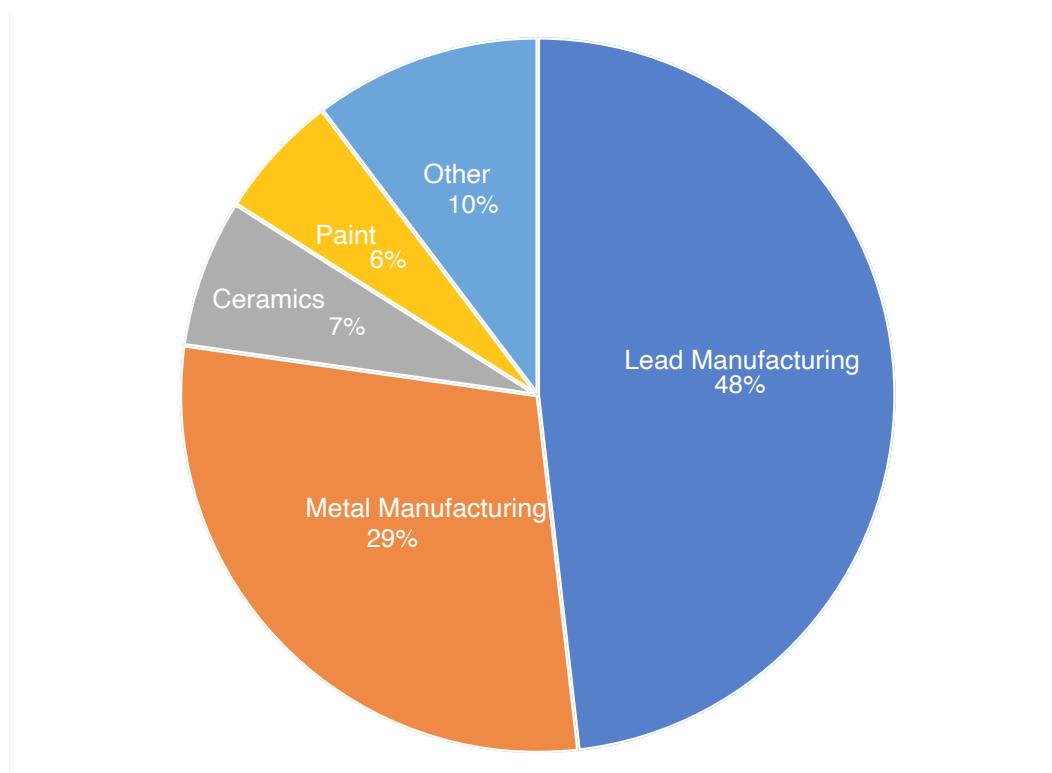
Table 7: Back of the Envelope Calculations

	(1)	(2)
	Annual Infant Deaths Averted	Value in 2023\$
IV counties only, Lead Emission	253	\$2.9 billion
IV counties only, All Lead	302	\$3.4 billion
With non-wind-monitored cnties, Lead Emis	307	\$3.5 billion
With non-wind-monitored cnties, All Lead	412	\$4.7 billion

*Notes:* The IV counties only, lead emission estimates are generated by using the average of lead emissions (fugitive + stack) from 1988-1991 and 2015-2018 in the first stage to generate the reduction in air lead concentration. This is then multiplied by the coefficient on air lead concentration in column 5 of Table 3 and the annual number of births in the counties to get the annual infant deaths averted. The IV counties only, all lead estimates are generated by using the average decline in air lead concentration from 1988-1991 and 2015-2018. This is multiplied by the coefficient on air lead concentration in column 5 of Table 3 and the annual number of births in the counties to get the annual infant deaths averted. Deaths are multiplied by the EPA valuation of \$11.3 million per death averted (2023 USD) to get values. The estimates for “with non-wind-monitored cnties” are generated using data of the 127 IV counties plus another 100 counties that have lead emitting plants and lead monitors within 2 miles from the plants but may or may not have a wind-monitor within 10 miles from the plants. Details on the counties and the calculation on deaths averted are shown in Table A.8.

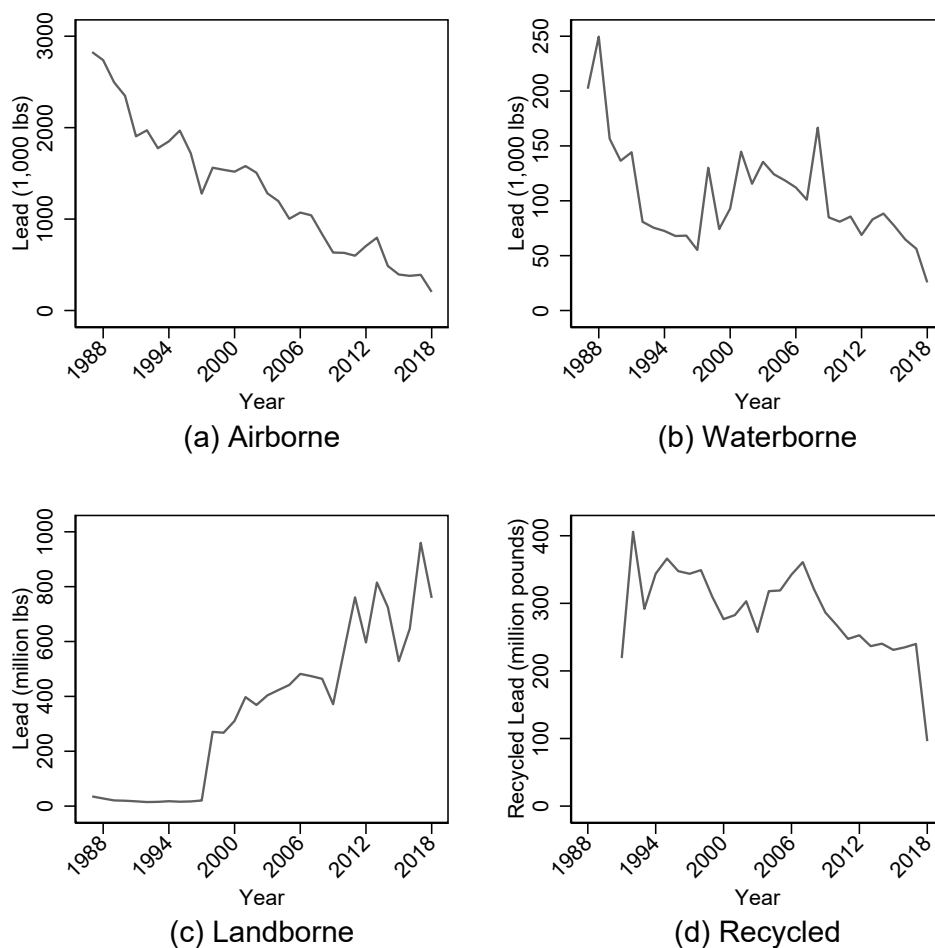
## A Supplemental Materials: Appendix Figures and Tables

