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ABSTRACT

The Impact of Lead Exposure on Fertility, Infant Mortality, and Infant Birth Outcomes*

Lead exposure has detrimental effects on fertility, infants, children, and adults. Despite the success in removing lead from on-road gasoline, industrial and aviation emissions continue to pose a substantial global challenge. Other major sources of exposure include dust, soil resuspension, and consumption of contaminated water or food. Both animal studies and evidence from humans support claims of an adverse relationship between lead pollution and human health. Since lead exposure is not randomly assigned, quasi-experimental studies play a crucial role in this knowledge base. Among these studies, extensive research links elevated blood lead levels in children to academic and behavioral outcomes, but more limited attention has been given to lead's impact on fertility, infant mortality, and infant health. This paper examines the existing quasi-experimental literature on lead and fertility, infant mortality, and infant birth outcomes, highlighting key results, methods, and implications for policymakers.

JEL Classification: I120, Q530, Q580
Keywords: lead exposure, fertility, mortality, infant health

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1 Introduction

Lead has a range of adverse effects on fertility, infants, children, and adults. Pathways of exposure to lead include breathing in airborne lead from airborne emissions, dust, and soil resuspension; consumption of waterborne lead or foodborne lead; and in rare cases skin exposure. The adverse effects are documented by animal studies and a range of evidence for humans. Harm is generally greater for children than adults, because of their more limited ability to excrete lead and children's rapid neurological development. One important mechanism through which lead harms humans is through its replacement of calcium. Calcium plays a key role in cellular functions, and its replacement with lead impairs these functions.

While removal of lead in gasoline generated enormous public health benefits, lead exposure remains significant in developed and developing countries. Much of the exposure comes from industrial emissions. Emissions are increasing in China and Mexico (Li et al., 2012; Tanaka et al., 2022). Developed countries, including the United States, still have substantial air lead emissions (EPA, 2018). A recent UNICEF report found that 1 in 3 children worldwide had blood lead levels (BLLs) above 5μg/dL, a critical threshold (Burki, 2020; Rees and Fuller, 2020).

A large literature on children links BLLs to short run academic and behavioral outcomes and in some cases to longer run outcomes including high school completion, crime, and labor market outcomes. Large studies using data from North Carolina found effects of preschool lead levels on educational outcomes in fourth grade (Miranda et al., 2007) and persistent effects from third through eighth grade (Shadbegian et al., 2019). Recent papers have begun to leverage large longitudinal data sets and quasi-experimental methods to provide estimates of exposure on outcomes (Gronqvist et al., 2020; Aizer and Currie, 2019; Aizer et al., 2018). Other papers in this symposium review dimensions of this important literature. [Discuss other papers in the symposium briefly]

In contrast, the literatures on lead and fertility and lead and infant mortality are much smaller, despite their social and economic importance as outcomes. An adverse effect on

fertility means that some children are not born as early as desired and some children are never born. Children not born and infant mortality are by definition not captured by the literature on the BLLs of children. If children not borne are assumed to have been valued at least the USDA cost of raising a child, the value is $288,450 (2022 USD) per child not including expenditure for college or inflation. The actual value of children not borne is likely to be much higher. For mortality, the U.S. Environmental Protection Agency (EPA) value of a statistical life is $11.2 million (2022 USD). To the extent that lead causes decreases in fertility and increases in mortality, the social costs are likely to be very large.

The literature on lead and infant health is also smaller, yet important. While related to the literature on children with high BLLs, the literatures only partially overlap. Some children will be exposed in utero, but not have high blood levels, because blood level tests only measure exposure within the last 30 days. As a simple exercise, we can think about children as being in 4 bins: i) in utero exposure and high BLL due to later exposure; ii) in utero exposure and normal BLL; iii) no in utero exposure but high BLL due to later exposure; and iv) no in utero exposure and normal BLL. The literature on BLLs and adverse outcomes captures two of these groups: children exposed in utero with high BLL and children not exposed in utero but with high BLL. Importantly, the literature does not include the group that was exposed in utero but has normal BLL at the time of the test. Costs to this group may be significant if in utero (or later) lead exposure adversely affected infant neurological development, other dimensions of health, or both.

This paper reviews the quasi-experimental literatures on lead and fertility, lead and infant mortality, and lead and infant birth outcomes and then discusses the relevance of these studies for policy. Why quasi-experimental studies? Because quasi-experimental (QE) techniques provide an opportunity to better understand the relationship between pollution and human health (Dominici et al., 2014). In a QE evaluation, the researcher compares outcomes of a treatment group with outcomes of a control group, just as in a classical experiment. Treatment status, however, is determined naturally by politics, an accident, a regulatory

action, or some other action beyond the researcher's control. The key difference from an observational study is that the QE approach is devoted to identifying treatment-induced variation in pollution that plausibly mitigates biases arising from confounding factors. Despite the "nonrandom" assignment of treatment status, it is possible to draw causal inferences from the differences in outcomes between the treatment and control groups.

The paper argues that BLL data should be used if available but is not necessary for quasi-experimental studies to be used in policy settings. In contrast to the literature on child BLLs and outcomes, the literatures on lead and fertility, infant mortality, and infant health rarely include measures of BLLs. While data on BLLs should be used if available, BLLs are not routinely collected in many settings. Even in cases where BLLs are available, the data has some limitations. These limitations include: the fact that BLLs are often noisily measured using finger pricks; both finger price and veinous draws capture short run exposure; and infants, children, and mothers with BLL data are not representative of the broader population. If BLL data are going to be required for policy purposes, much larger investments in BLL measurement need to be made. We argue that even in the absence of data on BLLs, quasi-experimental studies provide policy relevant evidence on lead exposure outcomes.

This review is organized as follows. Section 2 provides background on a range of topics including vectors of exposure, measuring lead exposure, the effects of lead on fertility, mortality, and infant health in animal experiments, and moving from animal experiments to human studies. Section 3 surveys quasi-experimental studies of the effects of lead on fertility, mortality, and infant health in humans and discusses valuation of these effects. Section 4 discusses the implications for policymakers.

2 Background

A primary reason why lead exposure can be so damaging is that lead is chemically similar to calcium (Garza et al., 2006; Ferreira De Mattos et al., 2017). Lead serves no purpose in the human body, while calcium is essential for a number of vital functions. Calcium plays an central role in many physiologic processes including both neurotransmission and the maintenance of a stable blood pressure. When lead enters the body it supplants some of the calcium used in these critical functions, leading to many issues including neurological and cardiovascular impairment. In other words, lead is a deadly neurotoxin that impairs body functions and increases the risk of disease and death.

