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

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Abstract

This paper uses U.S. Toxic Release Inventory data on 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 fugitive lead emissions prevented 34-59 infant deaths per year, generating benefits of $380-$670 million annually in 2023 dollars.

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1 Introduction

Air lead concentrations remain substantial in developed and developing countries in the late twentieth and early twenty first centuries, largely due to industrial emissions. Emissions are increasing in China and Mexico (Li et al., 2012; Tanaka et al., 2022). Even with the decline in lead in gasoline, developed countries, including the United States, still have substantial air lead emissions (EPA, 2018). In part as a result of these ongoing emissions, child blood lead levels remain high worldwide. A recent UNICEF report found that 1 in 3 children worldwide had blood lead levels above 5μg/dL, a commonly used benchmark for high lead exposure (Burki, 2020; Rees and Fuller, 2020).

Although there is a large literature on the adverse effects of lead on child cognitive and behavioral outcomes, the literature on lead exposure and infant health is surprisingly small (see a review by Clay et al., 2024). The small literature reflects a lack of studies, as opposed to studies finding a lack of harm. Animal studies suggest that exposure to lead during pregnancy and infancy are harmful for development, in part because of the substitution of lead for calcium in critical cellular functions (Dyatlov and Lawrence, 2002; Sharma et al., 2012; 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). Little is known, however, about the extent of harm to infant health from airborne lead in settings with modern medical care and at modern exposure levels.

One reason for the small literature may be the challenges in estimating the effects of lead exposure on infants, including avoidance behavior, omitted variable bias, and measurement error. Avoidance behavior may include reductions in outdoor activities and investments that improve indoor air quality (Neidell, 2009; Moretti and Neidell, 2011; Ito and Zhang, 2020). Avoidance behavior is likely to be particularly relevant for infant health, because pregnant women have strong incentives to engage in avoidance. Estimation is confounded by many factors that may also generate omitted variable bias, including emissions of other pollutants,

weather conditions, and impacts of industrial activities on local employment and economic conditions (Ruhm, 2000; Chay and Greenstone, 2003; Currie and Schmieder, 2009; Agarwal et al., 2010; Heutel and Ruhm, 2013). Measurement error arising from the disconnection between where air lead is measured and where individuals are exposed and from the timing of exposure may generate bias as well (Lleras-Muney, 2010; Moretti and Neidell, 2011; Knittel et al., 2016; Deryugina et al., 2019).

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. 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 10μg/dL, while stack emissions are not. 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. Thus, not surprisingly, we show that fugitive lead emissions *interacted* with wind speed has low predictive power for county socioeconomic characteristics and mothers' characteristics.

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 fugitive lead emissions prevented 34-59 infant deaths per year, generating benefits of \$384-\$667 million annually in 2023 dollars. Our estimates also imply that the full decline in air lead concentration from all TRI sources prevented 241 infant deaths per year, generating benefits of \$2.7 billion per year.

Second, higher lead concentration increases deaths from sudden unexplained infant death (SUID), respiratory and nervous system causes, and low birthweight.[1] 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),

[1]Sudden unexplained infant death includes sudden infant death syndrome (SIDS), accidental suffocation and strangulation on bed (ASSB), and other unexplained deaths.

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.[2]

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 subliterature 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.[3]

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;

[2]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.
[3]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.

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.[4] 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 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;

[4]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.

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.[5] 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.[6] Four industry groups – lead manufacturing, 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.[7]

[5]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.

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

[7]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).

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 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.[8] 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, avoid-

[8]In 1996, the use of lead in gasoline for on-road vehicles was banned entirely in the United States.

ance 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 10μg/dL. Our main sample includes 127 counties, representing 26% of the US population in 1990, as discussed in the data section. The CDC funds 35 state and local health departments for lead surveillance.[9] 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 10μg/dL. The coefficient on air fugitive lead emissions is positive, statistically significant, and large. In contrast, the coefficient on air stack lead emissions is positive, but not statistically significant and small. A one standard deviation increase in fugitive lead emissions is associated with a 0.83-0.98 percentage point increase in the percentage of children with elevated levels. Given that 6.57 percent of children had elevated blood lead levels in these counties, this translates into a 12.6-14.9% increase.

[9]The funding supports collection in 29 states, the District of Columbia, and five cities – Chicago, Houston, Los Angeles, New York City, and Philadelphia

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 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 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.[10]

To our knowledge, there are no studies that show a causal relationship between air lead and infant mortality.[11] This reflects a lack of studies, as opposed to studies finding a lack

[10] 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.

[11] 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.

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).[12] 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 percent. 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 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

[12] 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.

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.[13]

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 ($\mu g/m^3$). The number of lead monitors varies over time from 2.1 to 4.1 monitors per county.[14] AQS monitor data on particulate matter (PM) and carbon monoxide (CO) are also used in some specifications.

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.[15] 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

[13] 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.

[14] Alternatively, 60 to 261 monitors in the 127 sample counties. Notice, however, that the number of counties also varies over time in the sample.

[15] Expanding the radius does not include many more plants in the sample, but makes the estimates noisier.

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 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.[16] In 1990, 26% 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.

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

[16] Analysis below shows that plant lead emissions are only detectable at EPA lead monitors within 2 miles.

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.[17] 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.[18] We control for the stack lead emissions (S_{ct}), fugitive and stack emissions of

[17] 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.

[18] We will compare first stage and IV results with third, fifth and sixth order polynomials as well.

other TRI-reported chemicals ($Chem_{ct}$), and their interactions with wind.[19] The regression includes county fixed effects (η_c) and region-by-year fixed effects (λ_{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 ($Media_{ct}$).[20]

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, 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

[19] Appendix Table A.2 presents the empirical variation in key explanatory variables.

[20] 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.

country, having a frequency of about 30 percent 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:

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

where $InfMort_{ct}$ is infant mortality in county c in year t.[21] 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.

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 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

[21] We also examine premature birth (< 37 weeks) and low birthweight (< 2500 grams).

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.[22] The F-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.[23] 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. This decrease in air lead concentration would decrease

[22] Again, this is not surprising given the complexity in modeling air pollution due to atmospheric turbulence (e.g., Nieustadt and van Dop, 1982; Raputa and Lezhenin, 2020).

[23] 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.

infant mortality by 0.241 per thousand live births or about 3.1% of mean infant mortality. We discuss deaths averted due to changes in fugitive lead emissions and all 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 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. In columns 3 and 4, which restrict attention to infant deaths from April to December, the coefficient estimates are higher, which suggests that there is some attenuation bias due to mismeasured exposure. For mortality in the first month 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. Compared to the results in columns 1 and 2, the point estimates in columns 3 and 4 are larger and the coefficient for deaths within one month is more statistically significant. This suggests that using current year exposure for infants with significant exposure in the previous year is causing some attenuation bias.

In columns 5 and 6, which restrict attention to infant deaths from July to December, the coefficient estimates are similar or higher than in columns 3 and 4. For mortality in the first month 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. The point estimate in column 5 is nearly

identical to the point estimate in column 3. The point estimate in column 6 is larger than the point estimate in column 4, although the two are not statistically significantly different. This may reflect the fact that some infants who died in April through June at ages beyond one month had significant exposure in the previous year. Dropping them may be further reducing attenuation bias.

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 $\mu g/m^3$ 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 $\mu g/m^3$. 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, 2007,0; Aizer et al., 2018; Aizer and Currie, 2019).

