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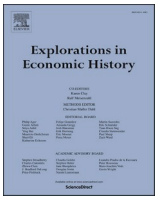

Early-life lead exposure and male longevity: Evidence from historical municipal water systems

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ABSTRACT

Several research strands document the life-cycle impacts of lead exposure during early life. Yet little is known about the long-run effects of lead exposure during early life on old-age mortality outcomes. In this study, we employ Social Security Administration death records linked to the full-count 1940 census and document that birth-city lead status negatively affects later life old age longevity. These impacts are larger for cities with acidic water and older pipeline systems that allow higher lead levels to leach into drinking water. Further, we show that the impacts are almost exclusively concentrated on the lead status of the birth-city and not the city of residence later in life. An instrumental variable strategy suggests reductions in longevity associated with birth-city lead status of about 9.6 months. We also find education, socioeconomic standing, and income reductions during early adulthood as candidate mechanisms. Finally, we use WWII enlistment data and observe reductions in measures of cognitive ability among lead-exposed individuals.

1. Introduction

Following the late 19th century industrial revolution, there was a sharp rise in products that employed lead as their constituents. For instance, farm management specialists started using lead arsenate at unprecedented levels during the first decades of the 20th century. During the same period, many cities installed city-wide pipe water systems, many of which employed lead as their primary product or a combination of lead and other materials such as iron. Although the negative health impacts of lead were known to public health specialists and critics regularly argued against using lead specifically in the water system, lack of universal consensus and low levels of regulation and monitoring resulted in limited interventions (Hamilton, 1914; Oliver, 1914; Weston, 1900).

There is now a relatively large and established literature that points to the short-term and long-term impacts of lead exposure (Aizer et al., 2018; Aizer and Currie, 2019; Billings and Schnepel, 2018; Dave and Yang, 2022; Feigenbaum and Muller, 2016; Wodtke et al.,

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2022). Based on the World Health Organization's recent reports, about 30 % of the global burden of idiopathic intellectual disability among children and about 4.6 % of the burden of cardiovascular disease is due to cumulative lead exposure (World Health Organization, 2021). Moreover, there are about 1 million deaths in the world annually due to lead exposure, roughly half of the total deaths due to known hazardous chemicals (World Health Organization, 2022). Studies suggest that prenatal exposure to lead is associated with higher risks of pregnancy complications (Bellinger, 2005), increases in fetal death (Roy and Edwards, 2021), higher infant mortality rates (Troesken, 2008), and adverse birth outcomes (Bui et al., 2022; Dave and Yang, 2022). In the long run, prenatal and childhood exposure to lead is associated with behavioral problems (Reyes, 2015), cognitive development (Coscia et al., 2003; Dietrich et al., 1991; Schnaas et al., 2006), I.Q. (Nevin, 2000), elevated blood pressure (Farzan et al., 2018), kidney functioning (Skröder et al., 2016), crime (Feigenbaum and Muller, 2016; Reyes, 2007), educational outcomes (Miranda et al., 2007; Sorensen et al., 2019), and old-age Alzheimer's disease (Eid et al., 2016).

