Lead Effects Through Generations*

Xinming Du† Krzysztof Zaremba‡

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Abstract

While economic and health dynamics across generations have been well studied, the role of environmental factors in driving intergenerational persistence remains underexplored. This paper examines the first- and second-generation effects of leadborne pollution on health and fertility outcomes. We exploit the phase-out of leaded gasoline in Mexico in the 1990s, which led to a sharp decline in air lead pollution. Using a shift-share design to identify variation in prenatal lead exposure, we find that a full reduction in lead exposure results in a 1.97 per thousand reduction in fetal deaths, a 0.53 per thousand increase in birth rates, and a 3.33 per thousand decrease in infant mortality. Female children exposed to higher in-utero lead levels are more likely to give birth earlier and less likely to migrate. In the second generation, the effects of in utero exposure vary by local socioeconomic conditions: in better-off municipalities, long-term effects are null, whereas in marginalized municipalities, children of exposed mothers experience significantly lower birth weights and elevated rates of preterm birth. These findings highlight how structural disadvantage mediates the intergenerational transmission of environmental shocks.

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†Department of Economics, National University of Singapore. Email: xdu@nus.edu.sg
‡Instituto Tecnológico Autónomo de México. Email: zaremba@itam.mx

1 Introduction

The dynamics of economic and health status across generations have been extensively explored in the intergenerational mobility literature (e.g., Black and Devereux, 2011; Chetty et al., 2014). Parental income and childhood challenges are strongly correlated with children's future outcomes, often leading to lower-paying jobs. This correlation is typically explained by the long-term effects of early life adversity on later opportunities. While much attention has focused on economic shocks, the role of environmental factors like pollution in contributing to intergenerational persistence in negative outcomes has been largely overlooked. Environmental pollution is a significant shock that can affect both health and economic outcomes across generations, but research on its intergenerational effects remains limited.[1]

This paper fills in this gap by examining both the immediate and intergenerational effects of pollution, with a focus on its impact on health and fertility outcomes in Mexico. Air pollution has well-established adverse effects on human health and productivity, contributing to 6.7 million premature deaths annually (Murray et al., 2020). While extensive research has examined the direct effects of air pollution on physical and mental health (e.g., Chay and Greenstone, 2003; Deryugina et al., 2019) and labor outcomes (e.g., Zivin and Neidell, 2012), less is known about its intergenerational effects. Pollution can affect future generations biologically, by influencing gene expression (Genc et al., 2012), and economically, by impacting health, productivity, and income. These economic impacts, in turn, shape household decisions, including investments in children's health and education. Failing to account for these intergenerational effects may lead to an underestimation of the broader costs of pollution, especially in developing countries where pollution is more severe.

Our study focuses on one specific type of air pollution: leadborne pollution. Lead is a harmful pollutant that affects human health, fertility, and cognitive function, with larger estimated elasticities compared to other air pollutants (e.g., Reyes, 2007, 2015). Its effects are most severe during early childhood, when children's developing bodies are vulnerable. Unlike other non-metal pollutants, lead is not easily eliminated from the body once absorbed, making its long-term effects more concerning. The human

[1] One exception is Colmer and Voorheis (2020), which examines how the Clean Air Act in the U.S. influenced college attendance in subsequent generations.

body cannot distinguish lead from calcium, which means populations in countries with higher calcium levels may be less affected by lead pollution. Nevertheless, lead remains a significant issue worldwide especially in developing countries, as evidenced by the fact that 22% of children in Mexico aged 1-4 have blood lead levels above the recommended threshold of 5 μg/dL (ENSANUT, 2023).

We exploit the sharp removal of leaded gasoline in Mexico to explore the effects of leadborne pollution. Leaded gasoline, introduced globally in the 1920s to improve vehicle efficiency, became a major source of toxic pollution, impacting children's health and cognitive development. Mexico's phaseout of leaded gasoline was part of a global trend, with unleaded gasoline becoming available in 1990. Over the following years, the lead content in gasoline was gradually reduced, and by 1997, leaded gasoline was entirely eliminated. This policy shift resulted in a rapid decline in lead exposure, with sales of leaded gasoline dropping from over 2 million barrels per month in the early 1990s to nearly zero by 1997, while sales of unleaded gasoline steadily increased. These changes, marked by substantial reductions in lead content, offer a natural experiment to examine both the immediate health consequences and the intergenerational effects of removing lead from gasoline.

We study the first and second-generation effects of prenatal lead exposure, beginning with the first generation. Specifically, we examine how prenatal exposure to lead during the phase-out of leaded gasoline from 1990 to 2000 affected birth outcomes, such as fetal death rates, birth rates, and infant mortality. Our identification strategy uses a shift-share design, following (Clay et al., 2021), which leverages exogenous variation in prenatal lead exposure due to federal regulatory changes, combined with spatial heterogeneity in proximity to major roads.

We demonstrate a strong first stage, showing that airborne lead drops significantly with reduced leaded gasoline sales, confirming that the policy effectively reduced lead exposure. In contrast, ground monitor-reported total suspended particulates (PST) and satellite-derived aerosol optical depth (AOD) do not respond to changes in leaded gasoline sales. This suggests that the pollution reduction is specific to airborne lead, rather than a general improvement in overall air quality.

Our results for the first-generation show significant improvements in birth outcomes following the reduction in prenatal lead exposure. We find that a full reduction

in lead exposure corresponds to a 1.97 per 1,000 reduction in fetal deaths, a 0.526 per 1,000 increase in birth rates, and a 3.33 per 1,000 reduction in infant mortality. These effects indicate that reducing prenatal lead exposure not only improves fetal survival but also leads to better neonatal health. Notably, male infants appear particularly vulnerable to prenatal lead exposure, with a larger reduction in infant mortality for male infants compared to females, consistent with previous research on sex-based differences in fetal susceptibility to environmental toxins (e.g., Trivers and Willard, 1973; Catalano et al., 2006; DiPietro and Voegtline, 2017).

We find no evidence of selection into childbearing due to the lead exposure phase-out. Our analysis examines whether parental characteristics, including maternal age, education, and marital status, shifted in response to changes in lead exposure. The results show no systematic changes in these characteristics, suggesting that the improvements in birth outcomes are not driven by changes in the composition of parents. The observed effects on fetal and infant mortality are primarily due to the direct biological effects of reduced lead exposure rather than confounding demographic factors.

Moving to the second generation, we find that fertility and maternal characteristics are affected by prenatal lead exposure. Women exposed to lead in utero tend to have children at younger ages, with reductions in lead exposure resulting in a decrease in fertility rates among teenage and early 20s mothers. Specifically, reducing lead exposure from pre-policy levels to zero in areas with high road exposure would decrease teenage fertility by 22.74 per 1,000 women and fertility in the early 20s by 85.2 per 1,000 women, corresponding to 11% and 16.7% of the mean respectively. Furthermore, maternal characteristics such as age at childbirth were significantly affected, with a 0.15-year increase in maternal age observed in municipalities with high exposure to federal highways.

Moving to second-generation outcomes, we find no significant average effects of maternal in-utero lead exposure on birth outcomes including raw birth weight, low birth weight incidence, gestational age, or preterm birth. However, there are substantial heterogeneities across municipalities. In relatively well-off areas, we estimate precise null effects on these outcomes. In contrast, in disadvantaged municipalities, full maternal exposure to lead results in a 10.4-gram reduction in offspring birth

weight and a 0.46 percentage point increase in the probability of preterm birth. These results suggest that the intergenerational transmission of environmental shocks is not guaranteed but may depend on the ability to mitigate early-life harm, which is often more constrained in poorer settings. Consequently, the long-term impacts of pollution are not evenly distributed but tend to concentrate in communities already facing socioeconomic disadvantage.

Our paper addresses two key challenges in the study of intergenerational health effects. The first is linking early-life shocks experienced by one generation to health outcomes in the next. We overcome this by leveraging a unique feature of our data: second-generation birth records include detailed information on the mother's place and date of birth, allowing us to identify whether the mother, i.e., the first generation, was exposed to the lead phase-out while in utero. This enables a direct link between prenatal environmental exposure in the first generation and birth outcomes in the second.

The second challenge is focusing on a developing country where data is often limited. Most existing studies focus on developed countries (e.g., Grönqvist et al., 2020; Clay et al., 2021) where people generally have better access to healthcare and more options to avoid pollution. In contrast, developing countries like Mexico experience worse pollution and have fewer ways to mitigate it, leading to stronger immediate and intergenerational effects. Additionally, the impact of pollution can vary depending on the baseline pollution levels. Mexico, with its ongoing improvements in environmental quality, provides a wide range of pollution levels, allowing us to estimate how pollution affects health across different baseline conditions.

Our paper is related to two strands of existing literature. The first is the effect of lead pollution. Extensive research has shown the harmful impact of lead exposure on various outcomes, including infant mortality (Troesken, 2008), fertility rates (Grossman and Slusky, 2019), birth weight (Abouk and Adams, 2018; Wang et al., 2022), and educational outcomes (Reyes, 2015; Ferrie et al., 2012). Lead exposure comes from various sources, such as leaded water pipes (Ferrie et al., 2012; Clay et al., 2014), leaded gasoline (Clay et al., 2021; Aizer and Currie, 2019), and industrial emissions (Tanaka et al., 2022; Clay et al., 2022). Our paper adds to this literature by providing the first evidence on the second-generation effects of prenatal lead exposure.