2.1 Vectors of exposure

Individuals can be exposed to lead in air, soil, and water. Airborne lead can arise from contemporaneous emissions, soil resuspension, and building demolition. In the United States, current emissions are primarily from industry and aviation. For industrial lead, according to the Toxics Release Inventory (TRI) over 70% of the industrial lead emissions are from lead and other metal manufacturing.[1] Airborne lead concentration, as measured by the relatively few EPA lead monitors, are higher in locations closer to these industrial plants. A substantial share of the current flow is attributable to lead-formulated aviation gasoline (avgas), used in a large fraction of piston-engine aircrafts.

A number of papers document a positive relationship between air lead and child BLL. Clay et al. (2022) find that child BLLs are strongly related to air industrial fugitive lead emissions. Hollingsworth and Rudik (2021) show that child BLLs are strongly associated with emissions from automotive races using leaded gasoline. Miranda et al. (2011), Zahran et al. (2017), and Zahran et al. (2022) find that child BLLs increase dose-responsively in proximity to airports, particularly when the flow of piston-engine aircraft traffic is more

[1]TRI tracks the management of certain toxic chemicals that may pose a threat to human health and the environment. U.S. facilities in different industry sectors must report annually how much of each chemical is released to the environment and/or managed through recycling, energy recovery, and treatment.

intense.

Lead in the air can also emerge from soil resuspension of legacy lead historically deposited in topsoil. The accumulation might have happened during the period when lead was used in gasoline (Resongles et al., 2021). Zahran et al. (2013) shows that a 1% increase in the amount of resuspended soil results in a 0.39% increase in the concentration of lead in the atmosphere. Furthermore, they study BLLs in children and provide evidence that the resuspension of lead-contaminated soil explains observed seasonal variation in child BLLs. Lastly, building demolition could be another source of lead exposure, because older housing units are likely to contain lead-based paint (Jacobs et al., 2013).

Besides resuspension in the atmosphere, lead-contaminated soil can be a vector of exposure through ingestion and skin contact as well. Urban residents are at risk from exposure to legacy lead dust in topsoil resulting from smelting, industrial discharges, leaded gasoline emissions, leaded paint, and incineration. From 2007 to 2010, the U.S. Geological Survey (USGS) collected soil samples at more than 4,800 sites (one site per 1,600 square kilometers) throughout the conterminous United States. Levels of lead in soil in non-urban areas generally ranged from below 10 to 95 parts per million (ppm) (EPA, 2013). In contrast, urban soil lead concentrations vary greatly and can be as high as 2,500 ppm. Soil lead contamination is particularly common in Superfund sites. Federal Superfund program guidance suggests a preliminary remediation goal of 400 ppm.[2]

Lead concentration in soil has been shown to affect BLLs (Mielke et al., 2019). Klemick et al. (2020) evaluated the effect of EPA's Superfund cleanup program on children's lead exposure more broadly. They linked two decades of children's BLL measurements with data on Superfund sites and other lead risk factors. Results indicate that Superfund cleanups lowered the risk of elevated BLL for children living within 2 km of a lead-contaminated site between 13 and 26%.

Lead in water is another vector of exposure. Lead contamination of drinking water may

[2]Some states like Missouri, Massachusetts, and Rhode Island, however, use more stringent levels, varying from 150 to 260 ppm.

be increasing because of lead-containing water infrastructures, changes in water sources, and changes in water treatment. A soluble metal, lead leaches into drinking water via lead-based plumbing or lead particles that detach from degrading plumbing components. Lead was restricted in plumbing material in 1986, but older homes and neighborhoods may still contain lead service lines, lead connections, lead solder, or other lead-based plumbing materials. Children can absorb 40 to 50% of an oral dose of water-soluble lead compared with 3 to 10% for adults.

Lead in water increases child BLLs. The incidence of elevated BLLs in children increased from 2.4 to 4.9% after Flint, Michigan, introduced a more corrosive water source into an aging water system without adequate corrosion control (Hanna-Attisha et al., 2016). In general, water lead at the tap level is poorly monitored, because the numbers of water samples from households, schools, and other buildings are small and non-randomly selected. It was the increase in elevated BLLs that led to further testing in Flint.

While lead in the air, soil, and water are the most common sources of lead exposure, there are other sources as well. These include lead-based paint, insecticides, traditional medicines or cosmetics, food, and contaminated consumer products. Understanding and mitigating all these sources of lead exposure is essential for protecting public health.

2.2 Measuring lead exposure

Although lead can be detected in plasma, serum, saliva, hair, urine, feces, nails, bones, and teeth, currently lead concentration in whole blood is the primary biomarker used to monitor exposure (Barbosa et al., 2005). After lead enters the body, it can travel along several pathways depending on its source and, by extension, its bioavailability. Lead primarily enters the body from breathing in dust or chemicals that contain lead or by ingesting food or liquids that contain lead. Once in the lungs, lead goes quickly to other parts of the body via the bloodstream. Similarly, once lead reaches the stomach, some is absorbed into the bloodstream and the remainder is excreted. Once in the blood, lead travels to the "soft

tissues" and organs such as the liver, kidneys, lungs, brain, spleen, muscles, and heart. The half-life of lead in blood is approximately 30 days. After several weeks, most of the lead moves into the bones and teeth, where it can remain for years. Once it is taken in and distributed to organs, the lead that is not stored in bones leaves the body via urine or feces.

There are two main data sources that measure incidence of lead exposure via blood lead levels (BLLs) in children in the United States – NHANES and CDC surveillance. The National Health and Nutrition Examination Survey (NHANES) is a population-based program of studies designed to assess the health and nutritional status of children and adults. Since 1976, as part of NHANES, CDC has used state-of-the-art technology to measure BLLs. We plot the NHANES data from 1976 to 2018 in Figure 1. The first panel plots the distribution of measured BLLs from each NHANES wave, the second panel displays the average BLL from each wave, and the third panel shows the share of NHANES participants that had blood lead above the modern standards for elevated blood lead levels (5 and 10 μg/dL). The data show evidence of clear, yet still incomplete, progress in reducing blood lead concentrations. The distributions need to be displayed on a log base 10 scale so the range of the BLLs from the earliest NHANES wave does not dwarf the more compressed range of more modern distributions. These data reveal that the average BLL in the U.S. has declined over time from 14.6μg/dL in 1976-1980 to 1.1μg/dL in 2017-2018. Every participant of the NHANES wave of 1976-1980 had BLLs over 5μg/dL. By the 2017-2018 wave, that share had dropped to 1.3%.