This paper examines whether exposure to lead in drinking water during early life and childhood reduces old-age longevity. Specifically, we leverage historical variation in the materials used in municipal water infrastructure across U.S. cities, focusing on whether an individual's birth city utilized lead pipes in its water delivery system. We employ Social Security Administration death records for male individuals linked to the full count 1940 census to examine the longevity differences between individuals born in cities with some lead in their pipeline water system and cities with no lead. Several factors make lead cities inherently different than non-lead cities. During the 19th century, lead was more expensive than its closest viable alternative, iron. On the other hand, lead has several advantages, including its higher longevity, durability, ease of use and installation, and malleability (Rabin, 2008). Therefore, cities with tighter budgets might choose more easily sourced alternatives. Progressive leadership of wealthier cities with better resources to invest in long-term infrastructures were more likely to use lead. However, in some cases, early warnings from public health experts about lead poisoning may have deterred its use, particularly in cities with more progressive or better-informed leadership (Thresh, 1901). Local authorities might consider these concerns when choosing materials for water pipelines. These selection mechanisms to employ lead in water systems may also act in other unobserved ways to impact population health. Therefore, any comparison between lead and non-lead cities is confounded by such selection mechanisms. Importantly, local authorities' decisions to employ lead in water systems were largely based on the costs and availability of lead refineries and distilleries. This fact is binding, especially during the 19th century when transportation costs constituted a large portion of the final costs of products (Jacks et al., 2010). Therefore, access to lead manufacturing facilities creates variations in transportation costs, hence the likelihood of a city implementing lead pipelines. We follow the method developed by Feigenbaum and Muller (2016) and exploit the distance between the city and several major lead refineries as an instrument and examine the association between birth-city lead status and later life longevity.

We employ Social Security Administration death records for male individuals over the years 1975–2005 to the full-count 1940 census. To infer individuals' city of birth and early-life environment, we additionally link these records to earlier full-count censuses from 1900 to 1930, allowing us to observe residential histories and assign lead exposure based on birth-city water infrastructure. While the ordinary least squares (OLS) estimates indicate a modest reduction in longevity of approximately 0.6 months associated with early-life exposure to lead, our instrumental variables (IV) strategy yields substantially larger effects—suggesting a decrease in lifespan of about 9.7 months. These effects are primarily concentrated among white individuals. Although we find negative and sizable coefficients for Black individuals, the estimates are imprecise, limiting strong conclusions. Additionally, we do not observe significant differences in the estimated effects based on maternal literacy status, suggesting that the impact of lead exposure may operate largely through biological mechanisms rather than being strongly mediated by early socioeconomic environment.

We further examine heterogeneity in the effects of early-life lead exposure by water chemistry, where more acidic water is expected to exacerbate lead leaching and thus strengthen negative health effects (Clay et al., 2014; Feigenbaum and Muller, 2016; Troesken, 2008). Although the estimated impact is larger in cities with acidic water—where corrosion accelerates lead leaching—the high standard errors and weak instrument limit the interpretability of these results. In contrast, we find more robust evidence when examining the age of municipal water pipelines. Cities with older pipeline systems experience significantly stronger negative effects on longevity, consistent with increased lead release over time due to the degradation of protective pipe coatings (Edwards and Dudi, 2004; EPA, 2024; Renner, 2009).

We also find that the negative effects of early-life lead exposure are significantly larger in states with above-median automobile density in 1940. In these states, birth-city lead status is associated with a reduction in longevity of approximately 11.1 months, compared to small and statistically insignificant effects in states with lower car density. This heterogeneity suggests that the health impacts of lead in drinking water may have been compounded by concurrent exposure to airborne lead emissions, particularly from leaded gasoline, which was widely used during this period and contributed substantially to environmental lead burden (Hernberg, 2000).

To explore potential mechanisms underlying the observed longevity reductions, we examine the effects of early-life lead exposure on intermediate outcomes measured in adulthood. Using the 1940 Census and WWII enlistment records, we find that individuals born in lead-exposed cities had lower educational attainment, reduced socioeconomic status, lower wage income, and diminished cognitive ability as measured by Army General Classification Test (AGCT) scores. Specifically, lead exposure is associated with 0.2 fewer years of schooling, a 29.6 % reduction in college attainment, a 14.7 % drop in wage income, and a 6.3 % decline in cognitive test scores. These findings suggest that impaired human capital formation is a key pathway through which early-life lead exposure affects old-age longevity.