Table 6 suggests that lead exposure may be disproportionately affecting nonwhite infants

at the margin. The coefficient on lead in column 1 for nonwhite infants is substantially larger than in column 2 for white infants. The implied effects for the 0.144 $\mu g/m^3$ decrease in air lead concentration are 0.44 per thousand live births for nonwhite infants and 0.20 for white infants, which are 3.7% and 3.4% of their means. Columns 3 and 4 suggest that these differences are present in the first month. The reasons for these differences are unclear. Some of the difference appears to reflect a differential likelihood of living within 2 miles of a lead plant. It is worth noting that because of the large standard errors, the two point estimates are not statistically significantly different from each other.

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. Because 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), we may have a multicollinearity issue in the estimation. That might be a reason for the imprecise estimate. 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.[24]

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 the 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.

[24]The CDC has guidance on occupational exposure to zinc only to very high levels of zinc fumes.

5.5 Infant Deaths Averted

In Table 7, we use our estimates from Table 3 and the regression underlying Appendix Figure A.10 to do back of the envelope calculations of the number of infant deaths averted. For the IV and reduced form, 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 the IV specification, the realized decline in fugitive lead emissions over these two periods implies a fall in air lead of 0.035 $\mu g/m^3$,[25] and a fall in infant mortality of 0.059 per 1,000 live births.[26] In the reduced form specification for the IV sample, the realized decline implies a fall in infant mortality of 0.034 per 1,000 live births. As usual, the IV estimates are larger than the reduced form estimates.

Table 7 summarizes the infant deaths averted and the value of these lives saved. The annual number of births in the sample counties was approximately 1 million in 2015-2018. Depending on the specification, the implied number of deaths averted in these counties per year is 34-59. At the EPA valuation of $11.3 million per death averted in 2023 USD, the benefits of infant lives saved are $384-667 million per year.[27] The full decline in air lead concentration from all sources in our sample counties was 0.144 $\mu g/m^3$.[28] This implies a fall in infant mortality of 0.241 per 1,000 live births or 241 deaths averted per year. The benefits of infant lives saved are $2.7 billion per year.

[25] For reference, the average air lead concentration in our sample is $0.08 \mu g/m^3$.

[26] 0.059=0.035*1.676, where 1.676 is the coefficient on air lead concentration in column 5 of Table 3.

[27] The value of a statistical life that the EPA uses is $7.4 million (2006 USD). This is $11.3 million (2023 USD).

[28] 0.144 is the difference between the air lead concentration for 1988-1991 and the air lead concentration for 2015-2018 for the IV sample.

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. Variation in fugitive lead emissions and wind speed is shown to determine lead exposure. While stack lead emissions occur routinely and may be subject to avoidance behavior, the unpredictable annual variations in fugitive lead and wind speed make it difficult to engage in avoidance behavior. We provide evidence that fugitive lead emissions interacted with wind speed has low predictive power for county socioeconomic characteristics and mothers' characteristics. We provide evidence that fugitive lead emissions and wind speeds are strongly linked to air lead concentration readings of EPA monitors.

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. Cause of death data show that lead increases deaths from low birthweight and sudden unexplained infant death. Differences in the estimates by race are imprecise but suggest that lead exposure may be disproportionately affecting nonwhite infants. Back of the envelope calculations indicate that declines in fugitive lead emissions generated benefits of \$384-\$667 million annually and the full decline in air lead concentration from all sources generated benefits of \$2.7 billion per year. These are only the benefits from avoided infant deaths. Given that lead exposure causes morbidity and mortality in other populations and has lasting impacts on child development, the benefits are likely substantially larger.

Returning to the broader picture, air emissions of lead from industry and some other sectors such as aviation continue to impact millions in the United States and globally. These new estimates can inform investments in reductions in air lead emissions. In the U.S.,

industrial firms and the aviation industry still emit hundreds of thousands of pounds of lead into the air.

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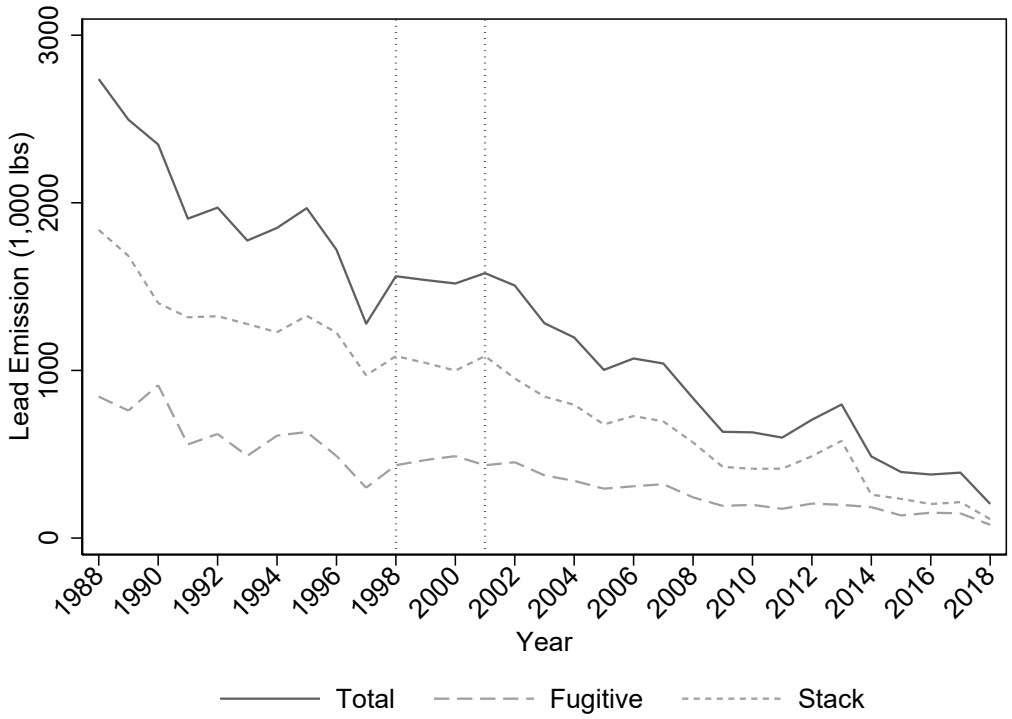
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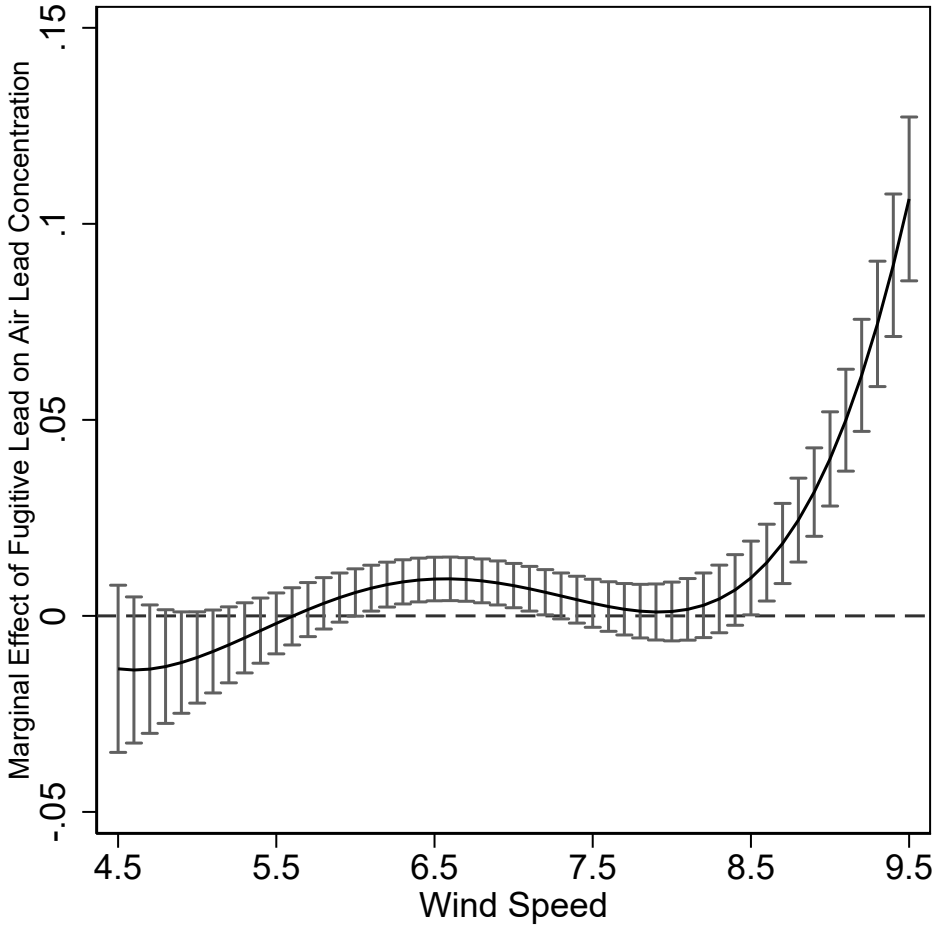
Tables and Figures