Figure A.1: Industry Distribution of Air Lead Emissions of Facilities



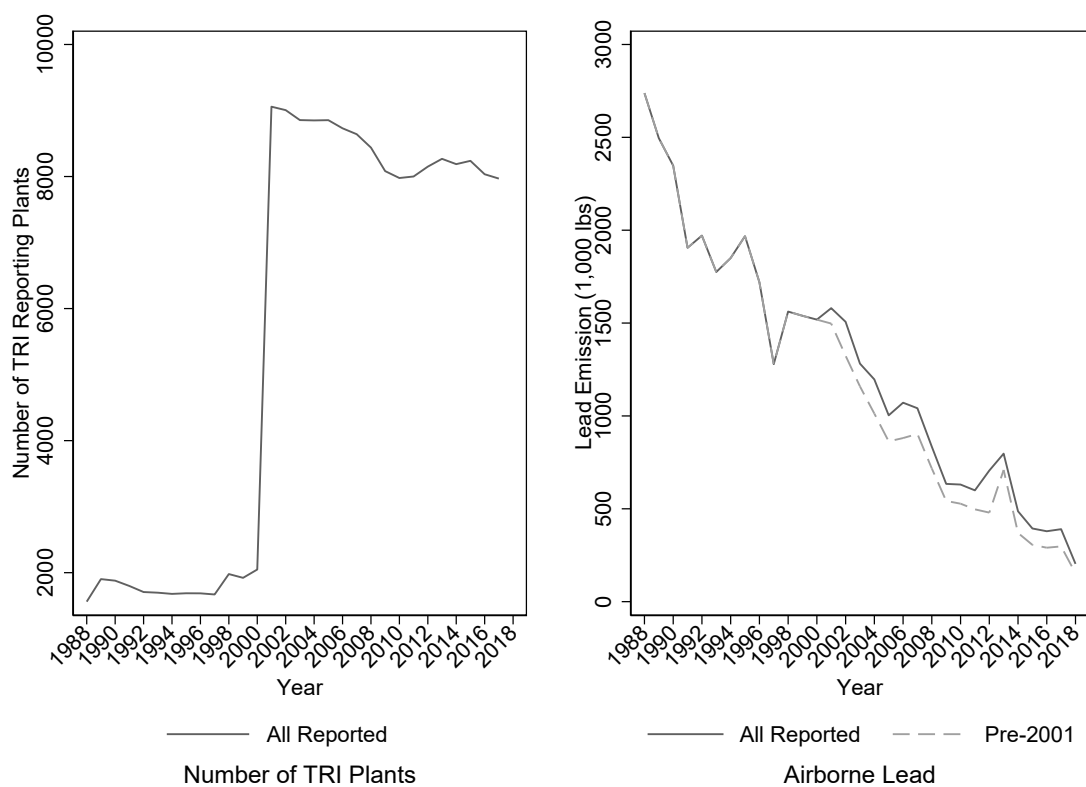
*Notes:* This pie-chart shows the industry distribution of airborne lead emissions (sum over time) by the sampling industrial facilities. The emissions include both fugitive and stack lead emissions. Calculates are weighted by the number of births in a county.

Figure A.2: Lead Emissions from Air, Water, and Land-borne Sources and Recycled Lead



*Notes:* Figure (a) to (c) plot the trends of air-, water-, and land-borne lead emissions by TRI plants. Figure (d) plots the lead recycled from production waste. Data on recycled lead started from 1991, following the Pollution Prevention Act (1990) that expanded TRI to include additional information on toxic chemicals in waste and on source reduction methods.

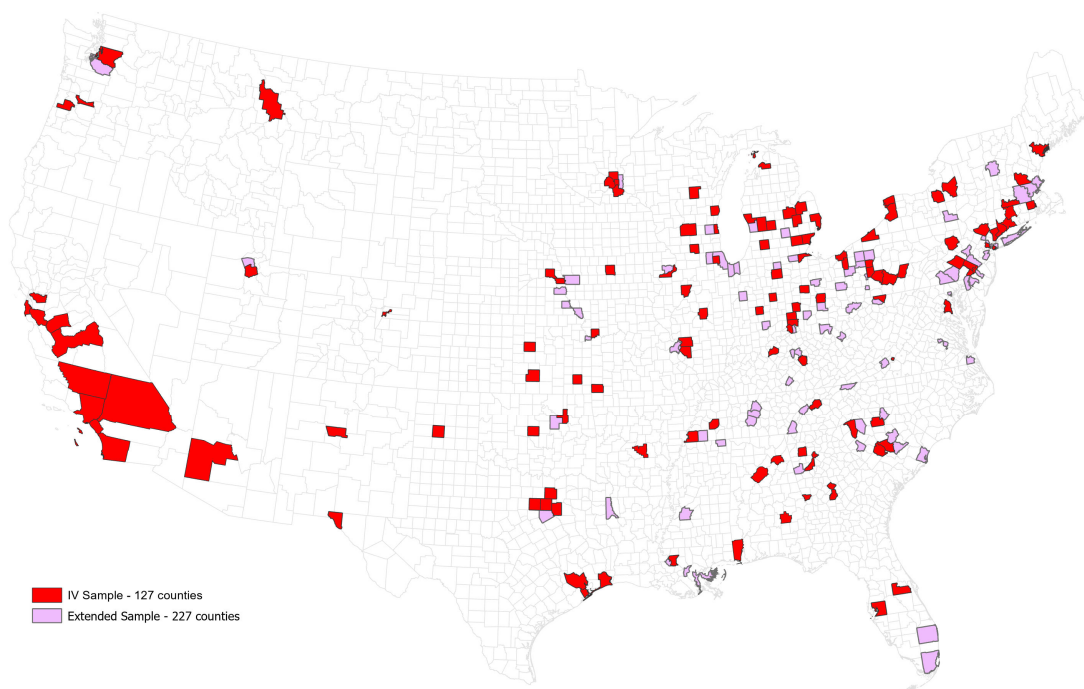
Figure A.3: Changes in Reporting in 2001



*Notes:* This figure shows trends in the number of reporting plants and their effect on air lead emissions in our sample during 1988 to 2018.