The contributions of this study to the literature are threefold. First, to our knowledge, this is the first study to establish a link between early-life and childhood exposure to lead and old-age longevity. While the harmful effects of lead have long been recognized, evidence on its long-term consequences remains limited. In this context, our work adds to the literature examining both short-term and long-term effects of lead exposure. For example, Pilsner et al. (2009) show that prenatal lead exposure affects DNA methylation,

potentially increasing disease risk across the life course. Wang et al. (2017) find that higher maternal cord blood lead levels are linked to poorer birth outcomes, particularly among boys. Thomason et al. (2019) report that infants exposed to lead in utero have reduced brain connectivity, which may affect later cognitive development. Clay et al. (2019) show that children living in areas with high soil lead levels are more likely to experience cognitive challenges. Grönqvist et al. (2020) find that reduced lead exposure from gasoline phaseout in Sweden improves test scores, education, and earnings. Lee et al. (2022) use historical water pipe data to show that childhood lead exposure negatively affects cognition in old age, though it does not influence the rate of cognitive decline. We contribute to this literature by evaluating the long-term effects of lead exposure on later life longevity. We should note that longevity and mortality outcomes are extreme but precise measures of health. They contain more accurate information on health at older ages compared with other subjective measures of health. Besides, studies have suggested that longevity reflects an array of economic and health outcomes (Buchman et al., 2012; Chetty et al., 2016; Halpern-Manners et al., 2020; Kinge et al., 2019; Lubitz et al., 2003; Sunder, 2005).

Second, this paper also contributes to a broader literature that documents the relationship between exposure to various sources of airborne and waterborne pollution and a wide range of short- and long-run outcomes, including infant health, human capital formation, labor market outcomes, and adult health (Beach et al., 2016; Brainerd and Menon, 2014; Chay and Greenstone, 2003; Currie et al., 2013, 2014; Ebenstein et al., 2015; Greenstone and Hanna, 2014; Grossman and Slusky, 2019; Jones, 2019; Mettetal, 2019; Sanders, 2012; Smith et al., 2006, 2011, 2012; Zhang and Xu, 2016). For instance, Sanders (2012) examine the effect of prenatal pollution exposure on test scores. He employs the space-time variation in the recession of early 1980s as a source of reduction in Total Suspended Particulates (TPS). He finds that a one-standard-deviation decrease in TSP is associated with 6 % of a standard-deviation rise in high school test scores. Fletcher and Noghanibehambari (2024) explore the effects of fetal exposure to pesticide pollution on old-age longevity. They exploit periodical emergence of cicadas as a source of shock to pesticide use in tree-crop-lands. They show that exposure to rises in pesticide use during first year of life is associated with about 2.2 months reduction in longevity. Zhang and Xu (2016) finds that access to treated drinking water in rural China significantly increased educational attainment among youth, raising completed grades of education by an average of 1.1 years. Clifford et al. (2016) find that exposure to air pollutants—especially traffic-related ones—is associated with neurodevelopmental delays in children and cognitive decline in older adults. Colmer and Voorheis (2020) examine how reductions in prenatal air pollution from the 1970 U.S. Clean Air Act had long-term intergenerational benefits. They find that children of parents who experienced lower in-utero exposure to air pollution were significantly more likely to attend college, with effects driven by improved parental resources and investments, rather than genetic inheritance. Despite the growing number of studies in this area, evidence on the long-term effects of lead exposure—especially in relation to later-life mortality—remains limited. Our paper contributes to this ongoing line of research.

Third, this paper adds to the literature that establishes a link between early-life conditions and later-life mortality outcomes (Aizer et al., 2016; Barker, 1994, 1995, 1997; Barker et al., 2002; Goodman-Bacon, 2021; Hayward and Gorman, 2004; Montez et al., 2014; Lindeboom et al., 2010; Montez and Hayward, 2011; Noghanibehambari and Fletcher, 2021; Smith et al., 2009; Van Den Berg et al., 2006, 2011). For instance, Van Den Berg et al. (2006) and Noghanibehambari et al. (2024) find that poor early-life economic conditions are linked to higher old-age mortality. Hayward and Gorman (2004), Montez and Hayward (2011), and Smith et al. (2009) show that family socioeconomic status in early life influences longevity. Aizer et al. (2016) find that childhood cash transfers are associated with increased lifespan. Lindeboom et al. (2010) highlight a connection between childhood nutrition and later-life longevity. Lleras-Muney (2005) and Aaronson et al. (2021) show that improvements in school resources lead to better mortality outcomes in later life. We contribute to this ongoing research by examining how early-life exposure to environmental toxins, specifically lead, shapes long-term survival.