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**	0.837*	0.898*	0.805**
	(0.363)	(0.485)	(0.521)	(0.399)
Air Stack Lead	0.057	0.049	0.049	0.048
	(0.062)	(0.044)	(0.047)	(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 percent of children in a county with elevated blood lead level ($\geq 10\mu$g/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***	0.140***	0.139***	0.138***	0.138***
	(0.046)	(0.042)	(0.043)	(0.042)	(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) IMR	(2) IMR	(3) IMR	(4) IMR	(5) IMR
Air Lead Concentration	1.998***	1.753***	1.717***	1.685***	1.676***
	(0.429)	(0.453)	(0.447)	(0.395)	(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) IMR 1m	(2) IMR 1y	(3) IMR AD1m	(4) IMR AD1y	(5) IMR JD1m	(6) IMR JD1y
Air Lead Concentration	0.728**	1.676***	1.155***	2.128***	1.143***	2.660***
	(0.323)	(0.435)	(0.336)	(0.430)	(0.387)	(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) LowBw	(2) SUID	(3) Resp.	(4) Nerv.	(5) Cong.	(6) Peri.	(7) Others
Panel A. IMR in the first year							
Air Lead	0.349**	0.407*	0.419***	0.121**	0.110	-0.238	0.544***
	(0.163)	(0.208)	(0.149)	(0.051)	(0.144)	(0.281)	(0.120)
Dep Mean	1.172	1.113	0.890	0.112	1.541	1.880	0.834
Panel B. IMR in the first month							
Air Lead	0.363**	0.020	0.242**	0.059**	0.080	-0.225	0.262***
	(0.162)	(0.034)	(0.117)	(0.023)	(0.136)	(0.295)	(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) IMR1yrnwh	(2) IMR1yrwh	(3) IMR1mnwh	(4) IMR1mwh
Air Lead Concentration	3.068**	1.415***	1.977**	0.478*
	(1.212)	(0.361)	(0.998)	(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.

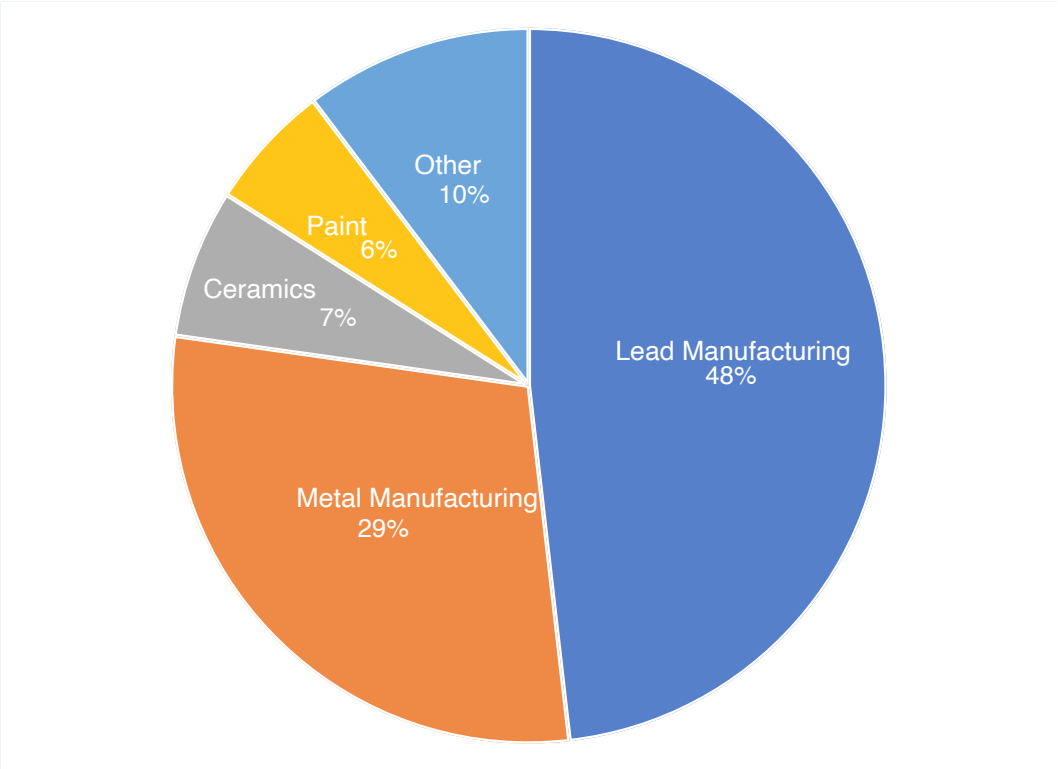
Table 7: Back of the Envelope Calculations

	(1) Annual Infant Deaths Averted	(2) Value in 2018$
IV, Fug Lead	59	$667 million
Red Form, Fug Lead	34	$384 million
IV, All Lead	241	$2.7 billion

Notes: The IV, Fugitive lead estimates are generated by using the average of fugitive lead 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 by 1 million to get the annual infant deaths averted. The reduced form estimates the average of fugitive lead from 1988-1991 and 2015-2018 to generate the reduction in infant deaths based on the regression underlying Appendix Figure A.10. The IV, 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 by 1 million 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.