We are also the first to study the effects of lead from traffic sources in developing countries, including first-generation effects. Existing literature on traffic-related lead emissions primarily relies on studies of leaded gasoline bans in the U.S. (e.g., Aizer and Currie, 2019; Clay et al., 2021) and Sweden (Grönqvist et al., 2020) to examine first-generation health, education, and labor outcomes. Some studies also use proximities to airports (Zahran et al., 2017) and the NASCAR exemption (Hollingsworth et al., 2020) to identify other sources of traffic lead, but all have focused on developed countries.

The second related strand of literature examines intergenerational mobility, focusing on how parental resources shape children's outcomes and the extent to which income, education, and social status persist across generations (e.g., Black and Devereux, 2011; Chetty et al., 2014; Chetty and Hendren, 2018; Card et al., 2022). Health has increasingly been recognized as part of this transmission process. Existing studies have documented the long-term impacts of parental conditions, such as exposure to economic shocks or limited access to education, on their children's health outcomes (Currie and Moretti, 2003; Aizer and Currie, 2014; Clark et al., 2021). Others show direct persistence in health across generations, including strong correlations in birth outcomes between parents and their children (Currie and Moretti, 2007; Chang et al., 2019; Giuntella et al., 2023). These findings suggest that early-life exposures affecting parents can have lasting effects on the next generation. An example illustrating this mechanism is the paper by East et al. (2023) that document in-utero exposure of mothers to Medicaid improves birth outcomes in the next generation. Our paper contributes to this literature by showing that the intergenerational transmission of environmental shocks is not uniform: while we find no average second-generation effects of prenatal lead exposure in well-off municipalities, significant harms persist in disadvantaged communities.

From a policy perspective, our paper highlights the public health benefits of the historical phaseout of leaded gasoline for on-road vehicles. The global effort to eliminate leaded gasoline has greatly reduced lead exposure, especially among children, leading to marked improvements in health outcomes. However, other sources of lead pollution remain significant risks. Industrial activities such as mining, smelting, and battery manufacturing, along with leaded aviation fuel used in propeller-driven aircraft, continue to contribute to lead exposure. In addition, everyday consumer

products, such as certain spices, cosmetics, cookware, and the improper recycling of electronic waste, contribute to contamination, especially in regions with weaker regulatory frameworks. These ongoing sources remain major concerns for lead contamination and disproportionately affect vulnerable populations. Policymakers must expand their focus to regulate these additional sources of lead pollution to continue making progress in reducing lead exposure and its associated health risks.

We also emphasize that the legacy effects of pollution from periods when environmental regulations were limited, may not be as large or irreversible as often feared. While pollution exposure in earlier years can have lasting impacts, these effects can be mitigated through later interventions. Investments in education, delaying maternal age, and increasing access to prenatal care are crucial steps that can significantly improve birth outcomes. These actions offer hope, as they demonstrate that the damage caused by historical pollution can be corrected through thoughtful policy efforts and public health initiatives. However, our findings also show that the ability to mitigate these harms is not equally distributed. In disadvantaged communities, where access to health and educational resources is more limited, the intergenerational effects of pollution are more likely to persist. This highlights the need for targeted interventions that enhance the capacity of vulnerable populations to buffer environmental shocks, ensuring that the long-term burden of pollution does not fall disproportionately on those least equipped to cope with it.

The rest of the paper is organized as follows. Section 2 provides a brief background overview, and Section 3 describes the data sources. Section 4 details the empirical strategy and presents results on the first-generation impacts of leadborne pollution. Section 5 explores the second-generation effects, including fertility decisions, maternal characteristics, and birth outcomes. Section 6 concludes the paper.

2 Background

2.1 Intergenerational effects of pollution

Intergenerational health effects have been widely studied, particularly through the lens of correlations between parents' and offspring's health outcomes. A prominent example of this is the link between a parent's birth weight and the health of their

children, suggesting that the conditions experienced early in life can have lasting effects that echo across generations. Studies such as Currie and Moretti (2007), Royer (2009), and Giuntella et al. (2023) document this pattern, highlighting the persistence of health disadvantages over time. These findings underscore the notion that early-life health shocks can set a trajectory that impacts not only the individual but also future generations. Another important strand of research focuses on the transmission of health capital across generations and its broader influence on socioeconomic outcomes. Poor health at birth or in early childhood has been shown to reduce educational attainment and labor market outcomes, and these disadvantages may persist and be passed down to the next generation (Venkataramani, 2011).

Pollution could have intergenerational effects. One medical mechanism through which pollution affects the second generation is by altering gene expression. Existing medical evidence suggests that pollution exposure modulates epigenetic markers, particularly DNA methylation (DNAm), which in turn may influence inflammation, disease development, and the risk of disease exacerbation (Rider and Carlsten, 2019). Air pollutants have been shown to change DNAm and reduce methylation levels. These effects have been observed across the human lifespan and are frequently linked to long-term negative health outcomes. After first-generation exposure, epigenetic modifications and changes in gene activity may be passed down to future generations. While evidence for transgenerational transmission exists in animal experiments (e.g., Whitelaw and Whitelaw, 2008; Pang and Curran, 2012; Berger, 2012; Aiken et al., 2016), there is currently no direct evidence in humans.

Focusing specifically on lead, lead pollution can have intergenerational effects via the mother-child link. Lead is stored in bones and can be released during pregnancy, exposing the fetus through maternal bone turnover. This process leads to the ingestion of lead by the developing baby, which can affect neurological development. The placenta, which is supposed to protect the fetus, is not effective at blocking heavy metals like lead, allowing it to pass into fetal tissues. During pregnancy, hormonal changes release lead from the mother's bones and teeth, where it has accumulated over the years due to environmental exposure. This exposure is harmful as lead is a neurotoxic metal and prenatal exposure can cause long-term neurocognitive damage (Rísová, 2019). Existing studies have shown that lead transport to the fetus

begins around the 13th week of gestation and continues until birth, with increasing concentrations of lead in fetal tissues as the organs grow. The blood-brain barrier, which is not fully developed, allows lead to enter the fetus's brain, where it can cause direct damage to brain structures (Barltrop, 1969; Mahaffey et al., 1973).

Beyond biological mechanisms, pollution may also affect future generations through broader socioeconomic channels. Exposure to pollution can serve as a negative shock to health and cognitive development (Currie and Neidell, 2005; Currie et al., 2009; La Nauze and Severnini, 2025), leading to reductions in educational achievement (Marcotte, 2017; Roth, 2017), productivity and earnings (Isen et al., 2017; Chang et al., 2019; He et al., 2019; Fu et al., 2021). These economic disadvantages can accumulate over time, creating a cycle that makes it more challenging for future generations to break free from poverty or poor health. This pattern mirrors other early-life shocks documented in the intergenerational mobility literature, where initial disadvantages have lasting effects on an individual's potential to succeed and improve their living conditions.

2.2 Lead pollution and regulation in Mexico

Lead exposure is a serious issue in Mexico. According to the current Official Standard, 1.38 million children aged 1-4 (17.2% of the total population in this age group) have lead poisoning, with blood lead levels $\geq$5 μg/dL (ENSANUT, 2023). The prevalence of lead poisoning is significantly higher among vulnerable groups: 23.7% in children facing greater deprivation, 28.7% in indigenous children, and 25.2% in children with chronic malnutrition. Vulnerability factors such as living in rural areas, being indigenous, suffering from chronic malnutrition, facing extreme poverty, and using lead-glazed pottery greatly increase the risk of lead poisoning. Among populations with all these vulnerability factors, the prevalence of lead poisoning rises to 51.5%, 15 times higher than in the least deprived populations. These data are alarming and highlight a serious environmental justice issue.

In Mexico, major sources of lead pollution include lead-glazed traditional pottery, residual lead from past use of leaded gasoline, and leaded paint. Lead-glazed pottery, commonly used in rural and artisanal communities for cooking and storing food, is a primary source of lead exposure, with studies linking frequent use to higher blood

lead levels. Additionally, lead is present in smelting and pottery making in rural areas, as well as in battery manufacturing and repair industries. Lead contamination extends to food products as well, with lead found in some canned foods and beverages, and detected in 18% of the most consumed foods in Mexico, including rice, wheat, soy, guajillo chili, processed meats, and baby food.

Our paper focuses on lead contamination from gasoline. Leaded gasoline was introduced globally in the 1920s to improve vehicle efficiency and engine performance. However, its toxic effects led to increasing concerns about its impact on air quality and public health. Mexico's efforts to remove lead from gasoline were part of a global trend, with Japan being the first country to fully ban leaded gasoline in 1986, followed by other countries gradually implementing bans throughout the late 20th century until 2021. These global actions have significantly contributed to lowering blood lead levels and improving public health worldwide. In Mexico, unleaded gasoline became available in 1990, and by 1991, its price began to decrease relative to leaded gasoline. The allowed lead limit in gasoline was progressively reduced—first from 0.5–1 ml/gal to 0.2–0.3 ml/gal in 1992, and then to 0.1–0.2 ml/gal in 1994. Finally, in 1997, Mexico fully eliminated leaded gasoline.