Due to the large number of people with extremely high levels of blood lead, including the pre-1999 NHANES waves in any time-series comparison will mask important trends in more recent data, where baseline levels of blood lead are much lower. For that reason, in Figure 2 we present the same plots, but only for the NHANES continuous waves that have operated from 1999-2018. Here we see continued progress in the reduction of BLLs. In the 1999-2000 wave the average BLL was 2.2 μg/dL, which decreased by half in 2017-2018.

CDC began collecting childhood blood lead surveillance data in 1995. CDC funds 35

state and local health departments for lead surveillance (29 states, the District of Columbia, and five cities – Chicago, Houston, Los Angeles, New York City, and Philadelphia). As part of their funding agreement, health departments are required to report data on a quarterly basis. Data reporting is voluntary in states CDC does not fund. Because it was not designed to be nationally representative, CDC's state-based data are not directly comparable to data in NHANES. That said, about 2.5 million blood lead tests are received by CDC each year and are grouped into a reportable surveillance format after careful cleaning and deduplication to ensure only one test per individual. These data reveal that over the 2012-2018 period about 17% of children under age 6 in those jurisdictions were tested for BLLs. In 2012, 5.2% had BLLs above 5μg/dL, but that percentage has dropped by half in 2018.

Recent evidence from a number of studies suggests that some children with elevated BLLs are not identified, because surveillance is not universal (Roberts et al., 2017; Gazze, 2022). Abbasi et al. (2022) use data from Illinois over 2010-2014 to compare *de jure* universal screening against targeted screening. Some U.S. states require *de jure* universal screening while others target screening. They estimate that 6,626 untested children had a BLL above 5μ/dL, in addition to the 18,115 detected cases. 83% of these undetected cases should have been screened under the current policy. They conclude that model-based targeted screening can improve upon both the status quo and expanded universal screening.

Teeth are an important complementary biomarker of lead exposure. Like bone, teeth accumulate lead over the long term, but they are superior to bone as an indicator of cumulative exposure because the losses from teeth are much slower (Altshuller et al., 1962; Barbosa et al., 2005).[3] Childrens' teeth are relatively easy to collect and analyze. Their teeth have a unique histology that make this biomatrix a time-capsule for retrospective exposure analysis of fetal and early life (Yu et al., 2021). Gerbi et al. (2022) assessed the prenatal relationship between child tooth dentine and maternal blood lead measurements.

[3]Lead stored in bones is continually released back into the bloodstream, a process known as endogenous contamination. This process is particularly significant for children and pregnant women. The continuous growth of young children implies constant bone remodeling for skeletal development, and pregnancy causes an increase in bone remodeling.

Trimester-averaged and weekly child dentine lead measurements were highly correlated with maternal blood levels in the corresponding trimesters. They also estimated maternal lead exposure during the 2nd and 3rd trimesters of pregnancy from weekly child dentine profiles. The predicted trimester-specific maternal lead levels were significantly correlated with actual measured blood values.

It is worth explicitly noting that measuring BLLs to obtain estimates of the incidence of lead exposure is unique among criteria pollutants regulated by EPA. We believe there are two main reasons why this happens. First, while there are accurate tests to measure BLLs since the 1970s, the field of environmental health has lacked accessible biomarkers to identify elevated risk to toxic exposure. Second, once toxic exposure is identified, there is a clear protocol to be followed in the case of lead, but not for other criteria pollutants.

According to CDC guidelines, if a child has lead in their blood above the reference value, their doctor may recommend follow-up services. These include finding and removing lead from the child's environment, feeding the child a diet high in iron and calcium, connecting the child to early educational services, and scheduling follow-up blood testing. For more severe cases, there is chelation therapy: a medication given by mouth binds with lead so that it is excreted in urine. Early identification of lead in the blood is key to reducing the long-term effects of lead exposure.

2.3 Animal experiments

Animal studies can be informative with respect to the effects of lead. Advantages include the ability to conduct randomized trials and examine different levels of exposure. Disadvantages include the fact that results might not translate into outcomes for humans and the high levels of exposure in animal studies. Nonetheless, we view animal studies as informative and review a number of studies below.

For both animal studies here and human studies in later sections, we separately discuss fertility, mortality, and infant outcomes. Some studies have multiple outcomes and so will

be discussed more than once.

Fertility

Maternal lead exposure has been shown to affect fertility in animal studies. In Sharma et al. (2012), for example, female Swiss mice were treated orally with lead acetate for three months. After two months of exposure, two male mice were introduced in each group of ten females. Relative to the control group, in the treated group there was a decline in implantation sites and litter size. In a different animal experiment still focused on maternal exposure, lead caused abortion. Aprioku and Siminialayi (2013) initially allowed Wistar albino rats to mate freely until a female rat was pregnant. Pregnant rats were then randomly distributed into three experimental groups of six rats each, and two groups were exposed to different levels of lead nitrate by oral gavage from day 0 of pregnancy until delivery. Relative to the control group, lead exposure significantly inhibited maternal body weight gain and caused abortion in a dose dependent manner – abortion of all pregnancies in the high dose group, and two thirds in the low dose group. In the latter group, the average number of births were also lower than the control group.

Paternal lead exposure has also been shown to affect fertility in animal studies. Al-Juboori et al. (2016) exposed one group of 20 Swiss Webster male mice to a high dose of lead acetate dissolved in drinking water for four months, another group with a low dose, and a third group was left unexposed. At the end of the treatment, each male mouse was housed with two females. The results showed a significant reduction in both the percentage of implantation rate and the number of offspring in the high dose group (average paternal BLL of 28μg/dL), but no difference for the lower dose group (average paternal BLL of 23.5μg/dL). One potential explanation for such results is that lead exposure reduces sperm quality and DNA integrity in mice. For instance, Li et al. (2018) exposed four groups of ten adult male mice to different lead acetate concentrations in drinking water for six weeks. Relative to a control group, high lead exposure affected sperm motility and increased the percentage of

spermatozoa with abnormal morphology. Furthermore, percentages of sperm cells showing DNA breaks and chromatin structure damage increased significantly.