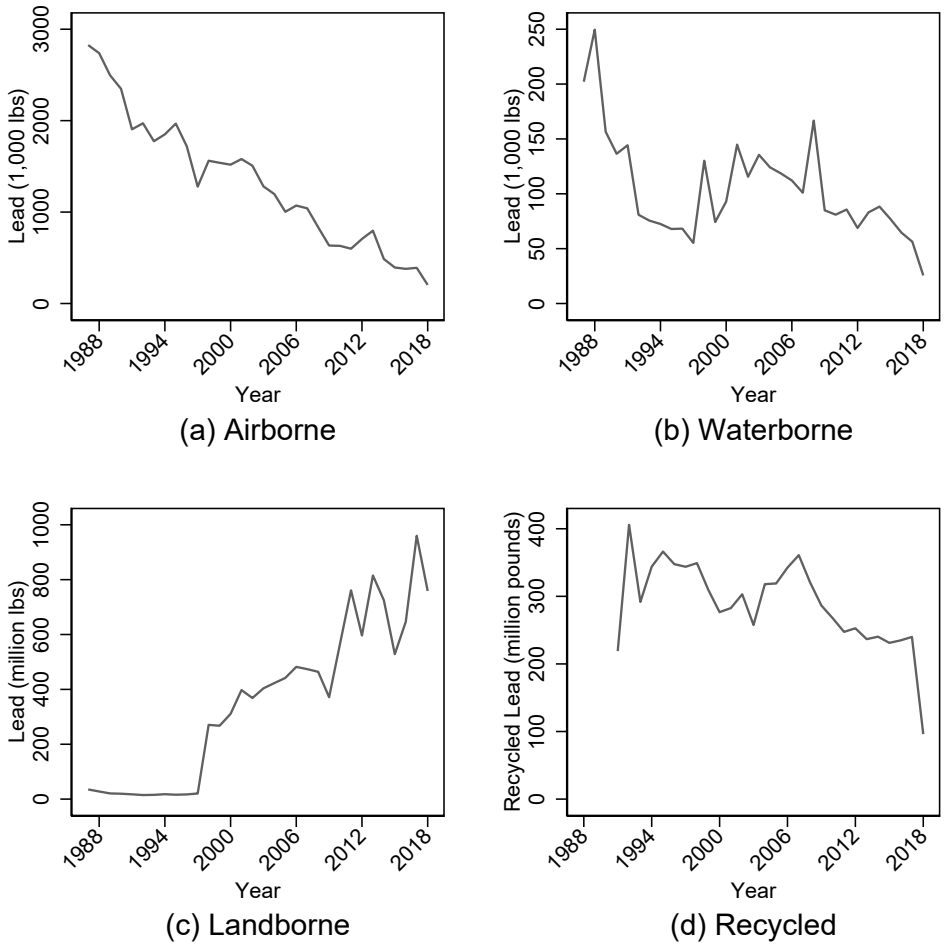
A Appendix Figures and Tables

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



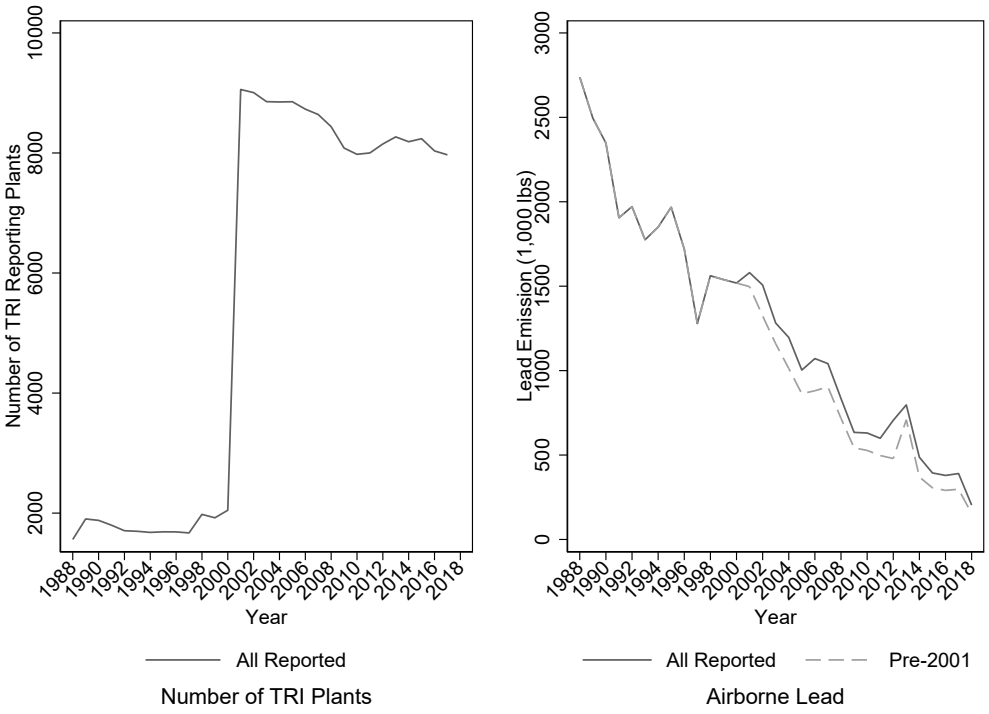
Notes: This pie-chart shows the industry distribution of airborne lead emissions (sum over time) by the sampling industrial facilities. The emissions include both fugitive and stack lead emissions. Calculates are weighted by the number of births in a county.

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



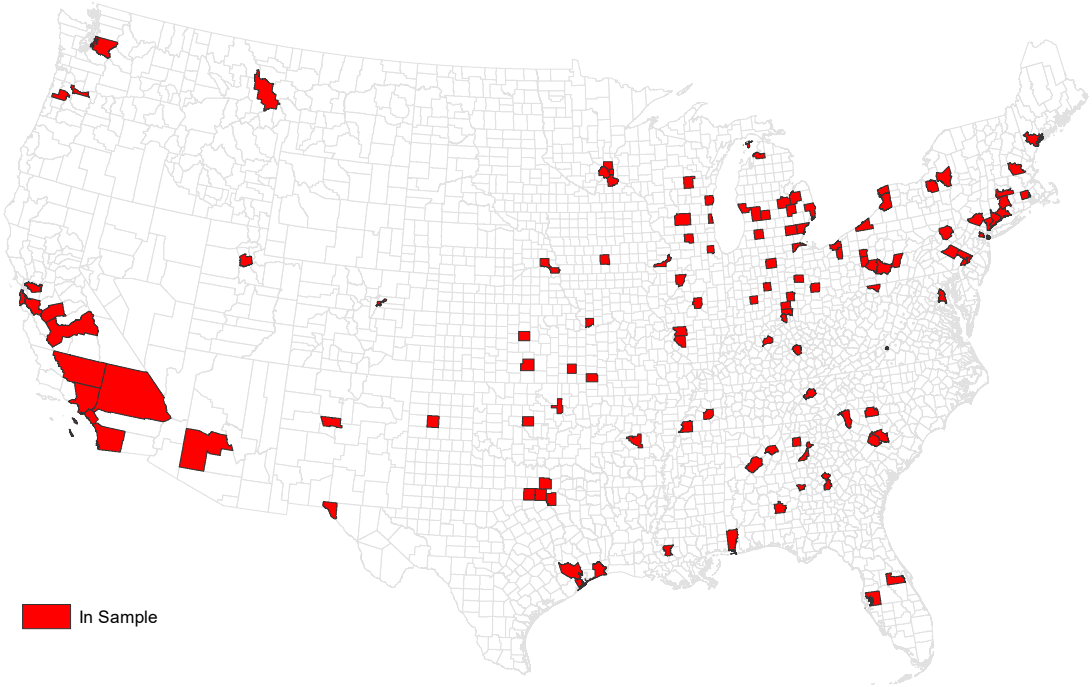
Notes: Figure (a) to (c) plot the trends of air-, water-, and land-borne lead emissions by TRI plants. Figure (d) plots the lead recycled from production waste. Data on recycled lead started from 1991, following the Pollution Prevention Act (1990) that expanded TRI to include additional information on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