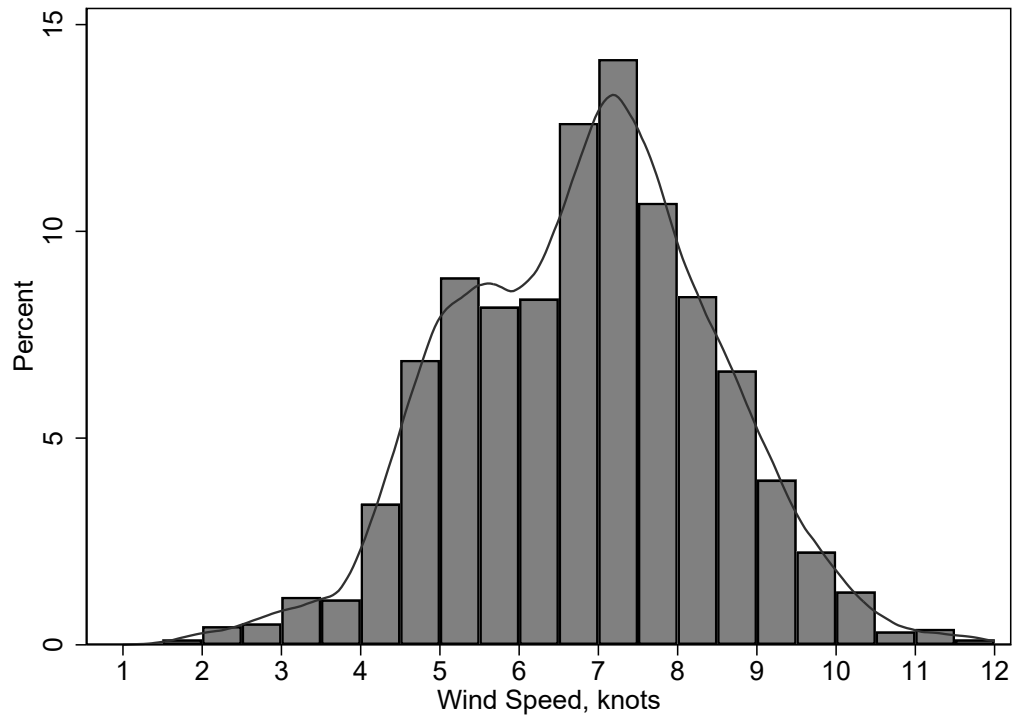


Figure A.4: Geographic Distribution of Counties in the IV Sample



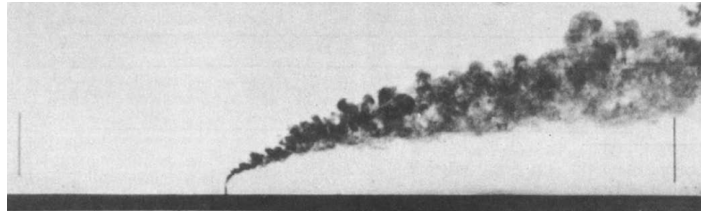
*Notes:* This map presents locations of the 127 counties in our sample (red) and the locations of the 227 counties in the extended sample (red + pink).

Figure A.5: Histogram of Wind Speeds



*Notes:* This figure plots the distribution of wind speeds using the pooled county-by-year wind data for the IV regressions.

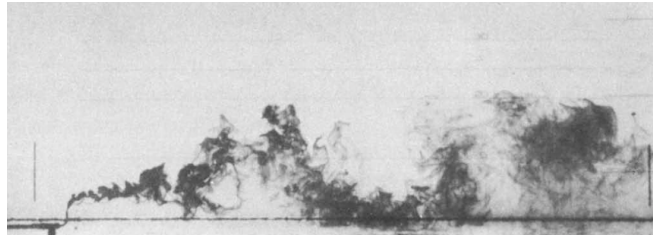
Figure A.6: Plume Patterns in the Laboratory



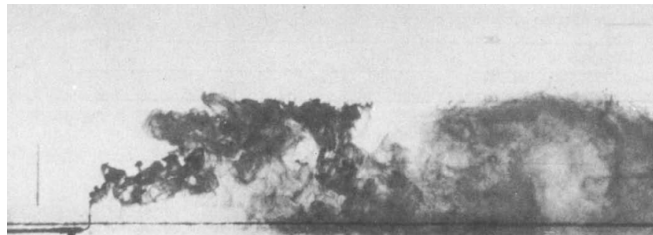
(a) Buoyant plume in a neutral, non-turbulent environment



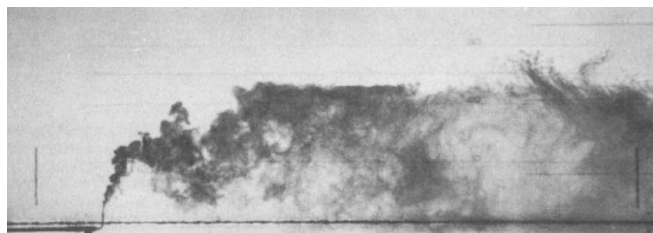
(b) Non-buoyant plume in a convectively turbulent environment



(c) Weakly buoyant plume in a convectively turbulent environment



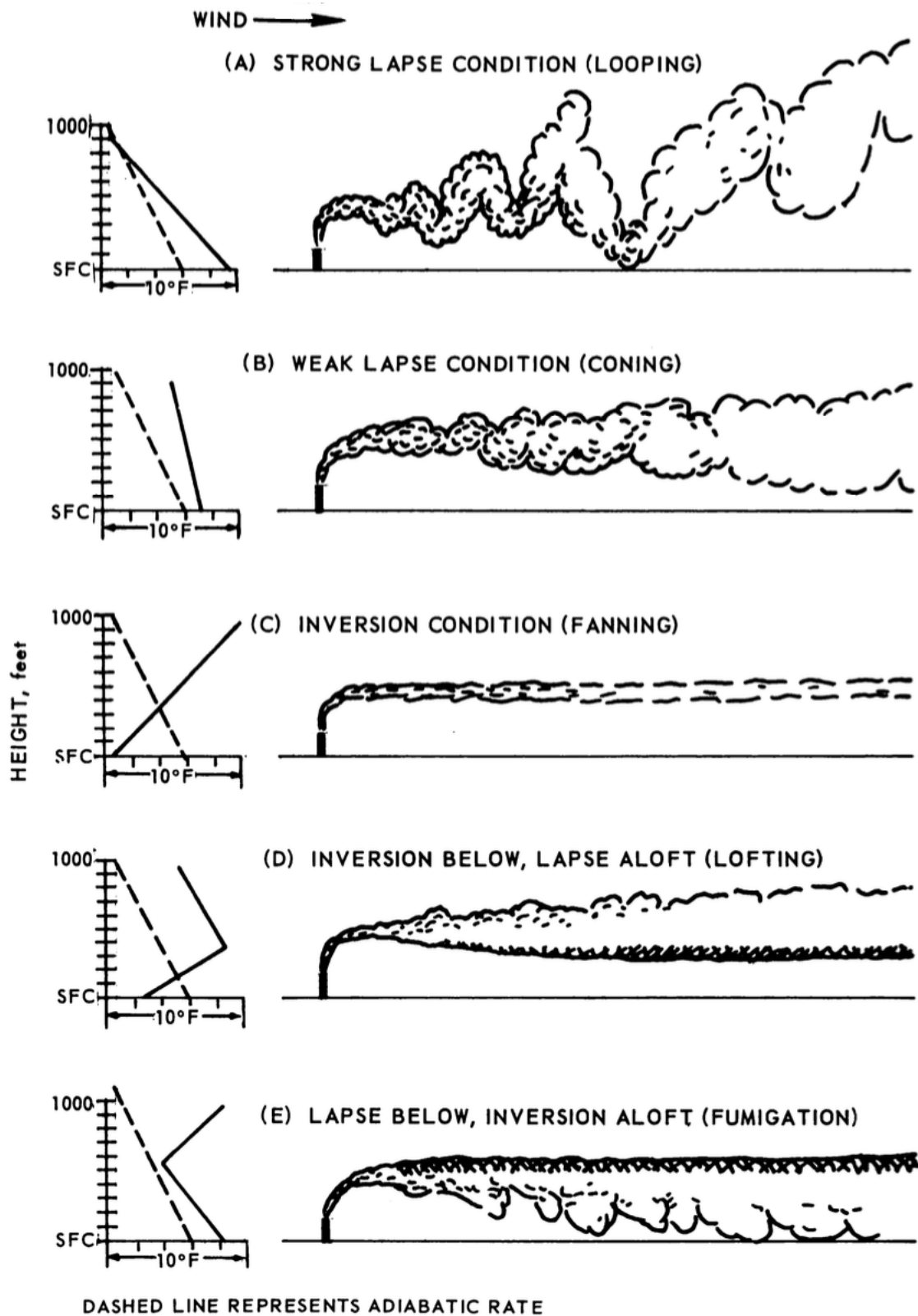
(d) Moderately buoyant plume in a convectively turbulent environment