Mortality

Maternal lead exposure has been shown to affect mortality. In Aprioku and Siminialayi (2013), which also studied fertility, about a third of births in the low dose group were stillbirths. Neonatal mortality recorded between days 1 to 15 post-delivery reached 100% in the low dose treated group, but was 0% in the control group.

Lead exposure can weaken the immune system and induce mortality. Thind and Yusuf Khan (1978), for instance, show that lead intoxication of mice increased mortality due to experimental wild Langat virus infection. Groups of 3- to 4-week-old mice were fed with either 5 grams of lead chloride or sodium chloride per 1,000 grams of food. After 4 weeks of feeding, lead fed mice had a mean lead level of 71.2 μg/100ml of blood as compared to 3.8 μg in the control mice. Twenty control and lead intoxicated mice were observed for 21 days to determine mortality after viral infection. Mortality was 43% higher among lead fed mice.

Infant Health

Exposure to lead in utero and after birth also affects health outcomes. In Sharma et al. (2012), relative to the control group, in the treated group there was a decline in body weight of pups. In Aprioku and Siminialayi (2013) body weights of offspring of control rats increased over time, but those of low dose treated rats decreased significantly. Dyatlov and Lawrence (2002) exposed mouse pups to lead acetate starting at birth via milk from mothers. After weaning (21 days after birth), lead was administered via drinking water. At day 22 after birth, young mice were infected with the bacterium Listeria monocytogenes. Mice exposed to lead exhibited enhanced and prolonged sickness relative to mice in the control group, due to Pb/cytokine-dependent processes. This finding indicates that immune systems may be weakened by lead exposure, which might worsen bacterial infection.

2.4 From animal experiments to human studies

While in laboratory settings one can reliably study the biological effects of lead exposure because all other factors are held constant, the consequences of lead exposure in the human population are unclear. Individuals can take actions to avoid or remediate exposure. In fact, real world estimates reflect not only the biological effects on the human body but also all of the defensive measures that individuals take to protect themselves, such as wearing masks, replacing contaminated topsoil in backyards and playgrounds, removing lead painting, and drinking bottled water (Deschênes et al., 2017; Ebenstein et al., 2017). The importance of these behavioral responses will depend on how informed individuals are about pollution and its effects on health, and whether they can afford and are willing to make those defensive investments.

Real world estimates of the relationship between health and pollution arise from observational data combined with statistical methods, mostly based on regression approaches, to attempt to hold all else constant like in the animal experiments. These methods "adjust" for observed confounding factors by including available measures of behavioral, socioeconomic, and locational differences as covariates in the regression model.

Recent analysis suggests that associational or regression approaches to inferring causal relationships on the basis of adjustment with observable confounders can be unreliable for three reasons: unobserved confounding variables, selection bias, and measurement error (Dominici et al., 2014). Although observational studies document a statistical association between pollution and health, the evidence may not reveal causal effects.

Because air pollution is not randomly assigned across locations, observational studies may not adequately control for a number of potential unobserved confounding determinants of health outcomes (Chay and Greenstone, 2003). For example, areas with higher pollution levels also tend to have higher population densities, different economic conditions, and higher crime rates, all of which could impact health. In addition, the lifetime exposure to pollution might be unknown. The analysis implicitly assumes that the current pollution

concentration observed at a site accurately measures each resident's contemporaneous and lifetime exposure.[4]

Estimates from observational studies may also be unreliable because of selection bias. Some fetuses may die due to lead exposure during pregnancy, so fertility will be lower. Survivors will end up being different from what one would observe in the counterfactual world with no exposure. If survivors are stronger, average birth weight may be higher and infant mortality lower. If instead they survive but experience developmental issues during pregnancy and/or are born prematurely, then average birth weight may be lower and infant mortality higher. Because lead exposure may cut off the bottom of the distribution of outcomes and shift the distribution, accounting for selection is crucial to identify causal effects.

Another challenge of observational studies is measurement error in pollution exposure. Because exposure is not measured exactly where individuals live or spend most of their time, but rather approximated by measurements in the closest available monitors or pollution sources, measurement error is unavoidably introduced. If measurement error is classical, there will be attenuation bias in the estimates of the relationship between pollution and health. However, this may not hold with several measures of pollution. Errors may correlate with the actual value of the underlying pollution measure, and the variance of the measurement error might vary with distance from pollution monitors (Lleras-Muney, 2010; Knittel et al., 2016). In such a situation, attenuation bias is not necessarily the case.

As highlighted in the introduction, quasi-experimental (QE) techniques provide an opportunity to improve understanding of the relation between pollution and human health (Dominici et al., 2014). In a QE study, the researcher compares outcomes between a treatment group and a control group, just as in a classical experiment. Nevertheless, treatment happens because of politics, accidents, regulatory actions, or some other actions beyond the researcher's control. The key difference with an observational study is that the QE approach

[4]The shorter possible exposure period and more limited geographic mobility of children may allow to more easily determine the effects of pollution on children relative to adults (Currie et al., 2014).

is devoted to identifying treatment-induced variation in pollution that plausibly mitigates biases in the estimated relationship between human health and pollution.

3 Quasi-experimental work

In this section we discuss research that uses quasi-experimental tools to estimate the impact of lead exposure on fertility, mortality, and infant health. In total we have identified nine papers. In terms of vectors of exposure, the majority (6) focus on the consequences of lead-contaminated water on infant health. Three examine impacts of ambient airborne lead concentrations, and one of these also includes an analysis on soil lead. In terms of outcomes, two papers examine fertility, three explore mortality, and the remaining four investigate infant health.

As we mentioned in the introduction, almost all of the papers we survey examine the effects of lead exposure directly on outcomes. One exception is Clay et al. (2022), which examines child BLLs for the county-years for which data are available. The remaining papers discussed here do not include child BLL analysis, largely because of a lack of available data.[5] It is worth keeping in mind that other evidence, which we reviewed in the background section, shows that exposure to lead in air, water, and soil does increase child blood lead levels.

Fertility

The quasi-experiment in Grossman and Slusky (2019) is the Flint water crisis. The state appointed emergency manager switched Flint's water source from the Detroit Water and Sewerage Department to the Flint River in April 2014. Corrosion inhibitors were not added to the water, causing lead from lead service pipes to leach into drinking water. The elevated water lead levels and child BLLs did not become known until September 2015. The city switched back to the Detroit water system in October 2015. Grossman and Slusky (2019)

[5]Systematic CDC data on child BLL at the county-year level do not appear to be available before 2005. Further, not all states participate in the data collection.

study the impact of the Flint water crisis on fertility using U.S. vital statistics birth data for the period 2008-2015. Flint is compared to other cities in Michigan and to other cities in the United States. The authors use difference-in-differences and synthetic control methods to estimate what would have happened in Flint in the absence of a crisis.