This study carries significant policy relevance. With the aging water pipe system in the U.S., many cities are increasingly vulnerable to lead contamination in drinking water (Allaire et al., 2018). This risk has been underscored by recent crises in Flint and Newark, where lead leaked into urban water supplies (Dave and Yang, 2022; Grossman and Slusky, 2019). The urgency of this issue is reflected in recent government actions, such as allocating about 1.5 % of the $1 trillion infrastructure bill passed in November 2021 to replace lead pipes. Moreover, understanding the long-run costs of lead exposure is important as it justifies the relatively high social costs of interventions (Pfadenhauer et al., 2016).

The rest of the paper is organized as follows. Section 2 provides a review of the background. Section 3 introduces data sources. Section 4 discusses the empirical methods. Section 5 reviews the results. Section 5.5 presents additional analyses that complement the main instrumental variable approach. Section 6 provides empirical evidence on the mechanisms underlying the observed effects. Finally, Section 7 concludes the paper.

2. Background

2.1. Water projects

During the 19th century, there was a notable increase in the circulation of knowledge and understanding of the microbiology of diseases, along with a growing recognition of the relevance of ensuring clean and uncontaminated water sources for the sake of public health (APHA, 1926). During this period, the United States embarked on a series of ambitious water projects to address various challenges related to water supply and water quality. This wave of water projects was driven by a growing population, urbanization, immigration, and the need for better management of water resources. Building water and sewer systems became inevitable with the growing understanding and spread of waterborne diseases such as cholera. Cities like New York, Boston, and Chicago invested in large-scale water supply systems. New York City, for instance, constructed the Croton Aqueduct (completed in 1842) and expanded it

in the late 19th century to meet rising demand.

During these decades, the country saw diverse materials used in water pipe systems. Cast iron pipes and galvanized steel, protected by a zinc coating, were largely used for their durability, longevity, and resistance to corrosion. Another material in high demand for water pipes was lead. Several technical factors and relative advantages of lead over its alternatives made it more popular nationwide. Lead water pipes could be tightly sealed, reducing the probability of leaks and ensuring a consistent flow of water. They were also easy to install, and plumbers were familiar with their features. Other reasons were their durability, availability, and corrosion resistance. In many cities, an alloy of elements, including lead and iron, was used. Further, copper, brass, and clay pipes also had their roles, with copper gaining favor for indoor plumbing due to its corrosion resistance, while clay pipes persisted in sewer systems in some regions.

2.2. Literature review

In this section, we review the literature on the life-cycle effects of lead exposure and discuss how each outcome could operate as a mediatory channel between early-life lead exposure and old-age longevity.

Medical studies suggest that pollution exposures during pregnancy change epigenetic programming, which results in a distorted growth path of the fetus (Almond and Currie, 2011; Vaiserman, 2014). Pilsner et al. (2009) provide evidence that in-utero lead exposure influences genomic DNA methylation. They argue that maternal cumulative lead burden changes epigenetic programming, increasing infants' life-cycle susceptibility to diseases. Dave and Yang (2022) explore the impacts of lead leakages in drinking water during the Newark lead-in-water crisis of 2016 on infants' health outcomes. They find that pregnant mothers in affected neighborhoods are 1.5 percentage points more likely to give birth to low birth weight infants, an increase of 18 % relative to the mean. Bui et al. (2022) explore the effects of de-leading racing cars' fuel on air quality and birth outcomes. They compare mothers' outcomes who live in the vicinity of the racetrack to those residing farther away and find that de-leading racing fuel is associated with about 100 g of additional birth weight. Wang et al. (2017) examine the association between maternal cord blood lead levels and birth outcomes. They find negative impacts on physical measures of health at birth that vary by gender, with the most effects concentrated among male infants. Several studies document the association between measures of health at birth and later-life outcomes, including mortality and longevity (Behrman and Rosenzweig, 2004; Black et al., 2007; Flouris et al., 2009; Maruyama and Heinesen, 2020; Royer, 2009; Samaras et al., 2003).