(e) Strongly buoyant plume in a convectively turbulent environment

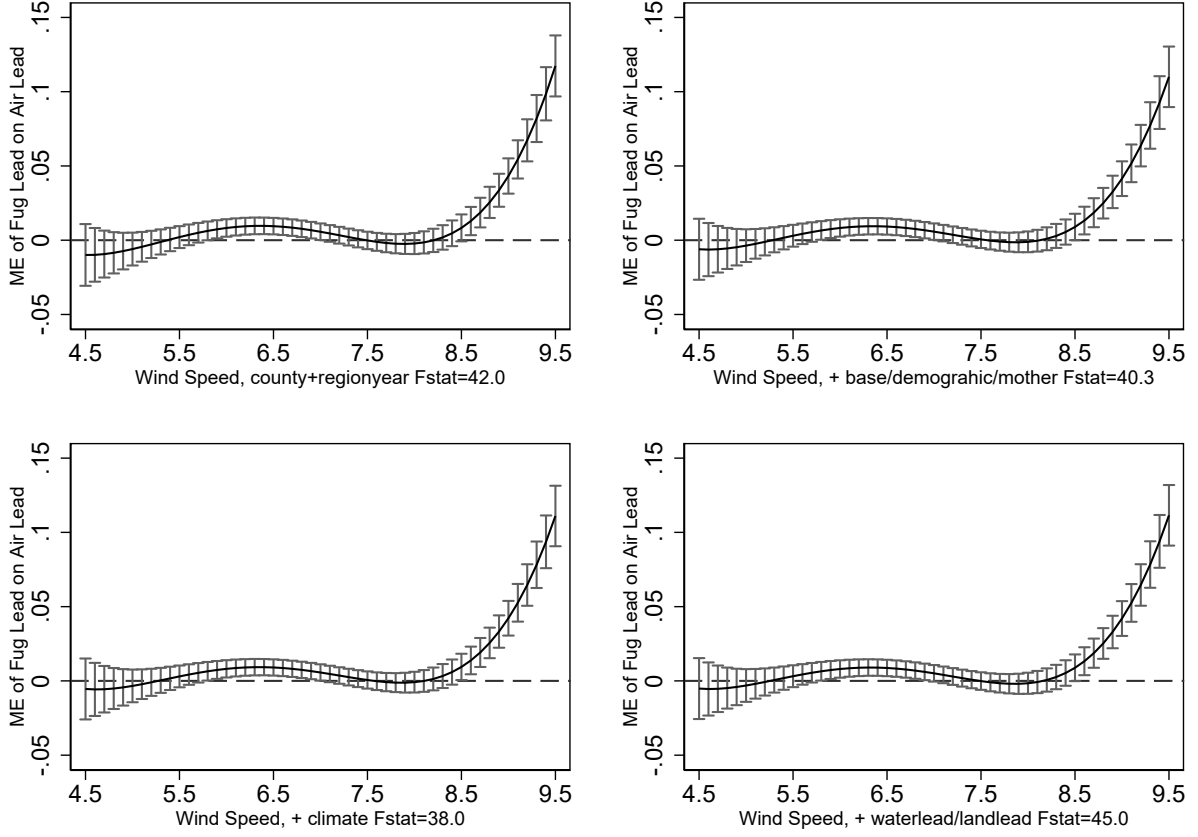
*Notes:* This figure plots potential plume patterns from a point source pollution. A convectively turbulent environment refers to a situation where convective processes lead to highly irregular and chaotic fluid motions. This movement typically happens in a circular or cyclical manner. “Convective” refers to the process of heat or mass transfer that occurs due to the movement of fluids (gases or liquids) caused by density variations within the fluid. Convection plays a crucial role in various natural processes such as air circulation in the Earth’s atmosphere and ocean currents. *Source:* Willis and Deardorff (1983).

Figure A.7: Plume Patterns



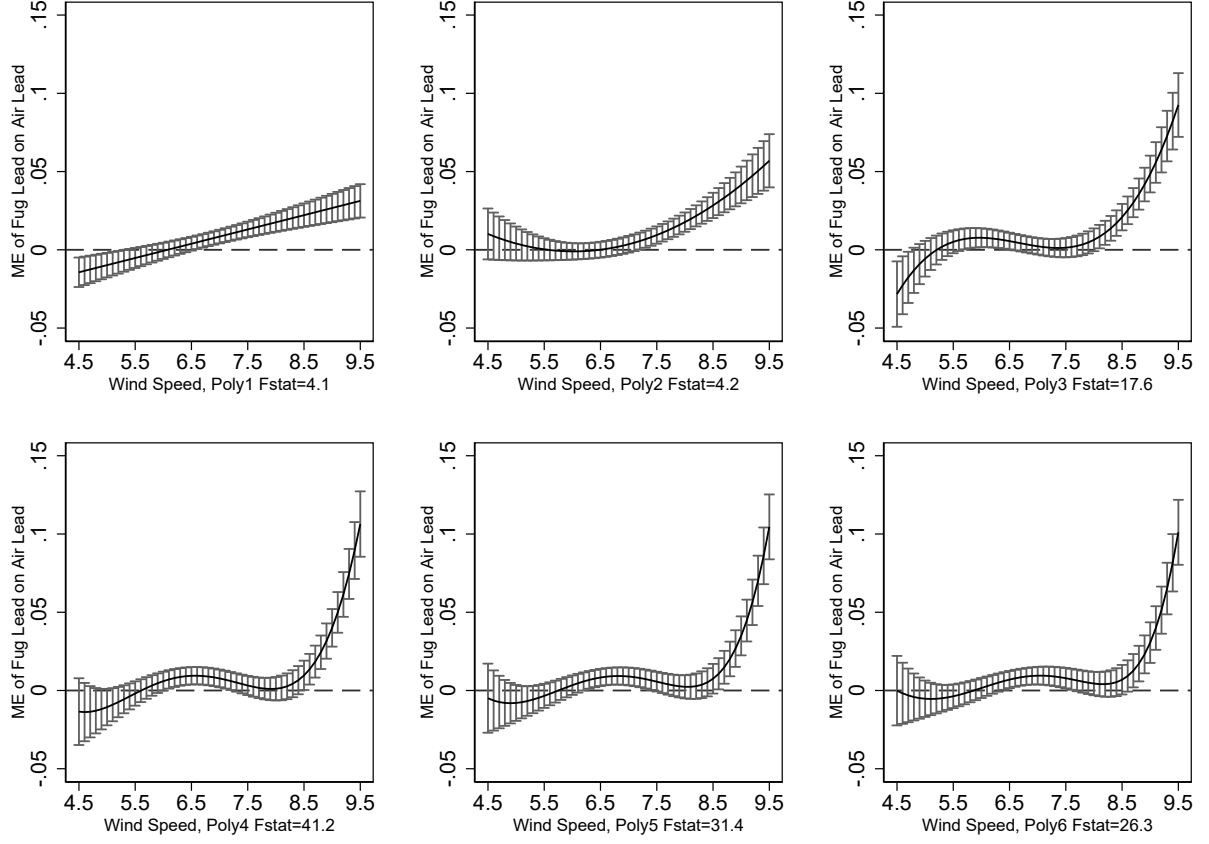
Notes: This figure plots potential plume patterns from a point source pollution. Source: USEPA (1968).