The authors find that the crisis resulted in a significant decrease in fertility rates. Using data from Michigan, the results suggest that the general fertility rate (GFR) in Flint fell by 7.5 live births per 1,000 women aged 15-49, representing a 12% decline. Log births fell by 15-18%. Estimates using synthetic control are somewhat larger. Analysis using all cities in the United States gives somewhat smaller estimates. The lack of knowledge of the higher lead content and time use data on sexual activity suggest that the declines in fertility were the result of fetal death and miscarriage.

Clay et al. (2021) use two different quasi-experiments. The first quasi-experiment leverages the October 1979 and July 1985 policy deadlines regarding the phasedown of lead in gasoline. The effect of the declines are compared in counties with and without highways in the 1944 Interstate Highway Plan. The highway plan was designed to achieve a range of policy goals including connection of major metropolitan areas, national defense, and connection with routes in Canada and Mexico. Thus, the plan can be thought of a quasi-experimental allocation of highways to counties. The analysis draws on U.S. vital statistics data for 1978-1988, 1980 and 1990 census data on children ever born, and EPA monitor data covering 40% of the U.S. population.

The authors find that declines in lead in gasoline caused significant increases in the general fertility rate and completed fertility. In IV estimates, the increase in the general fertility rate for women ages 15-44 years implied by the average observed decrease in airborne lead is 4 births per 1,000 women per year, which is 6% of mean fertility. Using the 1980 and 1990 census data on children ever born, IV estimates also show that the increase in completed fertility implied by the average observed decrease in airborne lead is 0.14 children per woman, which is 6.4% of mean fertility.

The second quasi-experiment uses the highway plan to predict soil lead levels. The U.S. Geological Survey sampled soil characteristics including soil lead during the 2000s using a grid-like sampling scheme. Soil lead levels were measured for 4,857 sites in 2,096 counties, covering 70% of the U.S. population. The 1944 highway plan generates variation in how much lead from gasoline was deposited and historically accumulated in the topsoil. This intent-to-treat (ITT) strategy addresses the unobserved association between lead in soil and defensive responses, and measurement error associated with the potential disconnection between soil sampling sites and household residences.

Clay et al. (2021) find that having an above median soil lead level resulted in a significant decrease in fertility rates. Estimates suggest that counties with lead concentration above the median have general fertility rates in 2005 that are 7.8 births per 1,000 women per year lower than counties below the median, which is 11% of mean fertility. To provide a sense of the magnitude, the paper compares the effects of lead to quasi-experimental estimates of the effects of the birth control pill on fertility from Bailey (2010, 2013). In the late 1950s and early 1960s, the pill decreased annual general fertility rates by approximately 7 births per 1,000 women of childbearing age (15-44 years). With the caveat that contraceptives are used voluntarily, and lead exposure is almost surely involuntary, the fertility effects of airborne lead in 1978-88 and lead in soil in the 2000s are similar in magnitude to the impact of the pill.

Infertility is costly, because it leads to additional expenditures by infertile couples and because some couples have fewer children than otherwise desired. Fertility-related expenditures are not well documented, but appear to run in the tens of thousands of dollars. The cost of not having a child is also difficult to assess. Suppose that, on average, the satisfaction parents would obtain from having children would be at least as large as the amount spent in bringing them up. In 2022 USD, the USDA estimates that the cost was $288,450, not including expenditures for college. This is likely a lower bound on the actual cost of not having a child.

Mortality

Troesken (2008) investigates the relationship between lead water pipes and infant mortality at the turn of the twentieth century using data from U.S. Census Bureau reports and city-specific water quality reports for the period 1897-1921. The main estimates in the paper come from comparisons of infant mortality in cities with lead service lines to the same measure in cities without leaded service lines, accounting for the age of the pipes as well as the corrosiveness of the water. Troesken (2008) finds that cities with leaded service lines have 39% higher infant mortality (139.5 more deaths per 1000 live births) than cities without such lines.

In addition to this straightforward comparison, there are two primary quasi-experiments explored in the paper. First, an instrumental variables strategy that leverages the fact that leaded lines were more likely to be used when (i) public companies installed the initial service lines, (ii) water companies made home owners pay and maintain their own lines, and (iii) the underlying water system was complex. The second quasi-experimental analysis in Troesken (2008) uses the fact that water Ph and the age of the service lines both affect the likelihood that lead is leached from the pipes into the water. Newer pipes leach more lead than older ones, and there is a non-linear relationship between water Ph and how much lead is expected to leach from the pipes into the water. Both analytical strategies yield similar results as to the simpler OLS estimates, finding large increases in infant mortality when leaded pipes are used and when more corrosive water is present in leaded service lines.

The second paper with a quasi-experimental estimation strategy is Clay et al. (2014). The authors examine the impact of lead exposure on mortality using data on lead levels in drinking water and mortality rates from 172 medium and large sized U.S. cities from 1900 to 1920. Similar to Troesken (2008), the main analyses leverage an instrumental variable related to water acidity. Having leaded pipes at average water Ph increases infant mortality by .074 per 100,000 (19%).

Finally, Clay et al. (2022) use annual variation in fugitive lead emissions from industrial

plants, which are unanticipated, interacted with annual variation in wind speed as a source of exogenous variation in EPA monitored air lead levels. Plants report both stack lead, which is from normal plant operations, and fugitive lead, which is unexpected and intermittent, to the Toxics Release Inventory (TRI). Controlling for stack lead and other chemical emissions and their interaction with wind speed, increases in fugitive lead interacted with wind speed increase monitored air lead levels. Wind spreads the fugitive lead, which tends to be emitted close to ground level, further. Annual county level data on child blood lead are available for a subset of county-years. These data show that fugitive lead had a statistically significant and large impact on child BLLs, while stack lead had no effect. The latter likely reflects the fact that stack lead is typically emitted from high stacks and disperses over a large geographic area, and is consistent with the null effect on air lead found by examining monitor data.