Figure 1 Panel A summarizes the timeline of the leaded gasoline ban in Mexico. Panel B shows monthly sales of leaded and lead-free gasoline, along with average lead concentration. Leaded gasoline sales exceeded 2 million barrels per month in the early 1990s but declined sharply after initial regulatory actions. By 1997, sales had effectively dropped to zero. In contrast, lead-free or low-lead gasoline sales started near zero in 1989, rose steadily, surpassed 1 million barrels per month by 1993, and exceeded 3 million by the early 2000s. Meanwhile, lead content in gasoline fell dramatically. In the late 1980s and early 1990s, total lead content was around 40 million milliliters per month. This dropped sharply around 1991 and again in 1994 due to changing lead content regulations, and reached near-zero levels by 1997.

2.3 Health and fertility impacts of lead

The human body responds rapidly to lead exposure. Existing studies show blood lead levels change quickly in response to acute lead intake or environmental exposure (Ara et al., 2015; World Health Organization, 2024). After exposure, lead contamination can

affect cognition, health, birth outcomes, and fertility. Historical records in the US from the late 19th to early 20th century show a 39% higher infant mortality rate in areas with leaded water pipes compared to those without (Troesken, 2008). Analyzing data from 172 cities between 1900 and 1920, Clay et al. (2014) find the average water lead content in regions with leaded pipes increased infant mortality by 19% relative to the mean mortality rate. Recent years have seen a notable decrease in fugitive lead emissions from 1980s to 2018 due to policies like the elimination of leaded gasoline and toxic release inventories. This decline has led to a consequential reduction in infant deaths (Clay et al., 2022).

In terms of fertility, the Flint water crisis, which involved lead exposure, reduced the number of births by 7.5 per 1,000 women, constituting 12% of the average fertility rate (Grossman and Slusky, 2019). The elimination of lead from gasoline has been linked to increased fertility in the US: the observed reduction in airborne lead corresponds to a 4-birth increase per 1,000 women, accounting for 6% of the mean fertility rate (Clay et al., 2021). Furthermore, lead exposure affects birth outcomes. The Flint water crisis leads to a reduction in birth weights by 32-49 grams (Abouk and Adams, 2018; Wang et al., 2022). Additionally, a change in US policy stringency regarding ambient airborne lead pollution, leading to the relocation of lead battery recycling from the US to Mexico, resulted in a 24-gram decrease in birth weight for infants born within two miles of Mexican recycling plants (Tanaka et al., 2022).

Apart from health, lead exposure also affects education and learning performance. Empirical evidence has shown the detrimental effects of lead on diminished IQ, reduced educational attainment, and labor outcomes (Ferrie et al., 2012; Reyes, 2015; Aizer and Currie, 2019; Grönqvist et al., 2020). Early-life exposure to lead has also been investigated as a potential factor contributing to changes in violent conflict trends and antisocial behaviors (Reyes, 2007, 2015; Aizer and Currie, 2019).

While most studies focus on the long-term effects of lead exposure during early life, lead pollution could also affect contemporaneous outcomes. Medical literature indicates that lead poisoning can affect protein and oxidative functions within a month (Dobrakowski et al., 2017). Similar rapid responses due to short-term lead exposure are observed in endocrine functions (Doumouchtsis et al., 2009), hormone levels (Aminian et al., 2013), blood pressure (Simões et al., 2011; Fioresi et al., 2014), and

memory loss (Fenga et al., 2016), which could naturally affect behaviors.

Anthropogenic sources serve as the primary contributors to environmental lead. Various sources have been employed as natural experiments. In the context of water, studies have exploited the presence of lead pipes (Ferrie et al., 2012; Clay et al., 2014), examined lead-related drinking water crises in places like Flint and Newark (Grossman and Slusky, 2019; Dave and Yang, 2022), and explored the effects of lead pipeline replacement (Marcus, 2023). Others have focused on lead paint in a school context: studies have identified lead impacts using classroom-level lead variation (Sauve-Syed, 2023) and comparing within-sibling performance (Gazze et al., 2021). Focusing on industrial sources, studies have examined locations in proximity to plants with toxic release inventories (Clay et al., 2022) or near battery recycling sites (Tanaka et al., 2022) to explore lead impacts. Another major source of lead pollution lies in the traffic sector. Some studies have taken advantage of regulatory changes in NASCAR gasoline content (Hollingsworth and Rudik, 2021; Hollingsworth et al., 2020) or proximity to airports (Zahran et al., 2017) as sources of variation. Other papers concentrate on the phase-out of leaded gasoline in developed nations like the US (Aizer and Currie, 2019; Clay et al., 2019, 2021) and Sweden (Grönqvist et al., 2020).

3 Data

We construct a novel dataset by combining multiple administrative and environmental data in Mexico. This allows us to track variation in lead exposure and its consequences across space, time, and generations.

3.1 Gasoline lead content

We use administrative data from PEMEX to construct a measure of lead exposure from gasoline. The data report monthly volumes of gasoline sold by type in the domestic market over the period 1990–2010. PEMEX held a monopoly over gasoline distribution in Mexico in this period. We focus on NOVA gasoline, which was the variant containing lead during this period.

Regulatory records specify the lead content mandated for NOVA gasoline at various points in time. We combine these regulatory standards with the observed quantities

of gasoline sold. Specifically, we multiply the volume of NOVA gasoline sold in the domestic market by its contemporaneous regulatory lead content. While actual compliance with these standards may have varied, our identification strategy relies on the exogenous variation in lead content induced by changes in the federal regulation.

3.2 Road infrastructure

We use digital records of the national highway network from the year 2000. The total length of the federal highway network remained relatively stable during the 1990s, expanding only modestly from 47,604 kilometers in 1990 to 48,363 kilometers by 2000 (INEGI, 2001). This temporal stability supports the use of the 2000 highway map, the oldest existing digital record, as a proxy for earlier infrastructure layout.

To measure population exposure to highways, we use data on the size of localities in 1990, where a locality corresponds to a distinct settlement or town. This allows us to approximate the share of population in a municipality exposed to highways.

3.3 Lead and air pollution

Airborne lead concentration. Nationwide data on airborne lead concentrations are not available for the period of interest. However, weekly measurements are available from 1990 onward for monitoring stations in Mexico City, collected by the Red Manual de Monitoreo Atmosférico (REDMA). Among various pollutants, REDMA reports lead concentrations in total suspended particles (PbTSP), providing a high-frequency measure of airborne lead exposure. Although geographically limited, these data allow to illustrate relationship between the phase-out of leaded gasoline and changes in ambient lead concentrations.

Aerosol optical depth. We also use aerosol optical depth (AOD) to measure overall air pollution. The data are obtained from the MERRA-2 product, available since 1980, with a resolution of 3 hours and 0.625×0.5 degrees.

3.4 Outcomes for the first generation

We construct measures of early-life outcomes for the first generation using a combination of administrative and vital statistics data from Mexican government sources,

focusing on the period 1990–1999.

Births. Data on live births are obtained from the *INEGI Consulta de Nacimientos Registrados* and vital statistics (*Estadística de Nacimientos*), which report the number of births by municipality, year, and month, which can also be split by the sex of the child. The exact birth outcomes such as birth weight or gestational age are not available in 1990–1999.

Population of women of childbearing age. To normalize birth measures, we use data from the *INEGI Consulta Población femenina de 12 años y más*, available by municipality in census years. For intercensal years, we linearly interpolate the population of women aged 12 and older at the municipal level.

Fetal deaths and conceptions. Data on fetal deaths comes from the administrative record *Estadística de Defunciones Fetales*, a micro-level dataset that records all fetal deaths. This source includes information on the municipality, date of death, and gestational age. We define conceptions as the sum of live births and fetal deaths.

Infant mortality. Data on infant deaths are drawn from the administrative microdata of the *Estadística de Defunciones Generales*, which includes all registered deaths. We define infant deaths as those occurring before the age of one. These deaths are attributed to the municipality of death, and the date of birth is inferred by subtracting age at death from the date of death.

Parental characteristics. Information on parental demographics and socioeconomic status is obtained from the *Estadística de Nacimientos* for the years 1989–2000. This administrative dataset includes individual-level data on all births, along with detailed characteristics of both parents.

3.5 Outcomes for the second generation

We construct measures of second-generation outcomes by combining multiple administrative data.

Fertility outcomes. We use administrative birth records from 2000 to 2022, which provide information on the municipality of residence and the age of each mother at the time of her child's birth. We approximate the mother's year of birth by subtracting her age from the child's year of birth and restrict the analysis to mothers born between 1990 and 2000. To construct denominators, we use natality data to count the number

of females born in each municipality in a given year 1990-2000, which provides an estimate of the size of each potential maternal cohort by location.

Birth outcomes and parental characteristics. We use microdata from the universe of birth records collected by the *Secretaría de Salud* (Registros de Nacimientos), available for the period 2008–2022. We restrict the sample to births occurring to mothers born between 1990 and 2000, the period over which we assess in-utero exposure to leaded gasoline. These data include detailed information on birth outcomes such as birth weight and gestational age, as well as parental characteristics. Crucially, this unique dataset includes the mother's municipality and date of birth, which allow us to assign maternal lead exposure in utero based on the timing and location of mother's birth.