Figure A.8: First Stage Adding Controls



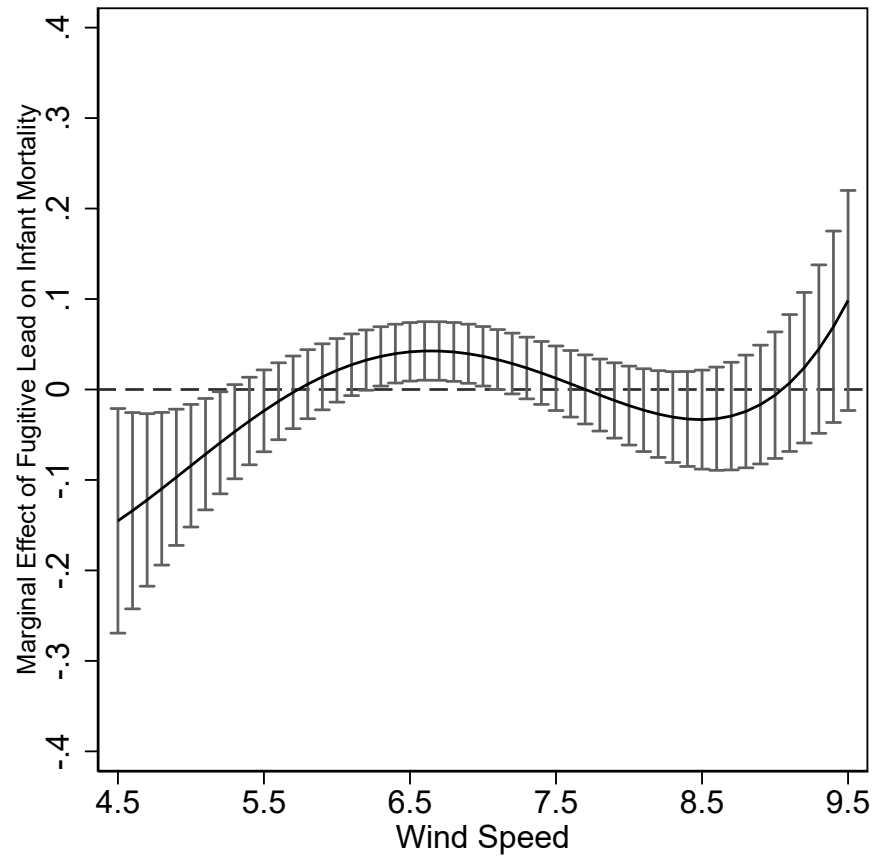
*Notes:* The figure plots the marginal effect of fugitive lead emissions ( $F_{ct}$ ) of plants on the air lead concentration readings at lead monitors as different sets of controls are added. These first stages correspond to columns 1-4 in Table 3.

Figure A.9: First Stage with Alternative Polynomials



*Notes:* The figure plots the marginal effect of fugitive lead emissions ( $F_{ct}$ ) of plants on the air lead concentration readings at lead monitors as the wind polynomial is increased from 1 to 6. These first stages include the full set of controls from columns 5 in Table 3.

Figure A.10: Reduced Form Specification



*Notes:* This figure reports the marginal effect of fugitive lead emissions on infant mortality as a function of wind speed (in knots). The specification is the reduced form version of the specification in column 5 of Table 3.

Table A.1: County Summary Statistics

	mean	sd
IMR 1yr, per 1,000	7.72	2.72
IMR 1mo, per 1,000	5.10	1.86
IMR Nonwh, per 1,000	11.10	4.90
IMR White, per 1,000	6.11	1.68
Premature, per 1,000	60.80	22.65
Birthweight, grams	3297.40	62.06
Low Bthwt, per 1,000	76.86	15.79
Births	51784.52	59008.90
Air Fug Lead, 1,000 lbs	1.71	3.79
Air Stack Lead, 1,000 lbs	4.65	12.48
Air Lead Concentration	0.08	0.22
Windspeed, knots	6.47	1.53
County Pop Density	2335.09	3693.82
County HH Income	48594.42	11813.09
County Pct Mfg Employ	14.05	4.97
County Pct Employ	91.62	2.76
County Pct White	66.07	13.86
County Pct HSchool	89.22	5.63
Mother White	0.74	0.14
Mother Hispanic	0.33	0.26
Mother Age over 35	0.13	0.05
Mother High School	0.71	0.17
Avg Temp, C	15.08	4.08
Avg Precip, Mm	797.92	471.79
Water Lead, 1,000 lbs	0.34	1.20
Land Lead, 1,000 lbs	35.53	268.35
Air Fug Dev, 1,000 lbs	39.69	113.32
Air Stack Dev, 1,000 lbs	96.69	236.71
Air Fug NDev, 1,000 lbs	274.40	751.98
Air Stack NDev, 1,000 lbs	767.85	1326.10
Air Fug HAP, 1,000 lbs	92.01	246.04
Air Stack HAP, 1,000 lbs	237.59	670.35
Number of Plants	6.22	7.34
Number of Monitors	1.98	1.76
(Within 2 miles to plants)		
Observations	1553	



Table A.2: Variation in Explanatory Variables of County Characteristics

	S.D. / Mean				
	p10	p25	p50	p75	p90
Wind Speed < 2 mi to Plants	0.05	0.06	0.08	0.11	0.15
Fug Lead	0.59	0.93	1.42	1.75	2.48
Fug Lead x Wind Speed	0.60	0.96	1.40	1.74	2.45
Stack Lead	0.43	0.72	1.09	1.66	2.45
Stack Lead x Wind Speed	0.46	0.78	1.09	1.63	2.48
Counties	127				
County-Year	1,553				

*Notes:* This table reports the variations of the key explanatory variables in the identification model - wind speed, fugitive lead emissions, stack lead emissions, and their interactions. We report the 10th, 25th, 50th 75th, and 90th percentiles of the standard deviation normalized by the mean of the variables.