Figure A.3: Changes in Reporting in 2001



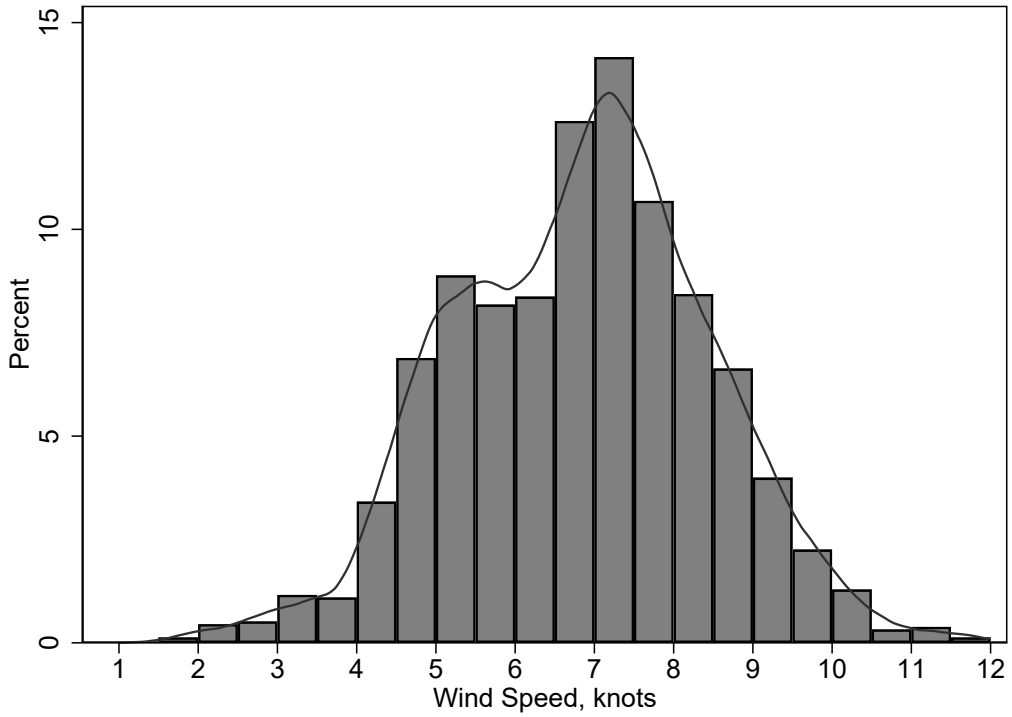
Notes: This figure shows trends in the number of reporting plants and their effect on air lead emissions in our sample during 1988 to 2018.

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



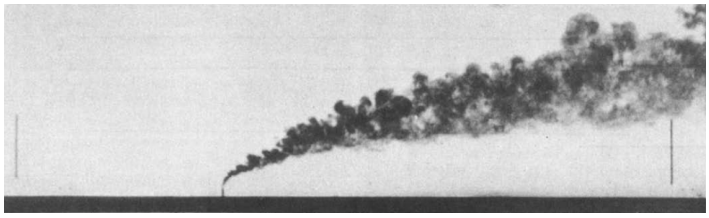
Notes: This map presents locations of the 127 counties in our sample.

Figure A.5: Histogram of Wind Speeds



Notes: This figure plots the distribution of wind speeds.

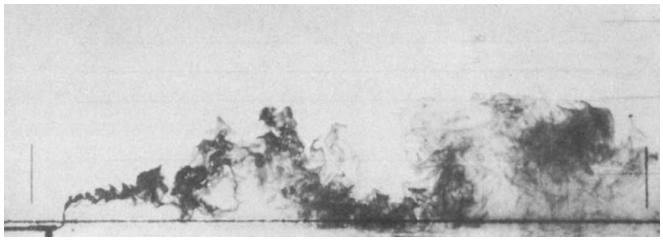
Figure A.6: Plume Patterns in the Laboratory



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



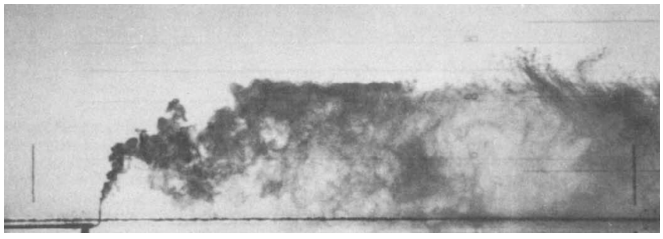
(b) Non-buoyant plume in a convectively turbulent environment



(c) Weakly buoyant plume in a convectively turbulent environment



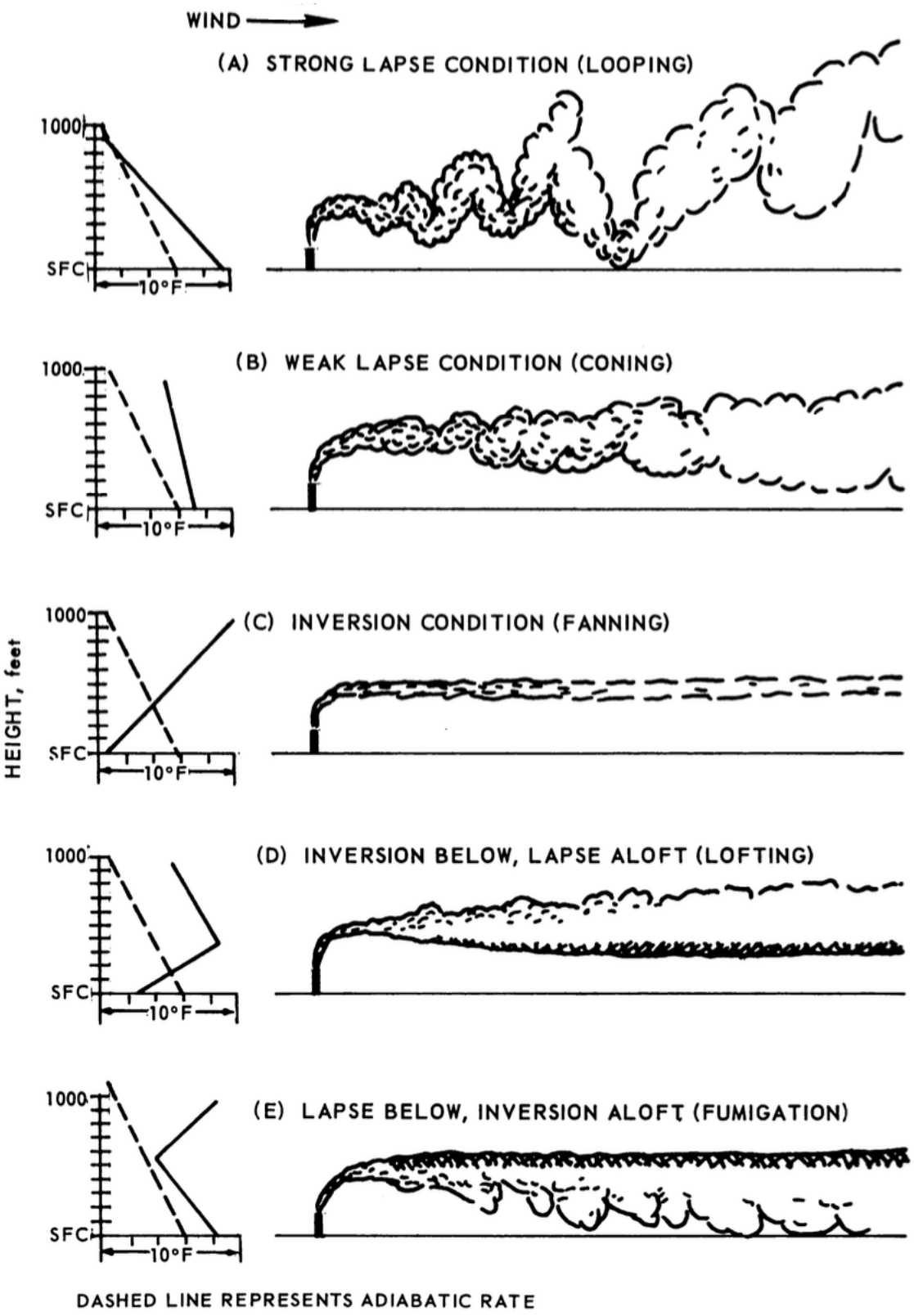
(d) Moderately buoyant plume in a convectively turbulent environment