The exogenous variation in EPA monitored air lead levels makes it possible to estimate the causal effect of lead on infant mortality for large urban counties, representing 26% of the U.S. population. Clay et al. (2022) find that higher air lead concentration causes higher infant mortality in the first month and in the first year. The observed decline in fugitive emissions between 1988 and 2018 reduced infant deaths by 39-54 deaths per year. The paper also finds that higher air lead concentrations increased deaths from causes that science suggests may be related to lead, such as low birthweight, sudden unexplained infant death (SUID), and respiratory causes, but had no impact on causes of death that science suggests should be unrelated to lead, such as deaths from congenital anomalies and deaths in the perinatal period.

The monetized value of these health damages is large. Current US EPA estimates for the Value of Statistical Life (VSL) are $11.2 million in 2022 $ (US EPA, 2006). Thus the *annual* cost of fugitive lead emissions are between $439 and $608 million. Historically, having leaded water pipes caused higher infant mortality, with Troesken (2008) finding an increase of 39% and Clay et al. (2014) finding a 19% increase. While conducting a complete damage assessment for the lifetime costs of lead water pipes is difficult, these large effect sizes clearly

indicate that the monetized health consequences from using leaded pipes to transport water is immense. Moreover, in each case, the VSL based calculation only captures damages from infant mortality, and does not include costs of non-fatal diseases, loss of IQ, reduced birthweight, or costs of mortality in other age groups.

Infant Health

Overall four quasi-experimental studies examine the effect of lead on infant birthweight or the prevalence of low birthweight. Both Abouk and Adams (2018) and Wang et al. (2022) use synthetic controls methodology to estimate the effect that the water crisis in Flint, Michigan had on infant health. The synthetic control method used by both studies creates a prediction for what would have happened to infant health in Flint had the water crisis not occurred, using data from similar cities unaffected by the crisis as the basis for the counterfactual. Abouk and Adams (2018) analyze birth outcomes during the early stages of the water crisis from April 2014 to October 2015. They select a number of cities that had similar pre-crisis average birthweights to children born in Flint to form the counterfactual estimate. Using this approach, the analysis finds that higher water lead levels are associated with a 49 gram decrease in birthweight. Wang et al. (2022) allow infant health measures from 162 U.S. cities to contribute to their counterfactual estimate. The analysis yields a similar result, finding that the Flint crisis lowered birth weights by 32 grams on average, and increased the share of low-birthweight births by 1.8 percentage points.

The final two papers examining infant birthweight are not related to the issues in Flint. Dave and Yang (2022) analyze the relationship between lead in drinking water and birth outcomes using data from Newark, New Jersey, between 2000 and 2007. They employ a difference-in-differences approach, exploiting a natural experiment in which one water treatment plant in the city switched to a less effective water treatment method (sodium silicate) that allowed for more leaching of lead from pipes carrying water. An advantage of this paper compared to the work that only examines Flint is that the analysis is within-

city, comparing outcomes for those that live close to one another, but who are served by different water treatment plants. Despite the setting being different, the analysis yields similar findings to the results seen in Flint: children born to mothers living in homes treated by the plant using the less effective corrosion inhibitor, after the switch were on average 31 grams smaller. The switch also induced a 1.5 percentage point increase in infants weighing less than 2,500 grams at birth (i.e., low birthweight).

Tanaka et al. (2022) explore the results of a quasi-experimental setting where U.S. changes in stringency for ambient airborne lead pollution caused a displacement of lead battery recycling from the U.S. to Mexico. This change caused a 24 gram decrease in the birthweight of babies born within two miles of Mexican recycling plants that experienced increased battery recycling due to the U.S. policy change. Similar analyses show a 2 percentage point increase in the share of children considered low-birthweight. The primary specification here is a difference-in-differences approach, and to better account for selection and omitted variables that may be related to both health at birth and location of residence, the analyses use a control group of infants who lived between two and four miles away from the recycling plants. In addition to helping to estimate the effect of lead pollution on infant health, this work highlights the importance of considering the potential displacement effects of environmental policies from wealthier nations to countries with more vulnerable populations.

Finally, Bui et al. (2022) examine how birth outcomes are affected by in utero exposure to lead. The authors use detailed data on all births in the Charlotte metropolitan area from 2004 to 2009 and exploit a quasi-experiment where in 2007 a large automotive racing group (NASCAR) voluntarily switched from using leaded to unleaded fuel. Deleading caused a large reduction in airborne lead exposure for those living close to The Charlotte Motor Speedway.[6] Prior work has shown that the voluntary deleading of automotive races in 2007

[6]The largest races at the racetrack use around 39kg of Tetraethyl Lead (TEL) in each race. This is more lead than is released by 70 percent of all lead-emitting facilities in the TRI, and about as much as the average airport uses in an entire year.

reduced airborne lead pollution, decreased rates of elevated blood lead in children, reduced elderly mortality rates, and improved standardized test scores in schools near racetracks (Hollingsworth and Rudik, 2021; Hollingsworth et al., 2022). The main analysis in Bui et al. (2022) is a difference-in-differences comparison that examines how changes in birth outcomes before and after deleading differed between those that were born to mothers that lived farther away from the racetrack (beyond 10km) and those that lived closer to the race track (within 4km). Findings show that lead exposure in utero led to a 103 gram decrease in birth weight and a 4.5 percentage point increase in the probability of an infant being considered low birthweight.

A large literature has explored the importance of birthweight, finding that higher weight at birth improves both short-run (e.g., lower infant mortality) and long-run outcomes (e.g., increased probability of completing high school, improved educational outcomes, and increased adult earnings) (Bharadwaj et al., 2018; Figlio et al., 2014; Behrman and Rosenzweig, 2004; Black et al., 2023). The most easily monetized result is the link between birthweight and future earnings, where a 500 gram increase in birthweight should increase annual adult earnings by about 2.8% (Lambiris et al., 2022) and being low birthweight (less than 2,500 grams) causes a 2.5 percent reduction in permanent income (Bharadwaj et al., 2018). As with the infant mortality results, the monetized value of these health damages is substantial. For those in the United States, the present value of expected future earnings at birth is $483,626.33 in 2022 USD.[7] For every child that is low birthweight, this represents a $12,091 decrease in the net present value of lifetime earnings. Similarly, lifetime earnings increase by $13,542 for every 500 grams heavier a child is at birth. This helps put some of the results linking lead exposure to worse infant outcomes into context. For example, the Newark water crisis was estimated to decrease birthweight by around 32 grams (Dave and Yang, 2022). Considering only the earnings losses, the net present value of these damages is $867 per

[7]Chetty et al. (2014) report that the present value of expected future earnings at age 12 is $689,535.50 in 2022 USD using a 3% real discount rate (5% discount minus 2% wage growth). Thus, at birth (age 0) the present value is $483,626.33.

affected child.