Table A.3: Prediction of County Characteristics

	(1)	(2)	(3)	(4)	(5)	(6)
	MFemp	Emp	Popdens	Hinc	Pwhite	Phighsch
KPFstat	3.772	1.235	5.659	3.905	1.139	0.552
CountyYear	1553	1553	1553	1553	1553	1553
Counties	127	127	127	127	127	127
Allcontrols	Y	Y	Y	Y	Y	Y

*Notes:* The dependent variable is listed in the column header: (MFemp) percent in manufacturing employment; (Emp) percent employed; (Popdens) population density per square mile; (Hinc) median household income; (Pwhite) percent white; (Phighsch) percent with high school education over people above age 25. All controls are the controls from column 5 of Table 3, but excludes the dependent variable. F-statistics are the joint significance of fugitive lead interacted with the wind variables. Regressions are weighted by the number of births. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

Table A.4: Prediction of Maternal Characteristics

	(1)	(2)	(3)	(4)
	Pmwhite	Pmhisp	Pmolder35	Pmhighsch
KPFstat	1.568	0.630	0.783	0.686
CountyYear	1553	1553	1553	1553
Counties	127	127	127	127
Allcontrols	Y	Y	Y	Y

*Notes:* The dependent variable is listed in the column header: (Pmwhite) percent mothers white; (Pmhisp) percent mothers hispanic; (Pmolder35) percent mothers older than 35; (Pmhighsch) percent mothers with high school education. All controls are the controls from column 5 of Table 3, but excludes the dependent variable. F-statistics are the joint significance of fugitive lead interacted with the wind variables. Regressions are weighted by the number of births. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

Table A.5: IV Estimates of Other Infant Health Outcomes

	(1)	(2)	(3)	(4)
	Prem	Prem	LowBw	LowBw
	AD	JD	AD	JD
Air Lead Conc.	2.708	2.614	2.119	1.661
	(2.739)	(2.708)	(1.963)	(2.179)
KP F-Stat	40.584	39.969	40.584	39.969
Dep Mean	86.655	85.228	76.970	77.074
County-Year	1553	1553	1553	1553
Counties	127	127	127	127
All Controls	Y	Y	Y	Y

*Notes:* This table reports regressions on premature (gestation weeks  $< 37$ ) per thousand live birth and low birth weight ( $< 2,500\text{g}$ ) per thousand live birth. Means of the dependent variables are reported under the coefficients. Regressions control for the full set of other controls described in Table 3. Standard errors are clustered at county level.

Table A.6: Other Fugitive Chemicals, PM10, and CO

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	IMR	IMR	IMR	IMR	IMR	IMR	IMR
Air Lead Conc.	1.529*** (0.458)	1.546*** (0.411)	1.676*** (0.435)	1.901** (0.804)	1.725** (0.842)	2.343** (0.915)	0.940 (1.640)
KPFstat	35.778	34.202	41.232	34.107	19.051	11.120	1.497
DepMean	7.718	7.718	7.718	7.782	7.753	7.718	7.718
CountyYear	1553	1553	1553	1282	1046	1553	1553
Counties	127	127	127	105	81	127	127
Allothercontrols	Y	Y	Y	Y	Y	Y	Y
DevChem	Y	Y	Y	Y	Y	Y	Y
NonDevChem		Y	Y	Y	Y	Y	Y
HAP			Y	Y	Y	Y	Y
PM10				Y	Y		
CO					Y		
Metal2						Y	Y
Zinc							Y

*Notes:* This table reports the results of adding controls for other chemicals and pollutants. The dependent variable of all regressions are infant mortality rate within the first year of births. All other controls are the controls from column 5 of Table 3, but excludes other chemicals. Column 6 and 7 control for the aggregate emission of metals including copper, manganese, chromium, nickel, barium, antimony, arsenic, cobalt, cadmium, selenium, silver, vanadium, mercury, and their compounds. Zinc and zinc compounds are controlled separately in col 7 only because of the high correlation of their emission and lead emission. These metals either have a large amount of emission or a high toxicity score in EPA's RSEI (Risk-Screening Environmental Indicators). F-statistics are the joint significance of fugitive lead interacted with the wind variables. Regressions are weighted by the number of births. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

Table A.7: Robustness Checks

	(1) IMR Base	(2) IMR Nozerofug	(3) IMR OtherPlantE	(4) IMR 10years	(5) IMR To2013	(6) IMR To2008
Air Lead Conc.	1.676*** (0.435)	1.695*** (0.441)	1.537*** (0.455)	1.603*** (0.527)	1.798*** (0.471)	1.728*** (0.493)
KPFstat	41.232	41.280	32.545	28.348	41.261	38.382
CountyYear	1553	1534	1550	1153	1344	1112
Counties	127	122	127	57	126	106
Allcontrols	Y	Y	Y	Y	Y	Y

*Notes:* This table reports the results of several robustness exercises. The dependent variable of all regressions are infant mortality rate within the first year of births. Column 1 is the baseline (col. 5, Table 3). All controls are the controls from column 5 of Table 3. Column 2 drops counties that report stack emissions but always report zero fugitive emissions. Most of the zero-emission reporters are those not estimating their emission using scientific methods but just choosing a range of their emission amount (which is typically zero). Column 3 controls for chemical emissions of other non-lead emitting plants in the county. Column 4 only includes counties with at least 10 years of lead monitor data in the sample. Columns 5 and 6 shorten the sample from 1988-2018 to 1988-2013 and to 1988-2008. F-statistics are the joint significance of fugitive lead interacted with the wind variables. Regressions are weighted by the number of births. Standard errors clustered at county level. \*\*\* denotes statistical significance at the 1 percent level, \*\* at the 5 percent level, and \* at the 10 percent level.

Table A.8: Back of the Envelope Calculation - Details

Sample	Cnty Num.	Lead Emis. /Yr (Fug+Stack)		$\Delta$ Lead Emis.	$\Delta$ Air Lead Conc.		Annual Births	Infant Lives Saved		
		Sum	1988-91 avg	2015-18 avg	(4)-(3)	by $\Delta$ Emis.		Actual	IV, Lead Emis.	IV, All Lead
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
IV	127	6,642	8.72	0.59	-8.13	0.121	0.144	1.25m	253	302
Extended	227	11,255	8.24	1.07	-7.17	0.106	0.143	1.72m	307	412

*Notes:* The IV sample includes counties with 1) TRI lead plants, 2) lead monitors within 2 miles from lead plants, and 3) wind monitors within 10 miles from lead plants. The extended sample includes counties with 1) and 2) only. Changes in lead emission and air lead concentration are the difference between the sample mean from 2015 to 2018 and the mean from 1988 to 1991. Annual number of births are the mean across years 2015 to 2018. Population in the IV counties is 77 million in 1990, about 31% of the U.S. total. Population in the extended sample is 111 million in 1990, about 44% of the U.S. total in 1990. Change in air lead concentration induced by change in lead emission is calculated based the estimated marginal effect of fugitive lead emission on air lead concentration (0.015) in the regression in figure 2. Column 9 = Change in air lead induced by change in lead emission (col 6) x Marginal effect of air lead on IMR (1.676) x Annual births (col 8) x 1000. Column 10 = Actual change in air lead (col 7) x Marginal effect of air lead on IMR (1.676) x Annual births (col 8) x 1000.