The effects of lead can be detected in infants' later-life mental development, cognitive development, and academic achievements (Gould, 2009; Goyer, 1996; Hollingsworth et al., 2022; Hu et al., 2006; Miranda et al., 2007; Nevin, 2000; Schnaas et al., 2006; Wodtke et al., 2022; N. Zhang et al., 2013). Thomason et al. (2019) examine the impact of in-utero exposure to lead on neural connectivity. They use infants' bloodspots and functional MRI data and find that lead-exposed newborns, compared with the control group reveal lower cross-hemisphere neural connectivity. They argue that this biological pathway can explain later-life reductions in cognitive ability and other regulatory functions. Clay et al. (2019) use the U.S. Census 2000 and show that 5-year-old children residing in counties with above-median surface soil lead contamination are more likely to have cognitive difficulties, including remembering, concentrating, or making decisions. Grönqvist et al. (2020) examine the impacts of life-course exposure to lead on later-life outcomes using the phaseout of leaded gasoline in Sweden. They find consistent and large impacts on test scores, high school completion, and earnings. Billings and Schnepel (2018) explore the effects of public health interventions among children with high levels of lead in their blood samples on their outcomes. They find that interventions such as lead remediation, nutritional assessment, and medical evaluations can eliminate the negative impacts on education and test scores. Sorensen et al. (2019) explore the impact of a hazard control program, a state and local joint effort to control the levels of lead in drinking water through the Flint water crisis, on children's later-life educational outcomes. They find that the program reduces the poisoning incidence by about 70 % from the baseline prevalence. Moreover, they show that each percentage-point decrease in lead poisoning is associated with 0.04 standard-deviations increase in math test scores. Aizer et al. (2018) use data from Rhode Island for children born between 1997–2005 to examine the effect of lead in blood on their test scores. They use the children's pre-school blood samples and their third-grade reading tests. They show that they show that a one-unit decrease in average blood lead level is associated with about 8 % in the probability of being below proficient in reading. The skill developments specifically through cognitive skills and educational attainments may affect old-age longevity through several channels, such as increases in income, occupational choice, social relations, peer selection, and labor market outcomes (Buckles et al., 2016; Cutler et al., 2015; Fletcher et al., 2021; Fletcher, 2012, 2015; Fletcher and Frisvold, 2014, 2015; Fletcher and Marksteiner, 2017; Fletcher and Noghanibehambari, 2021; Lleras-Muney, 2022; Lleras-Muney et al., 2020, 2005; Meghir et al., 2018; Savelyev, 2020; Savelyev et al., 2022).

Childhood lead burden can also affect later-life health outcomes. Studies suggest that about 90 % of lead is stored in bones (Rosin, 2009). Because bone development is disproportionately concentrated during early-life and childhood, children with more exposure store high amounts of lead in their bones and teeth. During old ages, when individuals face decreases in bone density, lead is released from bones and injected into the bloodstream. Therefore, individuals become internally exposed to lead load. Lee et al. (2022) use data from the Health and Retirement Study (HRS) linked with the 1940 census and examine the impact of lead burden during childhood on old-age cognition. They exploit the variation in cross-city differences in water pipe materials as a source of lead exposure. They find significant effects on later-life cognition but no effect on the rate of cognition decline. There is also suggestive evidence that childhood lead exposure is associated with adulthood and old-age chronic renal disease, cardiovascular diseases, blood pressure, hypertension, and dementia (Eid et al., 2016; Farzan et al., 2018; Lin et al., 2003; Mazumdar et al., 2012; Navas-Acien et al., 2007; Opler et al., 2004; Reuben, 2018; Rosin, 2009; Skröder et al., 2016). For instance, Skröder et al. (2016) employ longitudinal data from Bangladesh to assess the association between prenatal lead burden and children's kidney function. They find that exposure to lead during late pregnancy is associated with smaller kidney volume.