4 Implications for Policymakers

The quasi-experimental studies in our review provide evidence that lead exposure harms fertility, increases infant mortality, and in some cases adversely impacts infant birth outcomes. The studies highlight the importance of reducing lead exposure in water supplies and other sources to protect the health of vulnerable populations. In addition, this work emphasizes the need for policymakers to carefully consider the potential displacement effects of environmental regulations. That is, stricter regulations in one region may lead to increased lead exposure in other regions due to shifts in industrial activities. Ultimately, a comprehensive and well-coordinated approach to reducing lead exposure is necessary to protect public health and improve infant health outcomes globally.

We see these studies as having three implications for policymakers. First, in light of the significant levels of child lead exposure in developed and developing countries, we need to know more about in utero exposure. One possibility would be to require testing for lead when maternal blood testing is done. Many countries routinely test women for Rh factor, so BLL tests could be done in conjunction with this or other routine testing. In cases where tests of BLLs via venous draws are not feasible, less expensive finger prick tests could be used. In concert with this, we encourage policymakers to consider systematic collection of baby teeth. This would provide important retrospective evidence on exposure. Furthermore, for children with high levels of teeth lead, intervention to reduce exposure and improve outcomes may still be possible.

Broad surveillance testing would make it possible to take immediate action to investigate and address the causes for mothers with high BLLs. Calcium supplementation has been shown to modestly reduce BLLs in pregnant women (Ettinger et al., 2009). Air and water filters could be used to reduce exposure, paint can be sealed, soil can be replaced or clean

topsoil can be put on top. This could prevent further exposure in utero and as an infant. Depending on the cost, it may be efficient for firms to implement continuous monitoring to more rapidly detect and address fugitive emissions or even relocate to less densely populated areas. To the extent that there is community level monitoring of air lead and water lead, maternal BLLs could be linked to exposure through air and water, providing new data on dose-response curves. Broad survaillance testing would also permit maternal BLLs could be linked to infant and later life outcomes, providing new evidence on how in utero lead exposure affects individuals throughout life.

Second, more quasi-experimental studies are needed on all three topics. The need for more studies of the effects of lead on fertility and infant mortality is particularly critical. Studies on these topics are likely to have impact on policy, because of the high cost of infant mortality and infertility. Ideally studies would not rely on crises. It can be difficult to estimate robust, generalizable effects from small scale crises, like the water lead crisis in Flint, Michigan. More and better studies are needed for air lead.

Third, because of the large social cost to waiting, policymakers should not wait for more studies, for pregnant women to be routinely screened for BLLs, or both. Given the large magnitudes of effects, we need to use existing quasi-experimental studies in regulatory impact analyses when considering reductions in lead limits. It is worth explicitly noting that we do not require additional studies or data when considering reductions in PM2.5. Existing dose-response functions are used in that context and existing empirical relationships can be used for lead as well.

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Figure 1: Blood lead levels across time, 1976-2018