## A.1 Further Analysis: Air Lead Emissions and Concentrations

To examine the relationship between lead emissions, wind, and distance between emitting plant and EPA monitor, we begin by estimating the following model:

$$\begin{aligned} AirLead_{mt} = & \sum_d (F_{dmt} \times Wind_{mt}) \delta_d^F + \sum_d (S_{dmt} \times Wind_{mt}) \delta_d^S \\ & + Wind_{mt} \delta_w + \eta_m + \lambda_{rt} + \nu_{mt} \end{aligned} \quad (A.1)$$

The dependent variable  $AirLead_{mt}$  is the annual mean ambient lead concentration readings from monitor  $m$  in year  $t$ .  $F_{dmt}$  and  $S_{dmt}$  are the aggregated fugitive and stack lead emissions from plants in different distances to monitor  $m$  in year  $t$ , respectively. We interact the emission variables with a fourth order polynomial for wind speed near the plants linked to monitor  $m$ . We include the emissions from plants in 5 distance bins to the monitors: 0-1 miles, 1-2 miles, 2-3 miles, 3-5 miles, and 5-10 miles.<sup>32</sup> To capture differences across monitors and years, we include monitor fixed effects  $\eta_m$  and region-by-year fixed effects  $\lambda_{rt}$ . Standard errors are clustered at the monitor level.

Our results show that wind speed affects the relationship between fugitive emissions and ambient lead concentrations within 2 miles of the plant. In contrast, wind speed has very little effect on the relationship between stack emissions and ambient lead concentrations.<sup>33</sup>

Appendix Figure A.11 shows that wind speed has a positive effect on the relationship between fugitive emissions and ambient lead concentrations within 2 miles of the plant, but has very little effect on the relationship between stack emissions and ambient lead concentrations (see Appendix Figure A.12). The figures plot the marginal effect of lead emissions ( $\hat{\delta}_d$ ) on air lead concentration as a function of wind speed near the plants for different distance ranges from plant to monitor. Each panel presents the distance-specific wind gradient, showing how the marginal effect of lead emissions may change with higher average wind speed.

In Appendix Figure A.11, fugitive lead emissions have a nontrivial effect on air lead concentration within 1 mile from the plants. From 1 to 2 miles, fugitive lead emissions have a strong effect when wind speed is over about 7.5. In Appendix Figure A.12, stack lead emissions have a weak effect on air lead concentration within 2 miles from plants at mild and high wind speed and little impact on areas beyond 2 miles. This is because stack lead emissions occur more continuously and higher in the air than fugitive emissions and so are more disperse irrespective of wind speed.

Wind speed is important, because it affects the share of the county population that is exposed to fugitive lead emissions. When local wind speed is low, fugitive lead emissions only affect the neighborhoods extremely close to the plants. The 1990 block-group level data show less than 0.9% of county population living within 0.2 miles from the plants. When local wind speed is high, fugitive lead emissions affect many more people. 25.6% of the population in our sample counties lived within 2 miles of a lead emitting plant. Although 25.6% may seem high, these counties have high population density and multiple lead emitting plants.

<sup>32</sup>We also run a model similar to Currie et al. (2015), which uses continuous or discrete variables for the distance and a regression at plant-monitor level. The results are qualitatively similar.

<sup>33</sup>Results are similar if we add the other county-level control variables included in the first stage equation 1.

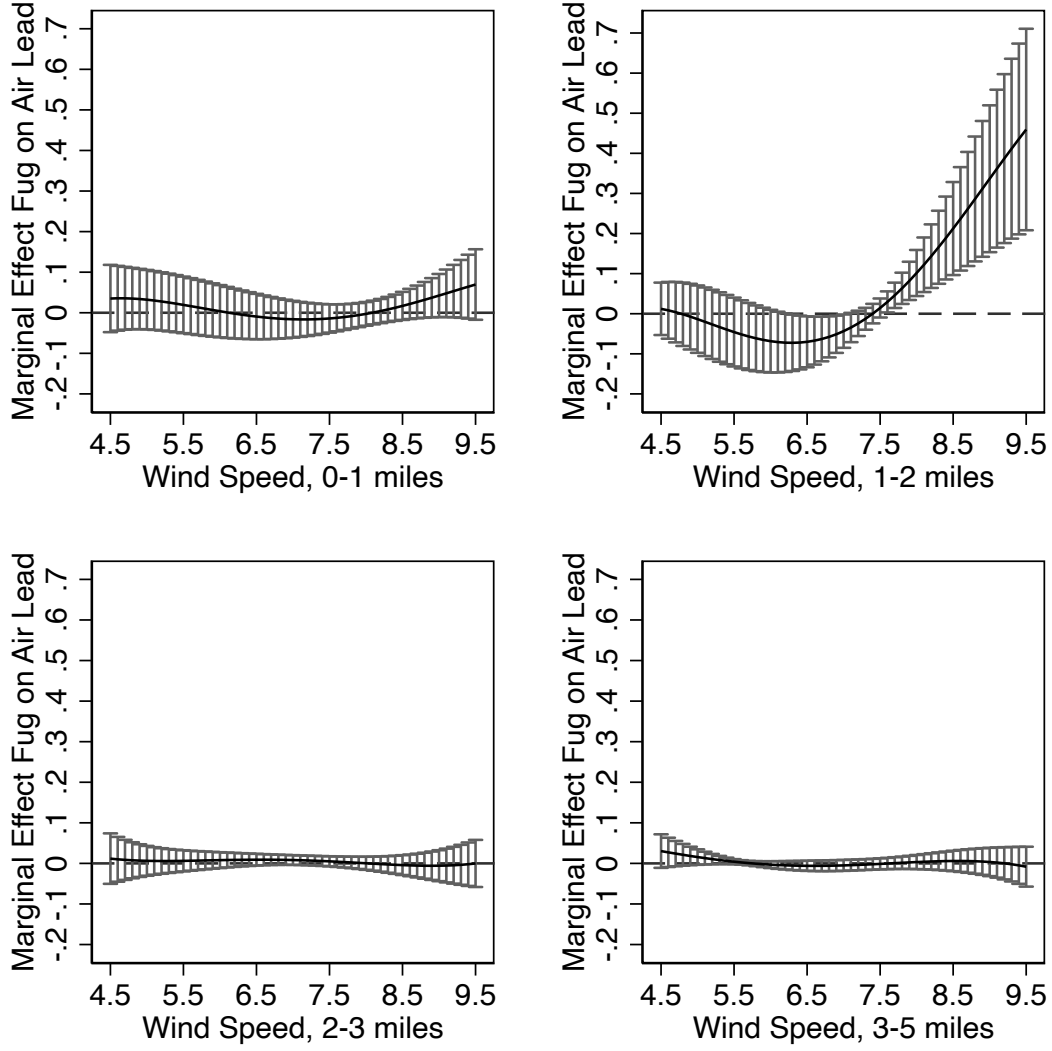


On average in 1990 there were about 7 lead emitting plants per county.<sup>34</sup>