Marginalization index. To measure structural socioeconomic disadvantage across municipalities, we use the 2010 Marginalization Index developed by the National Population Council (CONAPO). This composite index combines indicators related to education, housing conditions, and income (including the share of adults without primary education, the proportion of homes lacking piped water or electricity), and the population living in small rural localities. These components are aggregated using principal component analysis to produce a standardized marginalization score for each municipality. Municipalities are grouped into five levels of marginalization; we define those in the top two categories, high or very high, as marginalized and use this classification to examine heterogeneity in intergenerational effects. Municipalities in the marginalized group constitute 48% of all municipalities.

4 First generation effects

We begin by analyzing the direct effects of prenatal lead exposure on birth and early-life mortality outcomes for the cohort in utero between 1990 and 2000. Outcomes include (i) the number of births, (ii) fetal death rates, and (iii) infant mortality.

4.1 Empirical strategy

Following (Clay et al., 2021), our identification strategy exploits exogenous regulatory changes that led to sharp declines in the lead content of gasoline, combined with spatial heterogeneity in proximity to major roads. This approach allows us to isolate plausibly

exogenous variation in prenatal lead exposure across municipalities and over time.

Starting in the early 1990s, a series of federal regulations substantially reduced the lead content in gasoline sold nationwide. These regulatory shocks generated large, time-varying declines in aggregate lead exposure. We construct a measure of prenatal lead exposure using data on the monthly volume of leaded gasoline sold nationally. For each birth, we calculate the cumulative volume of lead sold during the month of birth and the preceding ten months, corresponding to the typical gestation period. We then normalize this measure by the average exposure level in 1991, prior to the implementation of lead phaseout policies. This normalized measure captures the percentage reduction in prenatal lead exposure relative to baseline and facilitates the interpretation of coefficients as effects of policy-induced declines in lead exposure.

While national lead content declined uniformly, exposure to airborne lead from gasoline combustion remained spatially heterogeneous due to differences in proximity to major roads. Lead emissions from gasoline are primarily transmitted via vehicular traffic, making road-adjacent populations disproportionately exposed. Some municipalities have substantial populations living far from roads, rendering them minimally affected by changes in gasoline lead content. This variation allows us to identify differential treatment intensity across space.

Due to data limitations, we do not observe individual maternal residential locations but instead use the municipality of residence, which is the finest geographic resolution available. Mexico comprises 2,469 municipalities (municipios), with an average area of 795 square kilometers (307 square miles). We classify each town within a municipality as either "near-road" or "non-near-road", depending on whether it is located within 5 kilometers of a federal highway. Using 1990 census data, we calculate the share of the municipal population residing in near-road areas.

Figure 4 illustrates this approach for the municipality of Cuatro Ciénegas. The red band depicts the 5-kilometer buffer around the federal highway, with blue dots representing towns within this buffer and green dots representing those outside. In this example, 76% of the population resides within 5 kilometers of the highway. Figure 5 presents the geographic distribution of near-road population shares across all municipalities, highlighting substantial variation, from 0% at the 25th percentile to over 90% at the 75th percentile (median = 47%).

Locations with high and low road exposure clearly differ along several important dimensions. Table 1 compares characteristics of localities close to highways to those farther away. In the Mexican context, unlike in many high-income countries, road proximity is positively associated with socioeconomic status. As a result, the lead phaseout may have disproportionately benefited more advantaged populations, potentially exacerbating pre-existing health inequalities.

Crucially, however, our identification strategy does not require that municipalities with differing levels of road exposure be similar in baseline characteristics. Instead, it relies on the assumption that, in the absence of changes in leaded gasoline content, outcomes such as fertility and early-life mortality would have evolved similarly across municipalities regardless of road proximity. That is, conditional on fixed effects, any differential trends across municipalities are assumed to arise solely from their differential exposure to the national shock, rather than from other contemporaneous, road-correlated changes.

Our empirical strategy adopts a shift-share Bartik-style identification design, exploiting plausibly exogenous variation in prenatal exposure to leaded gasoline. The *shift* is the national decline in the lead content of gasoline driven by federal regulation, while the *share* captures cross-sectional variation in municipal-level exposure to road networks. We implement this strategy by estimating the following equation:

$$y_{mt} = \beta \left(\text{Road Exposure}_m \times \text{Lead Exposure}_t \right) + \gamma_m + \delta_t + \varepsilon_{mt} \tag{1}$$

where y_{mt} includes fertility, fetal mortality, or infant mortality—in municipality m and year-month t. Road Exposure_m is the share of the municipal population living within 5 kilometers of a federal highway. Lead Exposure_t is the normalized national-level cumulative lead content in gasoline sold during the relevant prenatal window. The interaction term reflects the effective level of exposure to traffic-related airborne lead. Municipality fixed effects γ_m absorb time-invariant local characteristics, and time fixed effects δ_t capture common shocks and national trends.

The coefficient β identifies the effect of a one-unit increase in lead exposure on the outcome, scaled by the degree of road proximity. If lead exposure adversely affects birth outcomes, we expect $\beta > 0$ for fetal and infant mortality rates, and $\beta < 0$ for fertility.

4.2 First stage

The central premise of our analysis is that reductions in leaded gasoline sales resulted in lower levels of airborne lead. Ideally, we would observe this relationship directly at the national level. However, systematic monitoring of airborne lead during the relevant period was only available for Mexico City. As a result, we focus on the relationship between leaded gasoline sales and average weekly PbPST (particulate-bound lead) concentrations in Mexico City.

Figure 3 illustrates the relationship between lead in gasoline sold and PbPST in Mexico City. Visually, the decline in PbPST levels closely mirrors the reduction in leaded gasoline sales. To quantify this relationship, we regress PbPST concentrations on leaded gasoline sales. The resulting R^2 of 0.76 suggests that reductions in leaded gasoline can explain a substantial part of the declines in airborne lead in this period.

A potential concern is that contemporaneous changes in other pollutants perhaps due to concurrent regulatory changes could confound the observed relationships. This would be particularly problematic if such pollutants declined differentially by proximity to major roads. However, we are not aware of any major nationwide environmental regulations implemented during this period that would explain such trends. We also observe that the sales of new cars remained constant throughout the period 1990-2000. To further investigate, we examine the change of other pollutants in the data.

As a placebo test, Table 3 also reports the results from a regression of PST (total suspended particulates) on leaded gasoline sales. The relationship is considerably weaker, with an R^2 of just 0.066, suggesting that the reduction in pollution is specific to airborne lead rather than a broader decrease in ambient air pollution.

Since ground monitor data is limited to Mexico City, we complement our analysis with satellite-derived aerosol optical depth (AOD) which covers the whole country. Figure S1 plots the evolution of AOD over time by proximity to federal highways. We observe no differential change in AOD associated with road proximity. This is corroborated by the regression results in Table A, where we regress AOD (in levels and logs) on the interaction between nationwide lead amount sold in gasoline and an indicator for areas within 5 kilometers of a highway. The interaction terms are statistically insignificant and small, reinforcing the conclusion that other major

pollutants did not exhibit differential trends correlated with the leaded gasoline phase-out.

4.3 Effects on fetal deaths, births, and infant mortality

Table 2 presents the estimated effects of in-utero lead exposure on key first-generation outcomes: fetal deaths, live births, and infant mortality. Each column reports results from a separate regression using the interaction between the municipal share of the population living within 5 kilometers of a federal highway and the normalized measure of in-utero lead exposure. All specifications include municipality and year-month fixed effects, and standard errors are clustered at the municipality level.

Column (1) shows that lead exposure significantly affects fetal mortality. The coefficient indicates that in municipalities where 100% of the population lives near a highway, a decline in lead exposure from the pre-ban level in 1991 to zero would reduce the fetal death rate by 1.97 per 1,000 conceptions, 19% of the mean fetal death rate. Column (2) reports a statistically significant effect on birth rates. The estimated coefficient implies that a reduction in lead exposure results in an increase of 0.526 births per 1,000 women of reproductive age, equivalent to about 8% of the average birth rate. This increase likely reflects a combination of reductions in fetal loss and possible effects on conception, either through biological or behavioral channels. Column (3) documents a large and significant decline in infant mortality. A full exposure reduction corresponds to 3.33 fewer infant deaths per 1,000 live births—a 22% decline relative to the baseline mortality rate. These results suggest that prenatal lead exposure not only affects fetal survival but also has lasting consequences for neonatal health.

We further explore whether the infant mortality effects differ by child gender by interacting the main exposure variable with a gender dummy. In Table 3, the interaction of male with lead exposure and road proximity is large and statistically significant, suggesting that male infants are more adversely affected by lead. Specifically, the estimated change in infant mortality for female infants is 2.57 per 1,000, while the additional effect for males is 1.41 per 1,000, yielding a total effect of 3.98 for male infants. This pattern is consistent with evidence from medical research indicating that male fetuses are biologically more vulnerable to in-utero stressors such as environmental toxins (e.g., Trivers and Willard, 1973; Catalano et al., 2006; DiPietro and Voegtline,

2017).

While these effects are large in magnitude, they are in line with estimates from the existing literature. For fertility outcomes, the Flint water crisis led to a 15–18% decline in births (Grossman and Slusky, 2019), while the phase-out of leaded gasoline in the U.S. resulted in a 6% increase in fertility (Clay et al., 2021). Regarding infant mortality, historical exposure to lead through water infrastructure raised mortality by 19–39% in early-20th-century U.S. cities (Troesken, 2008; Clay et al., 2014), while industrial lead emissions have been linked to a 3% increase (Clay et al., 2022). Back-of-the-envelope calculations suggest the magnitude of the Mexico policy's public health benefits. In 1991 alone, a full elimination of lead exposure would have resulted in 3,381 fewer fetal deaths (relative to 29,344 observed), 139,453 additional live births (out of 2,735,312), and 6,005 fewer infant deaths (compared to 51,311 recorded).