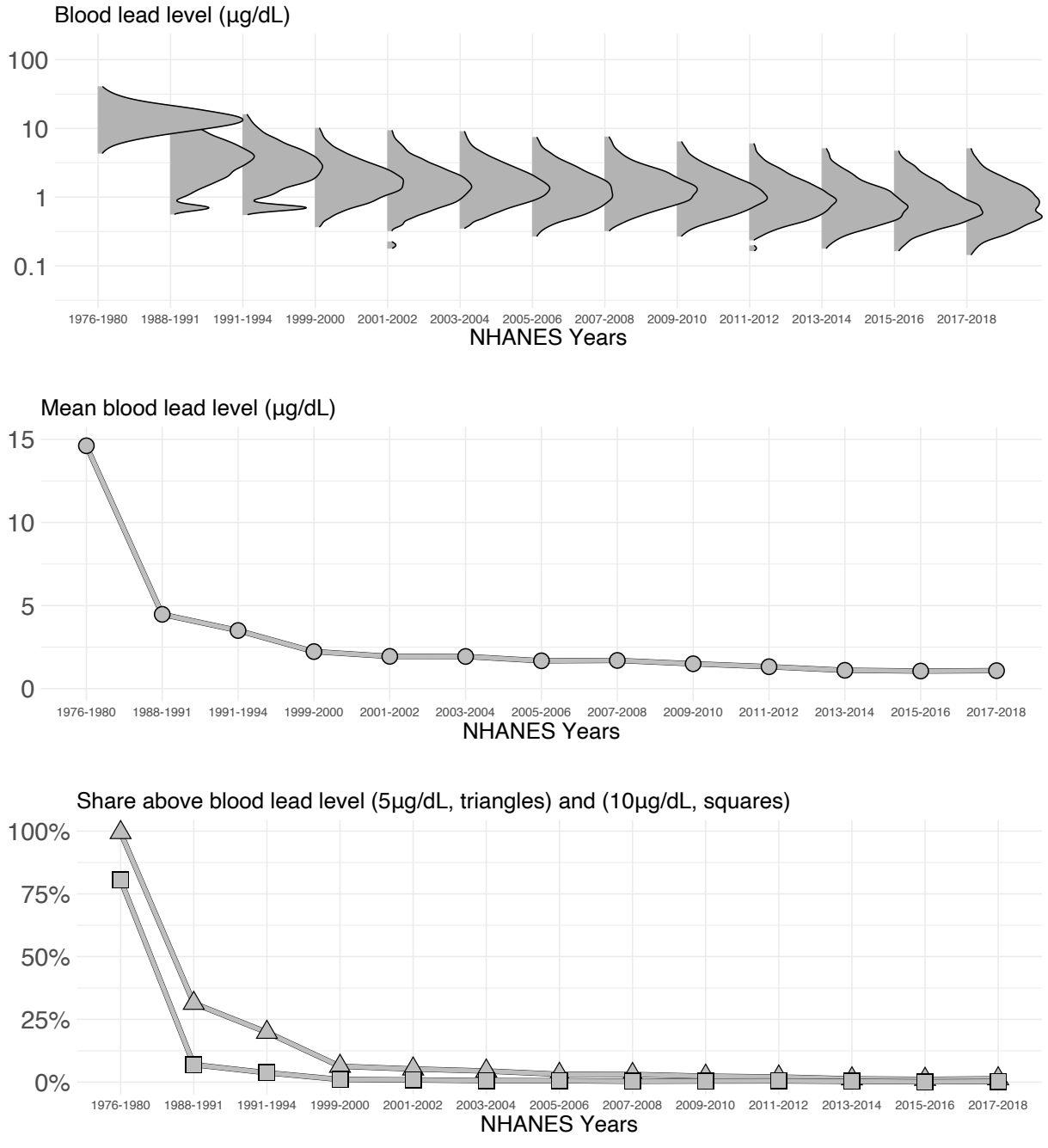

Figure 2: Blood lead levels across time, 1999-2018

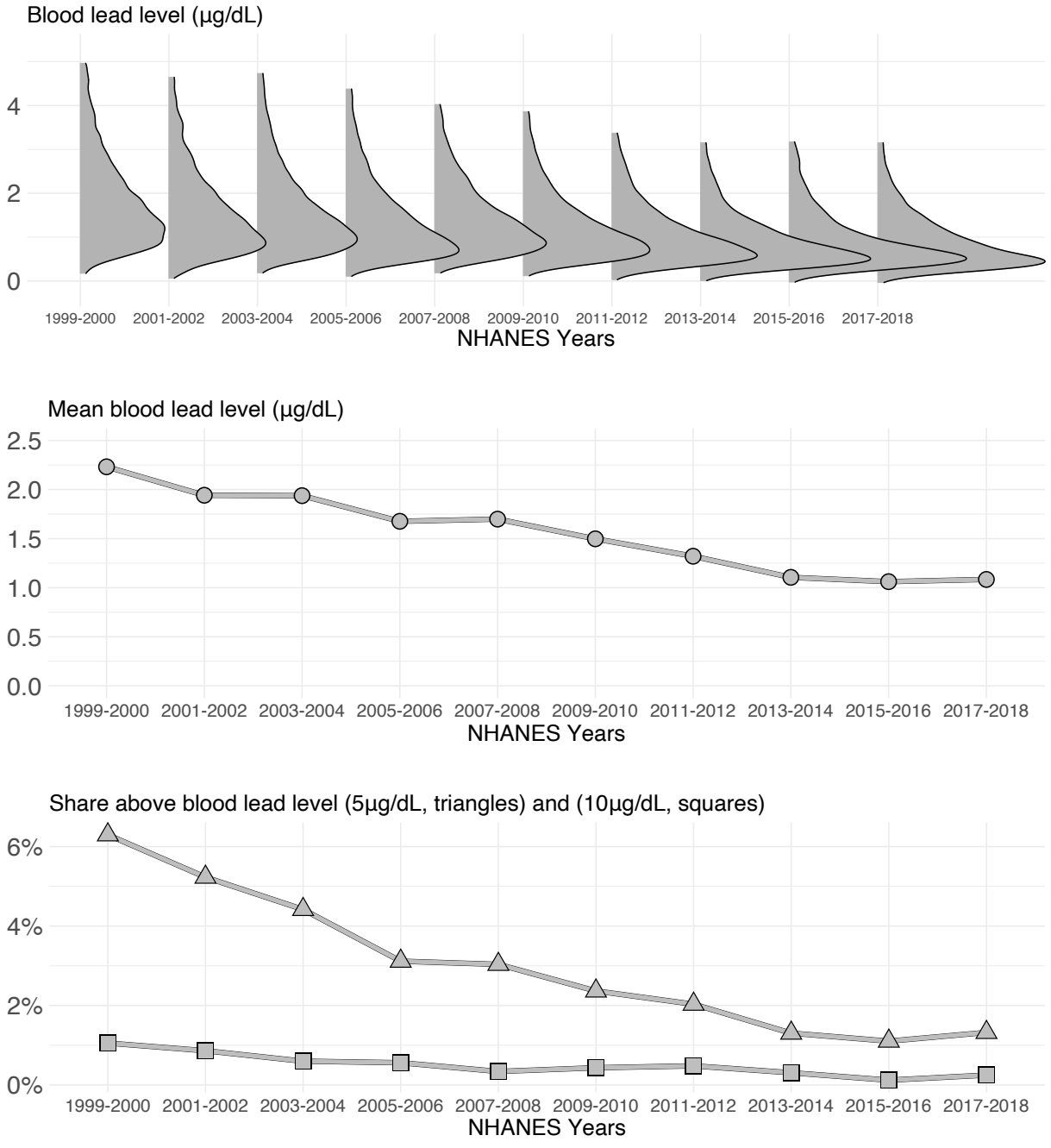

Table 1: Summary of main findings from nine quasi-experimental papers linking lead exposure to infant health

Outcome	Paper	Vector	Findings	Data	Years covered
Fertility rate					
	Grossman and Slusky (2019)	Water	12% reduction	All live births in Michigan	2008 to 2015
	Clay et al. (2021)	Air	6% reduction	US County-Level Natality Data, 1978–2007	1978 to 1988
	Clay et al. (2021)	Soil	11% reduction	US County-Level Natality Data, 1978–2007	2000 to 2009
Infant mortality					
	Clay et al. (2014)	Water	19% increase	172 medium and large sized cities	1900 to 1920
	Troesken (2008)	Water	39% increase	Massachusetts towns	1900
Birthweight					
	Abouk and Adams (2018)	Water	49g decrease	Cities with similar pre-2014 birthweight as Flint, MI	2005 to 2014
	Wang et al. (2022)	Water	32g decrease	U.S. Births from 162 cities	2008 to 2015
	Dave and Yang (2022)	Water	31g decrease	All births in Newark, NJ	2011 to 2019
	Tanaka et al. (2022)	Air	24g decrease	Births in Mexico within four miles of a battery-recycling plant	2005 to 2015
	Bui et al. (2022)	Air	103g decrease	Births in Charlotte, NC near automotive racing site	2004 to 2009
Low birthweight					
	Wang et al. (2022)	Water	1.8 pp increase	U.S. Births from 162 cities	2008 to 2015
	Dave and Yang (2022)	Water	1.5 pp increase	All births in Newark, NJ	2011 to 2019
	Tanaka et al. (2022)	Air	2 pp increase	Births in Mexico within four miles of a battery-recycling plant	2005 to 2015
	Bui et al. (2022)	Air	4.5 pp increase	Births in Charlotte, NC near automotive racing site	2004 to 2009