(e) Strongly buoyant plume in a convectively turbulent environment

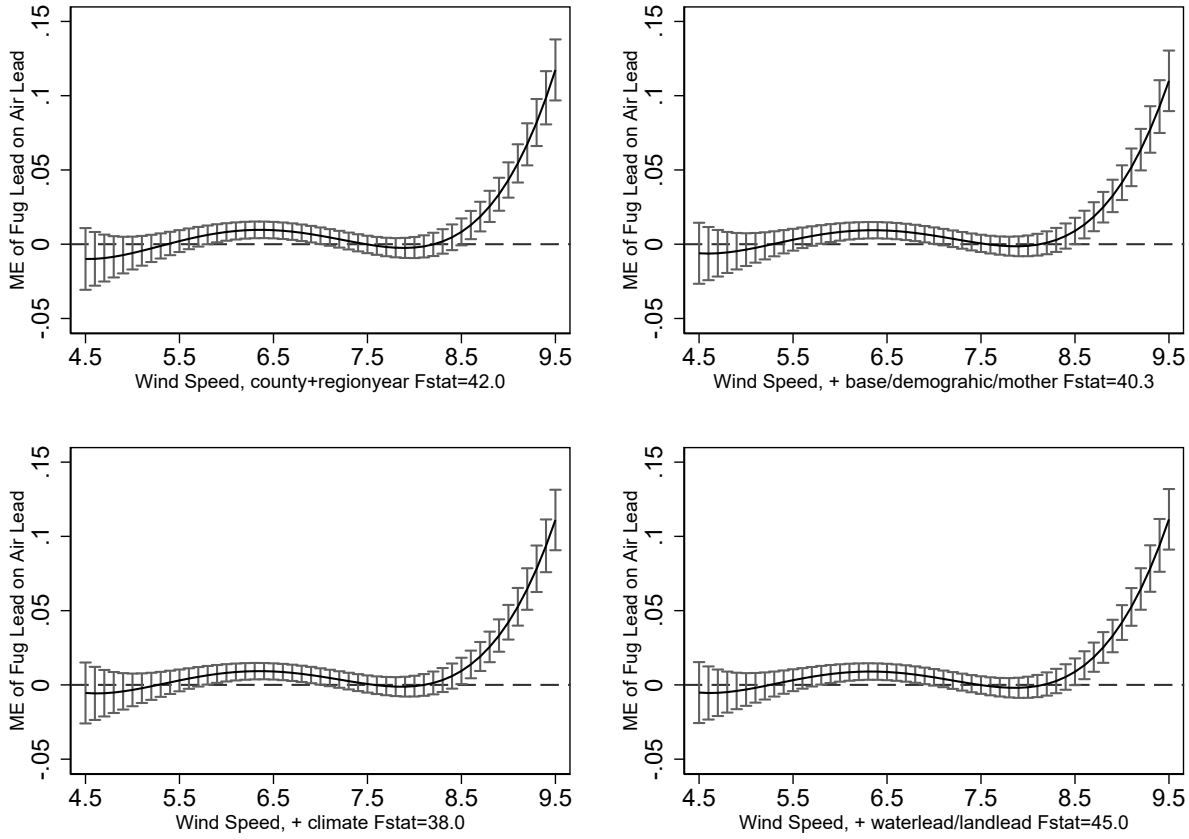
Notes: This figure plots potential plume patterns from a point source pollution. A convectively turbulent environment refers to a situation where convective processes lead to highly irregular and chaotic fluid motions. This movement typically happens in a circular or cyclical manner. "Convective" refers 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



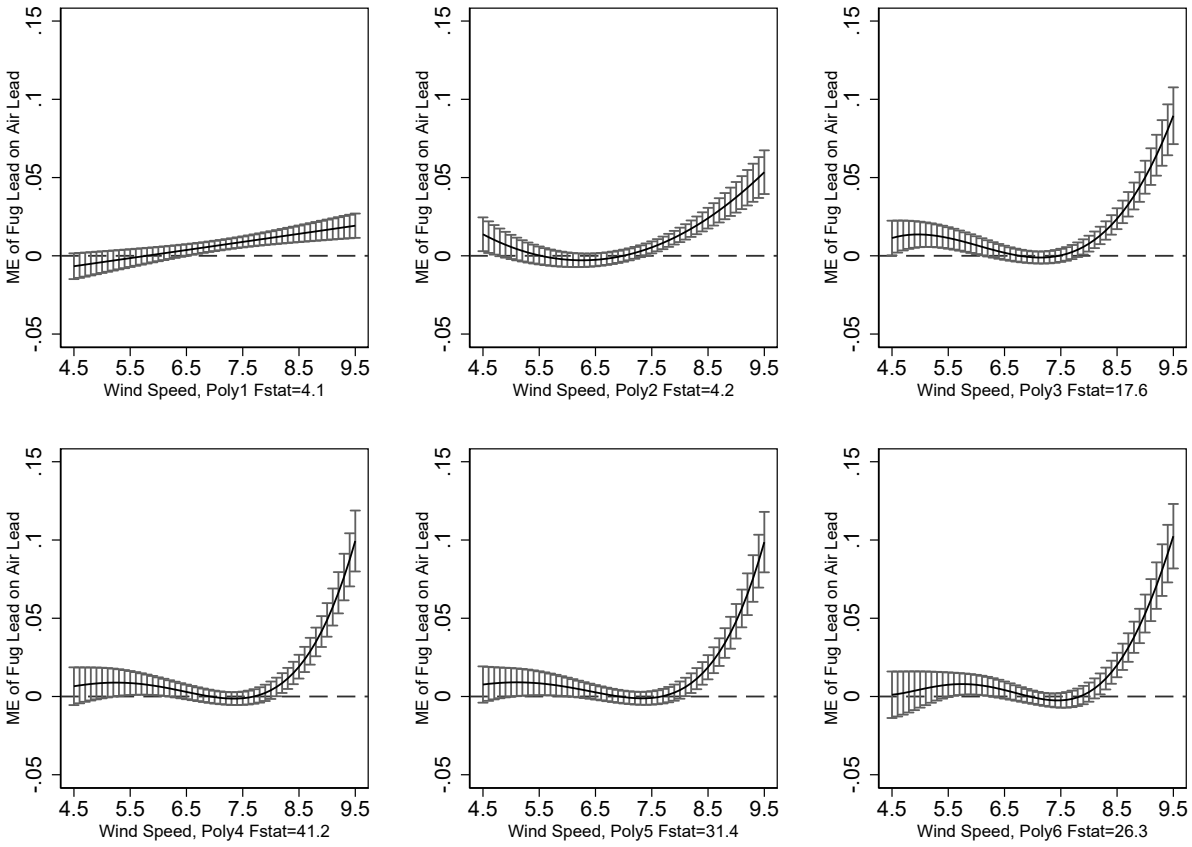
Notes: This figure plots potential plume patterns from a point source pollution. *Source*: USEPA (1968).