4.4 Selection into childbearing

An important question is whether the estimated effects on early-life outcomes might be driven, at least in part, by changes in the composition of parents, i.e., selection into childbearing rather than direct biological effects of prenatal lead exposure. To examine this possibility, we investigate whether parental characteristics changed in response to lead exposure during the phase-out period. Specifically, we test for changes in observable maternal and paternal characteristics, including the mother's age, marital status, and education, whether the father is reported, and the father's age and education.

The results in Tables 4 and 5 show no evidence of systematic changes in these characteristics in response to changes in lead exposure. Only the coefficient on mother's age is statistically significant, yet it is small in magnitude. This suggests that selection into parenthood is unlikely to explain the large effects we observe on fetal deaths, births, and infant mortality.

Taken together, our results indicate that the phase-out of leaded gasoline in Mexico led to large and immediate improvements in early-life health among the cohorts in utero during the policy transition. We next examine whether and how these effects persisted into the next generation, i.e., the children of those exposed in utero to the leaded gasoline phase-out.

5 Second generation effects

5.1 Empirical strategy

We now move to the second-generation effects. Before that, we first examine how in-utero exposure to lead affects female children's fertility decisions when they grow up. We use a similar identification strategy as in Section 4, and regress fertility rates on in-utero exposure to lead at birth. The estimation equation is as follows:

$$y_{mct} = \beta \left(\text{Road Exposure}_{m} \times \text{Lead Exposure}_{ct} \right) + \gamma_m + \delta_c + \varepsilon_{mct} \tag{2}$$

where y_{mct} is the number of children each potential mother has in municipality m, born in cohort c, in year t. Since the lead ban occurred in the 1990s, the maximum age for potential mothers is 24, so we focus on females aged 15–24 when coding fertility metrics. On the right-hand side, we use a similar shift-share design to code females' in-utero exposure. Road Exposure_{m} is the share of the municipal population living within 5 kilometers of a federal highway, fixed over time. $\text{Lead Exposure}_{ct}$ is the normalized national-level cumulative lead content in gasoline sold during the relevant prenatal window for cohort c. We control for mother birth year fixed effects in δ_c to capture cohort-specific nationwide differences, and municipality fixed effects γ_m absorb time-invariant local characteristics.

β is the coefficient of interest, measuring how differential in-utero exposure to lead affects fertility decisions. Given the absence of selection into motherhood by parents as shown in Tables 4 and 5, the only difference among these women is their varying in-utero exposure to lead.

For mothers who actually gave birth and their children, we examine maternal characteristics and second-generation outcomes by estimating the following equation:

$$y_{imct} = \beta \left(\text{Road Exposure}_{m} \times \text{Lead Exposure}_{ct} \right) + \gamma_m + \delta_c + X_{imct} + \varepsilon_{imct} \tag{3}$$

The analysis is at the individual mother or individual birth level. Regarding mother characteristics, outcomes include her age, migration decision dummy, years of education, and number of prenatal care visits. For the child, outcomes include the

raw birth weight, incidence of low birth weight, gestation length, and a preterm birth dummy for birth i, born in year t, whose mother was born in municipality m and in cohort c, where cohort is defined by year and month of birth. On the right-hand side, we code a similar shift-share lead gasoline exposure during i's mother's in-utero period by interacting national lead content with road intensity in mother's municipality of birth. We control for municipality and mother cohort fixed effects, as well as the mother's age.

Focusing on children, the coefficient of interest is β, capturing how in-utero exposure to lead affects the next generation's outcomes. This effect could result from the biological transmission of lead from mother to child or from changes in maternal characteristics and fertility decisions induced by the mother's own in-utero lead exposure. The former is likely to harm second-generation birth outcomes, while the latter has an ambiguous effect sign, which we test empirically.

5.2 Fertility outcomes and maternal characteristics

Table 6 presents the effects of in-utero exposure to lead on fertility decisions when these female children reach adulthood. In Column (1), the outcome variable is the number of children born to potential mothers aged 15 or younger. The estimate on the interaction term is positive but not statistically significant, suggesting minimal differences in fertility decisions between mothers with high and low lead exposure. In Column (2), we observe a similarly insignificant estimate on Road Exposure$_m$ $\times$ Lead Exposure$_{ct}$, with a comparable magnitude when scaled by the dependent variable mean. In Columns (3) and (4), we observe an increase in the number of births driven by mothers in late teenage years and in early 20s. Decreasing exposure from pre-policy levels to 0 in areas exposed to roads would decrease teenage fertility by 22.74 per 1000 women and fertility by early 20s by 85.2 per 1000 women. These changes correspond to 11% and 16.7% of the mean respectively. Taken together across all age groups, we find no evidence that lead exposure harms the fertility rate of the second generation. In contrast, we see that women exposed to lead tend to have higher fertility at early age.

Existing literature documents that lead contamination negatively affects fertility outcomes (e.g., Grossman and Slusky, 2019; Clay et al., 2021; Du and Taylor, 2024; Du and Zhang, 2024). This is further supported by medical evidence showing that

lead pollution disrupts steroidogenic function, leading to fetal abnormalities and embryotoxicity (Aquino et al., 2012). Additionally, oxidative stress induced by heavy metals can alter hormone function and impair embryo quality, contributing to female infertility (Rzymski et al., 2015). However, all of this empirical and biological evidence pertains to the first generation. Our estimates for the second generation indicate that in-utero exposure to lead increases fertility at young age. Nonetheless, we miss data on later in life fertility. Hence this effect may reflect change in timing of childbearing rather than change in lifetime fertility.

Among females who actually gave birth, Table 7 presents the effects of in-utero lead exposure on maternal characteristics. In Column (1), we find that exposure to lead significantly decreases maternal age at childbirth. This is consistent with results on fertility, which shows that mother's exposed to lead have higher number of children at young ages. The estimated coefficient suggests that, in municipalities where 100% of the population lives within 5 kilometers of a federal highway, a reduction in lead exposure from the pre-ban level in 1991 to zero would increase the future maternal age of exposed children by 0.15 years. This is a sizable effect, especially considering that the sample includes only potential mothers aged below 24, with an average maternal age of 21.4.

In Column (2), we observe that mothers exposed to lead during the in-utero period significantly reduce their likelihood of migrating. Since these mothers are more likely to live near highways as a result of our shift-share design, their reduced likelihood of migration means they are more likely to remain exposed to highway-related pollution over time. The estimated coefficient suggests that, in municipalities where 100% of the population lives within 5 kilometers of a federal highway, a reduction in lead exposure from the pre-ban level in 1991 to zero would increase the probability of migration by 0.041, or 9.3% relative to the mean.

Our results suggest that early-life lead exposure can have long-term impacts on the timing of key life decisions, such as childbearing and migration to cleaner environments. Giving birth at a younger age has been shown to lead to a range of socioeconomic and health disadvantages, including lower educational attainment, reduced labor market participation, and a higher risk of adverse birth outcomes (Geronimus and Korenman, 1992; Ribar, 1999; Hotz et al., 2005; Fletcher and Wolfe,

2009; Narita and Diaz, 2016; Chakraborty and Villa, 2024). Although these downstream consequences are not directly captured in our data, the elevated risks associated with younger maternal age may, in turn, have additional effects on the outcomes of the next generation.

In Columns (3) and (4), we find that mothers exposed to higher levels of lead in utero are more likely to have higher years of education and a larger number of prenatal care visits. The estimated coefficients suggest that, in municipalities where 100% of the population lives within 5 kilometers of a federal highway, a reduction in lead exposure from the pre-ban level in 1991 to zero would decrease the length of education by 0.4 years, or 3.4% of the average education length, and decrease prenatal visits by 0.08 times, or 1.2% of the mean. These magnitudes are relatively small compared to the reduction in maternal age and the mitigation decisions.

These results imply that exposed parents may invest more in their children to offset the negative effects of lead exposure. Moreover, there could be a positive selection into survival. Another potential explanation is the quality-quantity tradeoff. Exposed parents may observe higher fetal mortality rates and greater risks to child survival, prompting them to allocate remaining family resources more effectively to support the surviving children. This could result in longer years of education and more prenatal care for subsequent pregnancies. These mitigation efforts following initial exposure to lead are likely to improve the next generation's health outcomes.

5.3 Effects on birth outcomes

Table 8 reports the estimated effects of female in-utero lead exposure on her children's birth outcomes. On average, the estimates are very small and statistically insignificant across all columns, suggesting that maternal exposure to lead in utero has little to no effect on her child's birth weight, incidence of low birth weight, gestational age, or the likelihood of preterm birth.

However, these pooled effects mask important heterogeneity. Table 9 presents results from regressions that interact lead exposure with an indicator for whether the child was born in a marginalized municipality—defined using the CONAPO 2010 index, which classifies nearly half of all municipalities as disadvantaged.