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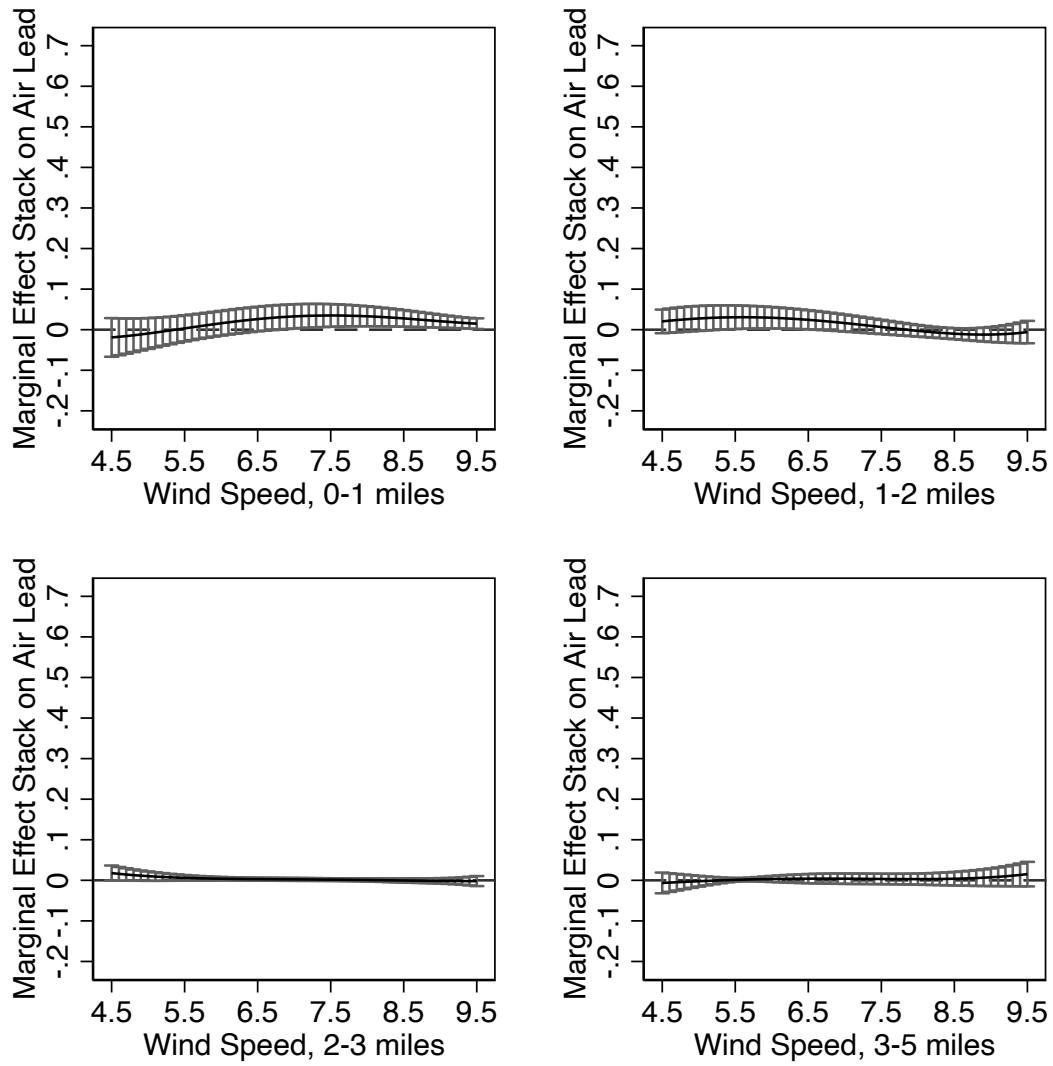
<sup>34</sup>A 2 mile circle around a plant covers 12.6 square miles.

Figure A.11: Effect of Fugitive Lead Emissions on Air Lead Concentration by Wind Speed



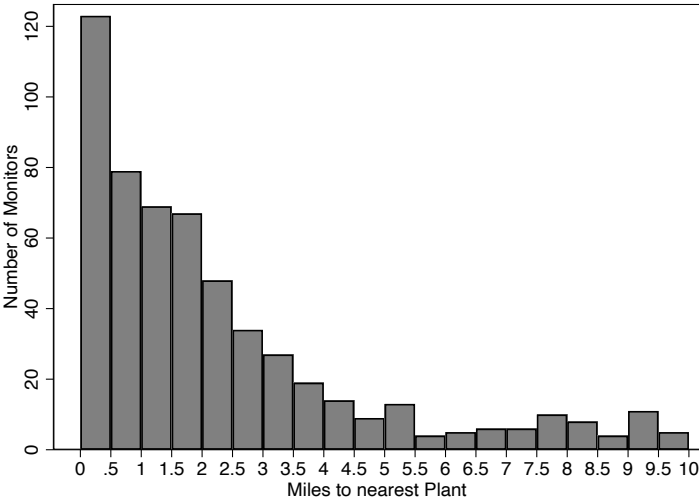
*Notes:* This figure displays the estimated coefficients  $\delta^F$  in equation A.1 – the marginal effect of fugitive lead emissions (in 1,000 pounds) on ambient lead concentration (in  $\mu\text{g}/\text{m}^3$ ) as a function of wind speed (in knots) for different distance ranges from monitors to plants. Wind speed is captured by weather monitoring stations within 10 miles of each plant. Figure A.13a and A.13b show the distribution of monitors over distance to plants and over wind speed, respectively. There is little impact of fugitive lead on ambient lead concentration shown beyond 2 miles, so we did not show the figure for 5 to 10 miles for simplicity. The mean and standard deviation (in brackets) for fugitive lead emissions by plants in each distance bin are listed below. 0-1mi bin is 0.116[1.518], 1-2mi bin is 0.037[0.701], 2-3mi bin is 0.035[0.531], 3-5mi bin is 0.039[0.696].

Figure A.12: Effect of Stack Lead Emissions on Air Lead Concentration by Wind Speed

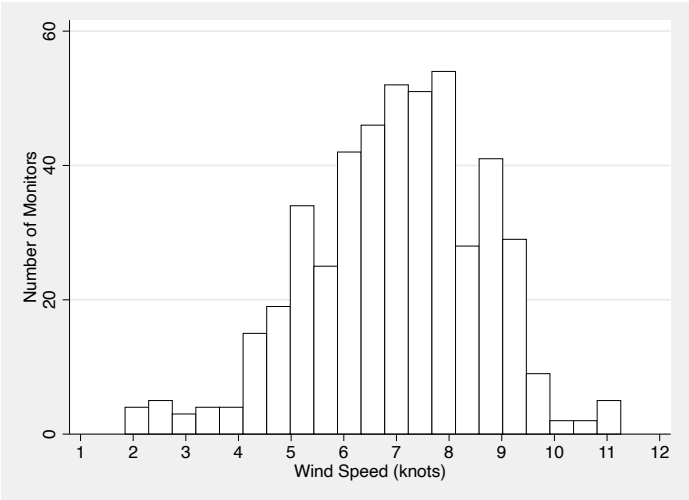


*Notes:* This figure displays the estimated coefficients  $\delta^S$  in equation A.1 – the marginal effect of stack lead emissions (in 1,000 pounds) on ambient lead concentration (in  $\mu\text{g}/\text{m}^3$ ) as a function of wind speed (in knots) for different distance ranges from monitors to plants.

Figure A.13: Number of Lead Monitors by Distance to Plants and by Wind Speed



(a) By Distance to Lead Plants



(b) By Wind Speed (within 10 miles)

*Notes:* Figure A.13a plots the number of monitors in 0.5 mile increments over miles from monitor to the nearest plant. There are 561 monitoring sites in this analysis sample. Figure A.13b plots the number of monitors in 1 knot increments over the average wind speed within 10 miles from the plant.