Figure A.8: First Stage Adding Controls



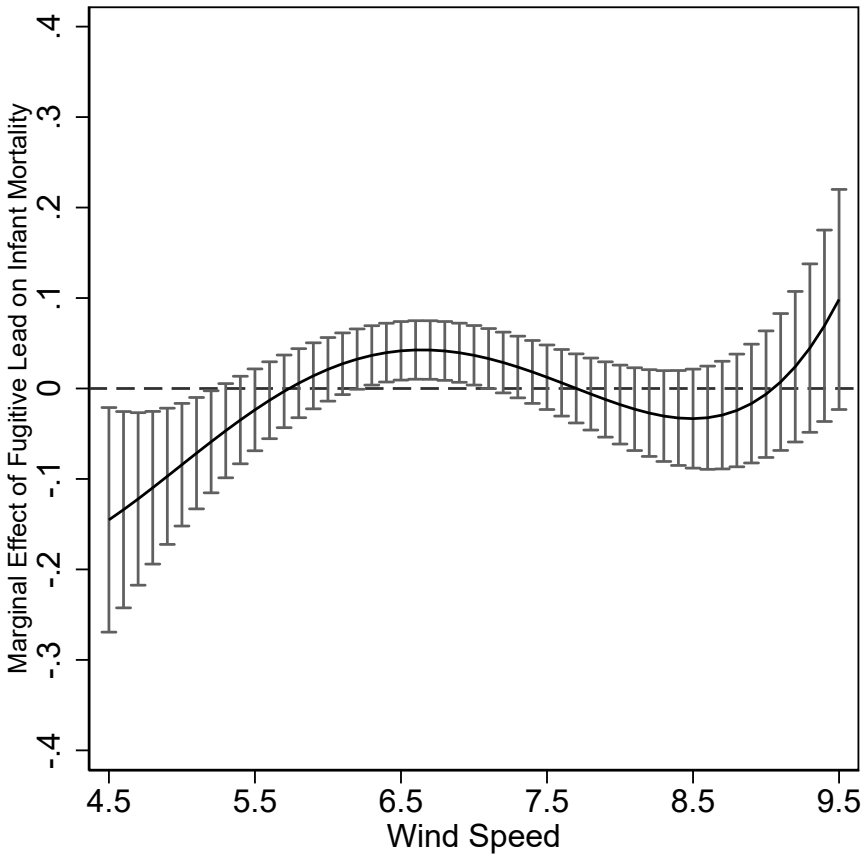
Notes: The figure plots the marginal effect of fugitive lead emissions (F_{ct}) of plants on the air lead concentration readings at lead monitors as different sets of controls are added. These first stages correspond to columns 1-4 in Table 3.

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 (Within 2 miles to plants)	1.98	1.76
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) MFemp	(2) Emp	(3) Popdens	(4) Hinc	(5) Pwhite	(6) 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) Pmwhite	(2) Pmhisp	(3) Pmolder35	(4) 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) Prem AD	(2) Prem JD	(3) LowBw AD	(4) LowBw 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 ($< 2{,}500$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) IMR	(2) IMR	(3) IMR	(4) IMR	(5) IMR	(6) IMR	(7) IMR
Air Lead Conc.	1.529***	1.546***	1.676***	1.901**	1.725**	2.343**	0.940
	(0.458)	(0.411)	(0.435)	(0.804)	(0.842)	(0.915)	(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. 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***	1.695***	1.537***	1.603***	1.798***	1.728***
	(0.435)	(0.441)	(0.455)	(0.527)	(0.471)	(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.

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:

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

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.[29] To capture differences across monitors and years, we include monitor fixed effects η_m and region-by-year fixed effects λ_{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.[30]

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.

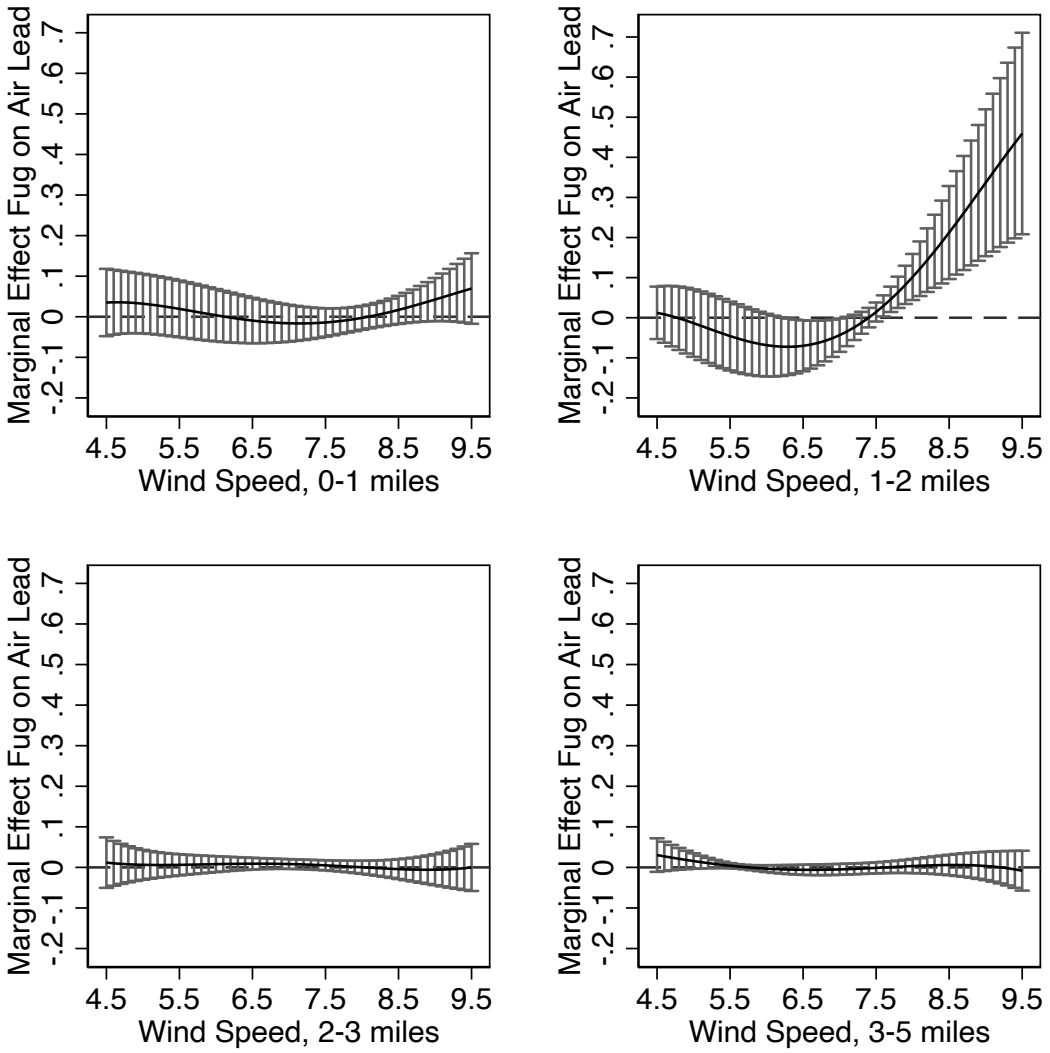
[29] We also run a model similar to Currie et al. (2015), which uses continuous or discrete variables for the distance and a regression at plant-monitor level. The results are qualitatively similar.