First, we find that in non-marginalized municipalities, second-generation birth

outcomes are unaffected by maternal lead exposure. The estimates are not only statistically insignificant but also very small: we can rule out effects on birth weight larger than 3.7 grams in either direction and changes in gestation length greater than 0.027 weeks (0.19 days). This suggests that in better-off settings, the intergenerational effects of lead exposure are likely close to zero.

In contrast, in marginalized municipalities, we find significant and sizable adverse effects. Full maternal exposure to lead at pre-policy levels is associated with a 10.43-gram reduction in offspring birth weight, a 0.28 percentage point increase in the probability of low birth weight, and a 0.46 percentage point increase in the probability of preterm birth. To contextualize this magnitude, we compare it to the findings of East et al. (2023) who study the effects of maternal in-utero exposure to Medicaid expansions. They estimate a reduced-form effect of approximately 4.7 grams on birth weight. Applying our effect size to the median municipality in terms of road proximity (with a road exposure share of 0.47), we obtain an implied birth weight effect of $0.47 \times 10.43 \simeq 4.9$ grams. This comparison suggests that deleading policies may yield second-generation public health benefits comparable in magnitude to those achieved through major social policy interventions such as Medicaid expansion.

The heterogeneity across municipalities likely reflects differential capacity to mitigate early-life shocks. The ability to offset environmental harm — through maternal education, access to prenatal care, or broader socioeconomic conditions — is more limited in disadvantaged areas. Our findings suggest that the intergenerational transmission of pollution is conditional and disproportionately persistent in contexts of structural disadvantage.

6 Discussion and conclusion

This paper examines the direct and intergenerational consequences of a large environmental shock, the phase-out of leaded gasoline, in a developing country context. Using administrative and environmental data from Mexico, we document substantial short-run improvements in early-life health following the reduction in airborne lead exposure. The lead phase-out significantly reduced fetal deaths, increased birth rates, and lowered infant mortality, with stronger effects observed among male infants. These

estimates are large, robust, and in line with findings from high-income countries.

Despite these pronounced first-generation effects, the transmission of harm to the second generation appears to be mediated by socioeconomic context. We find no adverse effects on birth outcomes among children of exposed mothers in well-off municipalities. In contrast, in disadvantaged municipalities, maternal in-utero lead exposure leads to significantly lower birth weight and shorter gestation in the next generation. These results suggest that individuals living in higher socioeconomic status or more favorable structural conditions are better equipped to buffer the long-term effects of early-life exposure.

Several limitations of our study are worth noting. First, individual-level birth outcomes are not available for the first generation, preventing direct biological comparisons across cohorts. Second, pollution data are geographically limited, with detailed airborne lead concentration measurements available only for Mexico City. Third, we do not observe individual biomarkers of exposure and rely on proxies based on gasoline content and road proximity. Finally, our analysis focuses on early-life outcomes. Longer-term second-generation effects on human capital or labor market outcomes remain an open question.

Despite these limitations, our results offer several policy-relevant insights. We demonstrate the substantial public health gains from the leaded gasoline ban and underscore the continued urgency of addressing other sources of lead pollution. More broadly, our findings suggest that early-life harm from pollution, while severe, is not necessarily irreversible. However, the unequal transmission of harm across socioeconomic contexts highlights the need for targeted interventions. In particular, policies that enhance access to healthcare, education, and reproductive support in disadvantaged areas may be critical to breaking the cycle of intergenerational harm. An important direction for future research is to identify which specific investments such as maternal education, healthcare access, nutritional support, or social protection, are most effective at breaking the intergenerational transmission of environmental harm.

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Figures and tables

Figure 1: Timeline of Mexico leaded gasoline ban

Panel A

TABLE Timeline

1920	Leaded gasoline introduced globally
1990	Unleaded gasoline becomes available in Mexico.
1991	Decreasing relative price of unleaded gasoline
1992	Decrease of allowed lead limit from 0.5-1 to 0.2-0.3 ml/gal.
1994	Further decrease from 0.2-0.3 g/L to 0.1-0.2 ml/gal.
1997	Complete elimination of lead in gasoline across Mexico

Panel B

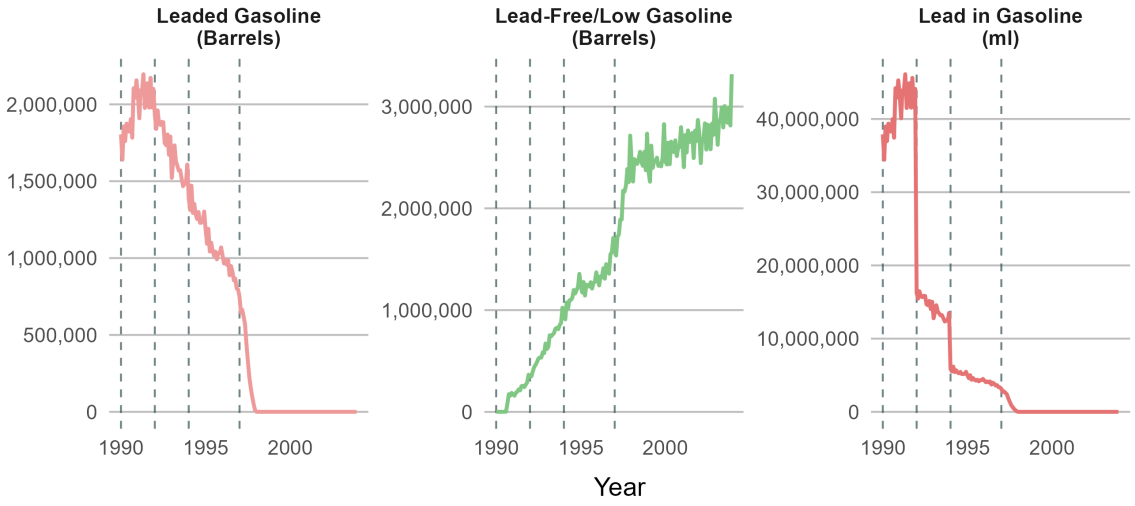

Figure 2: Mexican Highways

Notes: The map shows federal highways network in Mexico in 2000. The geographic layout corresponds to administrative boundaries of municipalities.

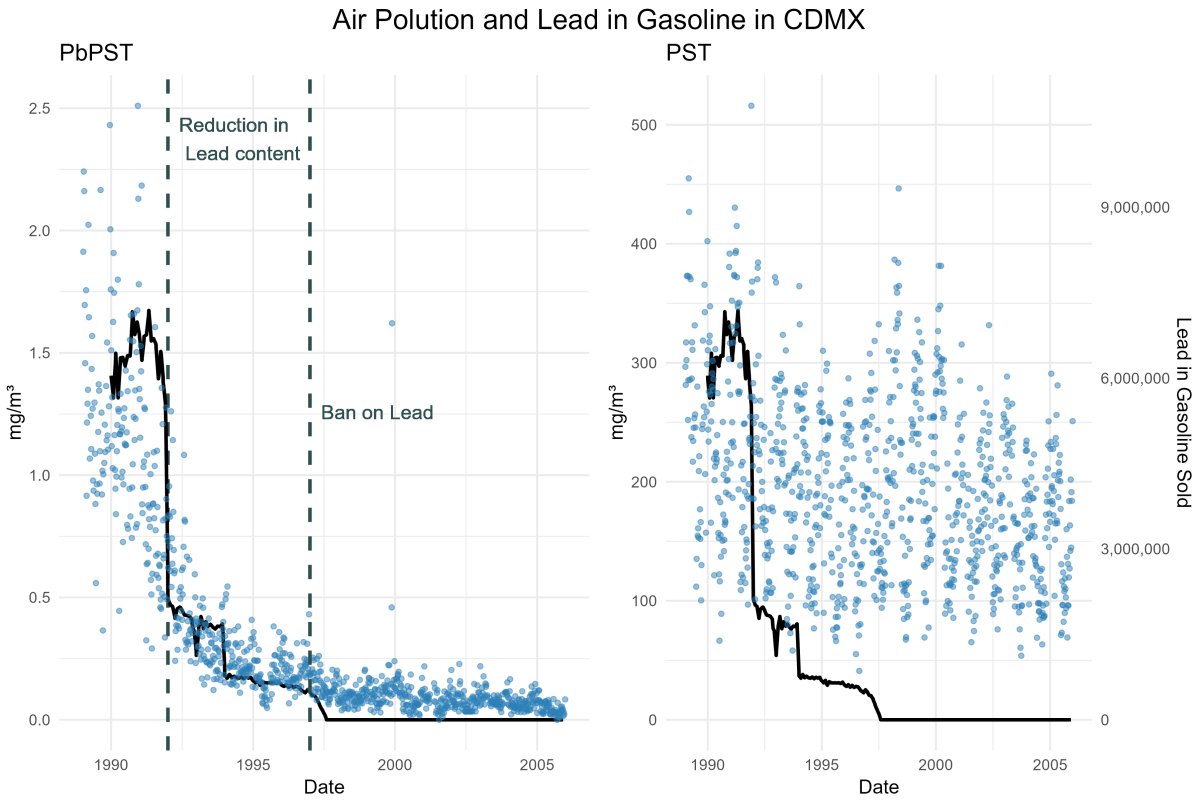

Figure 3: Mexico City Pollution

Notes: The left panel shows the concentration of lead content in total suspended particles (PbPST), while the right panel shows overall total suspended particles (PST) in CDMX. Both panels include overlaid data on lead in gasoline (right y-axis), with vertical dashed lines marking key policy changes: the reduction and subsequent ban of lead in gasoline.

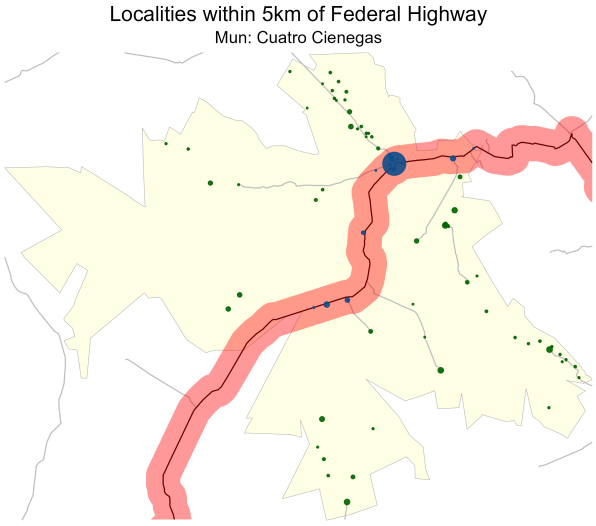

Figure 4: Population Living Close to Highways

Notes: The figure illustrates localities within 5 kilometers of a federal highway in the municipality of Cuatro Ciénegas. The shaded red buffer indicates areas classified as close to the highway. The green dots represent localities further than 5km from the higway, while blue dots represent those within 5km of the highway. Size of the dot corresponds to the size of the town.

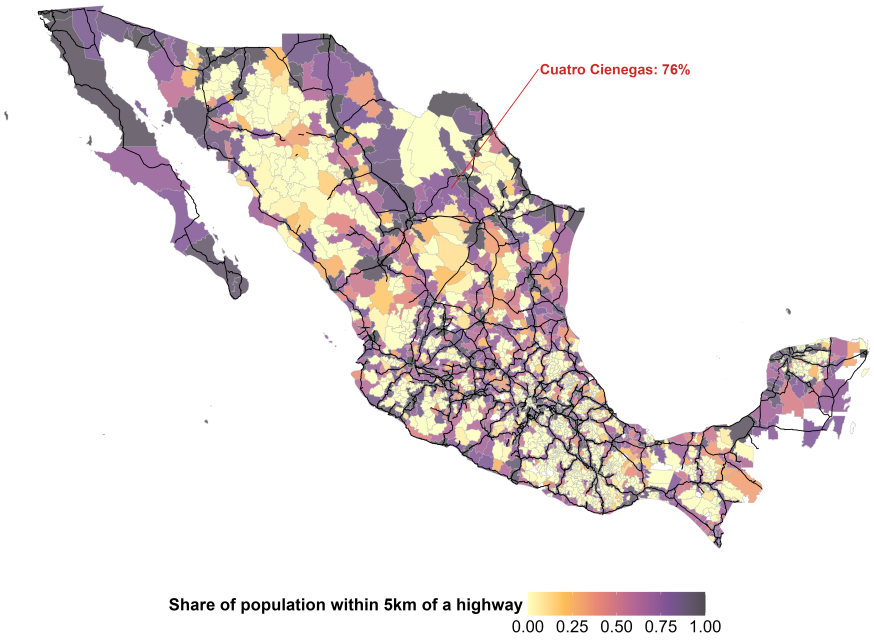

Figure 5: Municipal Exposure to Highways

Notes: This map shows the share of each municipality's population living within 5 kilometers of a federal highway. Darker areas indicate higher levels of exposure, with Cuatro Ciénegas highlighted as an example.

	Further than 5km		Within 5km			
	Mean	Std. Dev.	Mean	Std. Dev.	Difference	Std. Er.
Population	447.3	11301.9	1870.7	25400.2	1423.4***	148.5
Electricity (%)	51.2	44.0	71.9	37.6	20.7***	0.3
Piped Water (%)	16.6	28.9	30.7	35.4	14.2***	0.2
Analphabetism (%)	28.0	20.1	21.4	15.8	-6.6***	0.1
Indigenous (%)	18.3	35.4	9.7	25.5	-8.6***	0.2
N	63601		32171			

Table 1: Descriptive Statistics for Localities Close and Far from Highway

Notes: This table presents summary statistics for localities located within and beyond 5 kilometers of a federal highway.

Dependent Variables:	Fet. deaths per 1000 conceptions	Births per 1000 women	Inf. deaths per 1000 births
Model:	(1)	(2)	(3)
Variables			
Share close to road (5km) × Preg. Lead Exp.	1.797***	-0.5626**	3.327***
	(0.3758)	(0.2206)	(0.8163)
Fixed-effects			
Municipality	Yes	Yes	Yes
Year-Month	Yes	Yes	Yes
Fit statistics			
R^2	0.18818	0.74288	0.27111
Observations	256,457	437,346	370,699
Dependent variable mean	10.538	6.7953	15.030

Clustered (Municipality) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 2: Effects on Fetal Deaths and Births

Notes: Each column reports estimates from a separate regression of fetal deaths, births, or infant deaths on the interaction between the municipal share of the population living within 5 kilometers of a federal highway and a measure of pregnancy lead exposure. Pregnancy lead exposure is normalized to reflect the level relative to the average exposure in 1991, prior to the policy change. The analysis is at the municipality-month level, 1990-2000. All regressions include year-month and municipality fixed effects. Observations are weighted by the number of conceptions, women, or births, respectively.

Dependent Variable:	Infant deaths per 1000 births
Model:	(1)
Variables	
Male	3.023***
	(0.1578)
Share close to road (5km) × Pregnancy Lead Exposure	2.573***
	(0.7914)
Share close to road (5km) × Male	0.1908
	(0.2212)
Pregnancy Lead Exposure × Male	0.5201*
	(0.2940)
Share close to road (5km) × Pregnancy Lead Exposure × Male	1.410***
	(0.4355)
Fixed-effects	
Municipality	Yes
Year-Month	Yes
Fit statistics	
R^2	0.19810
Observations	502,199
Dependent variable mean	15.517

Clustered (Municipality) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 3: Infant Mortality by Gender

Notes: This table reports estimates from a regression of infant mortality (per 1,000 births) on interactions between the municipal share of the population living within 5 kilometers of a federal highway, a measure of pregnancy lead exposure, and child sex. Lead exposure is normalized to represent the level relative to the average exposure in 1991, prior to the policy change. The analysis is at the municipality-month level, 1990-2000. The specification includes year-month and municipality fixed effects. Observations are weighted by the number of births.

Dependent Variables:	Age of the mother at birth	Mother married	Years of mother's education
Model:	(1)	(2)	(3)
Variables			
Share close to road (5km) × Lead Exposure	-0.1082	-0.0020	0.1010**
	(0.0706)	(0.0043)	(0.0426)
Fixed-effects			
Municipality	Yes	Yes	Yes
Year-Month	Yes	Yes	Yes
Fit statistics			
R^2	0.01120	0.12640	0.24723
Observations	22,971,301	22,459,997	22,517,808
Dependent variable mean	25.684	0.62874	7.2585

Clustered (Municipality) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 4: Maternal Characteristics

Notes: This table reports estimates from regressions of demographic and educational outcomes of mothers giving birth between 1990 and 2000 on the interaction between the municipal share of the population living within 5 kilometers of a federal highway and the pregnancy lead exposure. Lead exposure is normalized to reflect the level relative to the average exposure in 1991, prior to the policy change. The analysis is at the individual level, 1990-2000. Each column represents a separate regression. All specifications include municipality and year-month fixed effects.

Dependent Variables:	Unknown father	Age of the father at birth	Years of father's education
Model:	(1)	(2)	(3)
Variables			
Share close to road (5km) × Lead Exposure	0.0012	-0.0886	0.0713
	(0.0011)	(0.0811)	(0.0540)
Fixed-effects			
Municipality	Yes	Yes	Yes
Year-Month	Yes	Yes	Yes
Fit statistics			
R^2	0.00682	0.01637	0.21913
Observations	23,105,610	21,394,790	20,982,407
Dependent variable mean	0.07404	29.110	7.8306

Clustered (Municipality) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 5: Paternal Characteristics

Notes: This table reports estimates from regressions of demographic and educational outcomes of fathers corresponding to births between 1990 and 2000 on the interaction between the municipal share of the population living within 5 kilometers of a federal highway and the pregnancy lead exposure. Lead exposure is normalized to reflect the level relative to the average exposure in 1991, prior to the policy change. The analysis is at the individual level, 1990-2000. Each column represents a separate regression. All specifications include municipality and year-month fixed effects.

Dependent Variables:	15 or less	17 or less	19 or less	24 or less
Model:	(1)	(2)	(3)	(4)
Variables				
Share close to road × Lead Exposure	1.784	7.171	22.74***	85.20***
	(1.735)	(4.941)	(8.403)	(19.87)
Fixed-effects				
Municipality	Yes	Yes	Yes	Yes
Mother-Birth-Year	Yes	Yes	Yes	Yes
Fit statistics				
R^2	0.59764	0.84502	0.91466	0.93361
Observations	26,415	26,415	26,415	26,415
Dependent variable mean	16.692	82.768	203.67	510.45

Clustered (Municipality) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 6: Effects on Fertility Outcomes of the Second Generation

Notes: This table reports estimates from regressions of fertility outcomes in the second generation on the interaction between the municipal share of the population living within 5 kilometers of a federal highway and lead exposure in utero. Exposure is defined based on the mother's municipality of birth and corresponds to the level of lead exposure in the year prior to her birth. The analysis is at the municipality-year-cohort level, 2000-2023. All specifications include mother's municipality and mother's birth year fixed effects. Observations are weighted by the number of women in each cell.