[30] 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.[31]

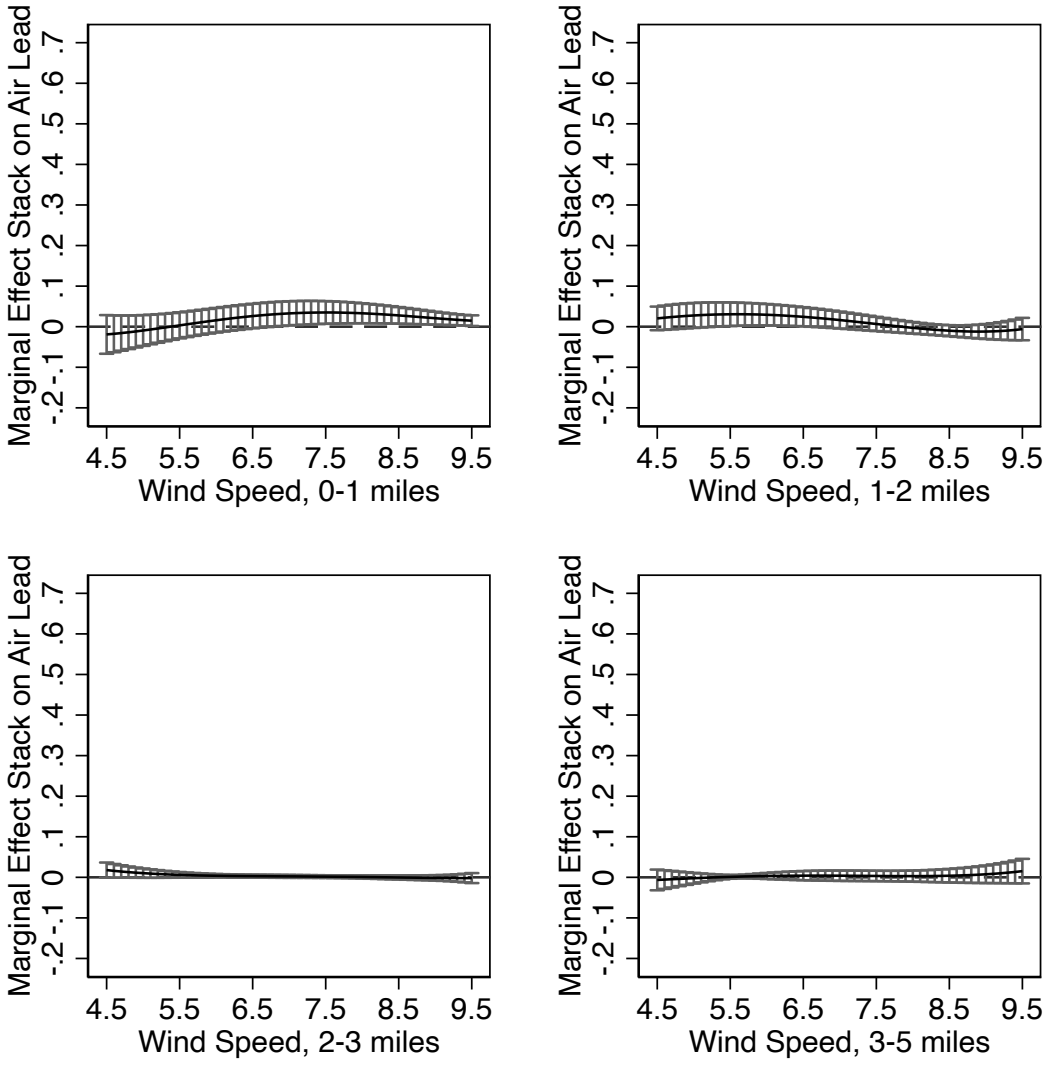
[31] A 2 mile circle around a plant covers 12.6 square miles.

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



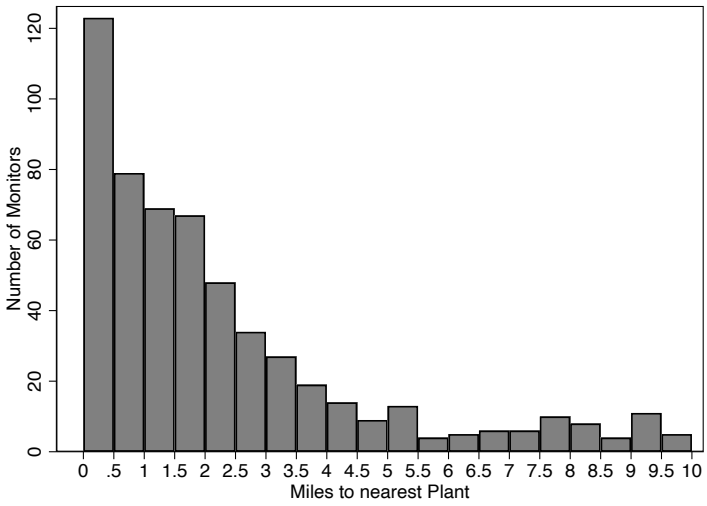
Notes: This figure displays the estimated coefficients δ^F in equation A.1 – the marginal effect of fugitive lead emissions (in 1,000 pounds) on ambient lead concentration (in $\mu g/m^3$) as a function of wind speed (in knots) for different distance ranges from monitors to plants. Wind speed is captured 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

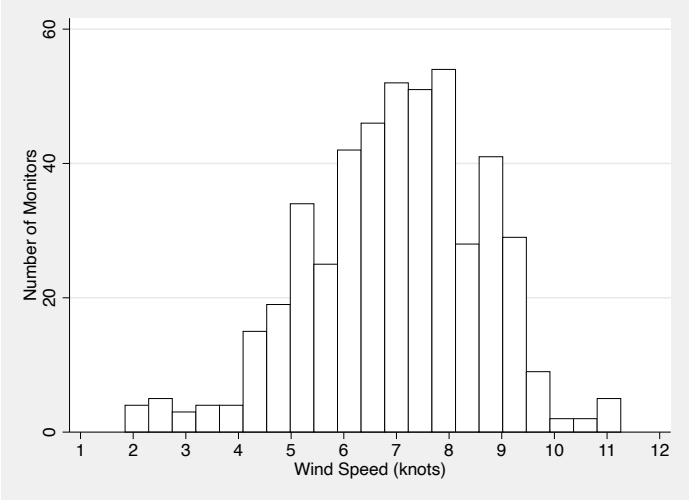


Notes: This figure displays the estimated coefficients δ^S in equation A.1 – the marginal effect of stack lead emissions (in 1,000 pounds) on ambient lead concentration (in $\mu g/m^3$) as a function of wind speed (in knots) for different distance ranges from monitors to plants.

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.