Dependent Variables: Model:	Mother's Age (1)	Migrant (2)	Years of Edu. (3)	M. Visits (4)
Variables				
Share close to road × Lead Exposure in Utero	-0.1453***	-0.0407***	0.4049***	0.0824***
	(0.0338)	(0.0052)	(0.0720)	(0.0308)
Fixed-effects				
Mother's Mun.	Yes	Yes	Yes	Yes
Mom's Birth Month-Year	Yes	Yes	Yes	Yes
Mother's Age		Yes	Yes	Yes
Fit statistics				
R^2	0.20684	0.14095	0.16654	0.05815
Observations	11,434,735	11,434,735	11,298,744	11,205,529
Dependent variable mean	21.459	0.43893	12.029	7.0234

Clustered (Mother's Mun.) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 7: Maternal Characteristics of the Second Generation

Notes: This table reports estimates from regressions of maternal characteristics among women who were exposed to lead in utero, on the interaction between the municipal share of the population living within 5 kilometers of a federal highway and mother's lead exposure in utero. Municipality and exposure are defined based on the mother's place and time of birth, with lead exposure normalized to reflect the level relative to the average exposure in 1991, prior to the policy change. Analysis is at the individual level, 2008-2023. All specifications include municipality-of-birth, mother's birth month-year, and current age fixed effects.

Dependent Variables: Model:	Birth Weight (g) (1)	Low Birth Weight (2)	Gestation (weeks) (3)	Preterm (4)
Variables				
Share close to road × Lead Exposure in Utero	-2.042	-0.0005	-0.0146	-0.0004
	(1.701)	(0.0006)	(0.0096)	(0.0007)
Fixed-effects				
Mother's Mun.	Yes	Yes	Yes	Yes
Mother's Age	Yes	Yes	Yes	Yes
Mom's Birth Month-Year	Yes	Yes	Yes	Yes
Fit statistics				
R^2	0.02573	0.00256	0.01260	0.00315
Observations	11,434,735	11,434,735	11,434,735	11,434,735
Dependent variable mean	3,121.7	0.06222	38.782	0.06157

Clustered (Mother's Mun.) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 8: Birth Outcomes in the Second Generation

Notes: This table reports estimates from regressions of birth outcomes in the second generation on the interaction between the municipal share of the population living within 5 kilometers of a federal highway and mother's lead exposure in utero. Municipality and exposure are defined based on the mother's place and time of birth, with lead exposure normalized to reflect the level relative to the average exposure in 1991, prior to the policy change. Analysis is at the individual level, 2008-2023. All specifications include municipality-of-birth, mother's birth month-year, and current age fixed effects.

Dependent Variables:	Birth Weight (g)	Low Birth Weight	Gestation (weeks)	Preterm
Model:	(1)	(2)	(3)	(4)
Variables				
Share close to road × Lead Exposure in Utero	-0.0798	-0.0003	-0.0063	-0.0004
	(1.857)	(0.0007)	(0.0107)	(0.0008)
Share close to road × Lead Exposure in Utero × Marginalized	-10.42***	0.0028*	-0.0236	0.0046***
	(3.664)	(0.0017)	(0.0150)	(0.0017)
Fixed-effects				
Mother's Mun.	Yes	Yes	Yes	Yes
Mother's Age	Yes	Yes	Yes	Yes
Mom's Birth Month-Year	Yes	Yes	Yes	Yes
Fit statistics				
R^2	0.02575	0.00260	0.01284	0.00323
Observations	11,401,137	11,401,137	11,401,137	11,401,137
Dependent variable mean	3,121.8	0.06218	38.782	0.06154

Clustered (Mother's Mun.) standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table 9: Birth Outcomes in the Second Generation: Heterogeneity

Notes: This table reports estimates from regressions of birth outcomes in the second generation on the interaction between the municipal share of the population living within 5 kilometers of a federal highway, mother's lead exposure in utero and level of marginalization of the municipality of child's birth. Municipality and exposure are defined based on the mother's place and time of birth, with lead exposure normalized to reflect the level relative to the average exposure in 1991, prior to the policy change. Marginalization index is constructed by the Mexican office of Conapo and we use the 2010 values. Analysis is at the individual level, 2008-2023. All specifications include municipality-of-birth, mother's birth month-year, and current age fixed effects.

Online Appendix

A Additional tables

Dependent Variable:	Pollution Level	
key	PbPST	PST
Model:	(1)	(2)
Variables		
Constant	-0.1210***	-0.0459
	(0.0133)	(0.0307)
Lead in Gasoline	0.7325***	0.2500***
	(0.0280)	(0.0351)
Fit statistics		
R^2	0.76246	0.06609
Observations	941	941

Heteroskedasticity-robust standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table S1: Airborne Lead and Lead in Gasoline Sold in Mexico City

Notes: This table presents estimates from regressions of weekly average PbPST and PST levels in Mexico City on lead in gasoline sold in Mexico City. Each variable is standarized and expressed in standard deviations. Years 1990 to 2005 are considered.

Dependent Variables:	AOD	log(AOD)
Model:	(1)	(2)
Variables		
Constant	0.1359***	-2.236***
	(0.0054)	(0.0474)
Lead in Gasoline	0.0003***	
	(6.48×10^{-5})	
Within 5km from Highway	0.0145*	0.1431**
	(0.0081)	(0.0657)
Lead in Gasoline × Within 5km from Highway	9.4×10^{-6}	
	(9.31×10^{-5})	
log(Lead in Gasoline+1)		0.1185***
		(0.0138)
log(Lead in Gasoline+1) × Within 5km from Highway		-0.0163
		(0.0193)
Fit statistics		
R^2	0.27267	0.35109
Observations	312	312
Dependent variable mean	0.17325	-1.8592

Heteroskedasticity-robust standard-errors in parentheses
*Signif. Codes: ***: 0.01, **: 0.05, *: 0.1*

Table S2: Aerosol Optical Depth and Leaded Gasoline Exposure

Notes: This table presents estimates from regressions of aerosol optical depth (AOD) and its logarithm on leaded gasoline exposure and proximity to highways. Observations are on month by location level, where location takes two value: close to highway (within 5km) or far from road. Column (1) reports results using AOD levels, while Column (2) uses log-transformed AOD as the outcome. Lead in Gasoline is expressed in 100 of liters of lead sold in gasoline in a given month. All specifications include an indicator for being located within 5 kilometers of a federal highway and its interaction with lead in gasoline.

B Additional figures

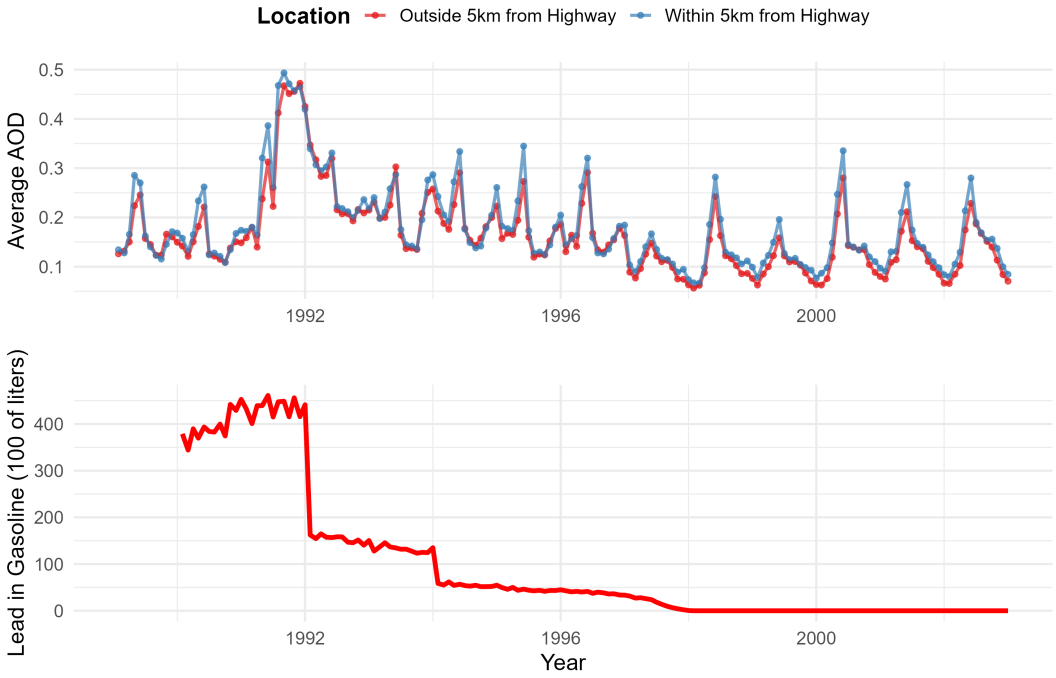

Figure S1: Aerosol Optical Depth and Leaded Gasoline Sales