In addition to these lagged effects, several studies document the direct impact of lead exposure on contemporaneous mortality outcomes. For instance, Troesken (2008) uses data from the early 20th century U.S. and shows that areas with lead water pipe systems revealed 25–50 % higher infant mortality rates than those with non-lead water pipes. Hollingsworth and Rudik (2021) show that the use of leaded gasoline in automotive racing fuel raises blood lead rates of residents in the vicinity of racing tracks, and it is also associated with increases in elderly mortality.

3. Data sources and sample construction

The primary source of data for this study comes from Social Security Administration (SSA) Death Master Files (hereafter DMF). The DMF data covers death for male individuals with a social security number who died between 1975–2005. We extract DMF from the CenSoc Project (Goldstein et al., 2021). There are three advantages to using CenSoc-extract DMF data for the purpose of this study. First, the DMF is linked to the full-count 1940 census. Hence, we are able to extract and infer (as explained below) the city of birth. This variable is essential in examining early-life exposures that operate at a very localized level. Second, there are limited linkages between the 1940 census and several other longitudinal studies, such as the Health and Retirement Study, National Health and Aging Trends Study, Panel Study of Income Dynamics, etc. However, the resulting linked data provides a very small sample size with low power.[1] In contrast, our analysis sample contains millions of observations, allowing us to detect statistical effects and implement heterogeneity analyses. Third, the linked sample has information about a wide array of family covariates and individual characteristics. We employ this information to search for mechanisms of impact and to implement balancing tests.

We extract data on the city-level pipeline materials from Feigenbaum and Muller (2016) and Clay et al. (2014). The data contains information about the primary materials used for each city's water pipes for 553 cities across the US. In order to merge water system data with DMF records, we need to infer the city of birth for each individual. In so doing, we start by linking DMF records to the full-count 1940 census extracted from Ruggles et al. (2020). We then use cross-census linking rules provided by Price et al. (2021) to merge the DMF-census-linked data with historical census 1900, 1910, 1920, and 1930. Including the city information in 1940, we have at least one and at most five city identifiers for each individual. For instance, for a person born in 1912, we potentially can observe their census city in 1920, 1930, and 1940. If merging provides null results, we can only observe his 1940 geographic identifier. Therefore, we have between 1–3 identifiers for this cohort. We use the earliest city that is observed for each individual to use as a proxy for the city of birth and childhood. We then merge DMF with the city-level lead database based on inferred city-of-birth. In further analyses for mechanisms of impact, we also employ a subsample of data from DMF records that are linked with World War II enlistment data extracted from Goldstein et al. (2021). This data contains information on Army General Classification Test (AGCT) scores reported by enlistment agencies. The AGCT was a standardized test used by the U.S. military to measure recruits' cognitive abilities and aptitudes during World War II. The AGCT score was the primary criterion for assigning enlistees to military tasks and positions. We use the AGCT as a measure of cognitive ability in early adulthood. Although we rely on the linkage provided by Goldstein et al. (2021), we acknowledge that Ferrie et al. (2012) were the first to assemble and analyze AGCT data in the context of historical environmental exposures.

Our analysis sample includes individuals born between 1900 and 1940 who are observed in the DMF data (death years 1975 – 2005). As such, individuals must have survived to at least age 35 (if born in 1940) to be included in the dataset, with the minimum required age increasing for earlier birth cohorts. This may introduce a survival-based sample selection, which may bias our estimates if early-life lead exposure disproportionately affected infant or early adult mortality, as suggested by the literature (Clay et al., 2014, 2024; Lanphear et al., 2018). If lead exposure increased mortality risks at younger ages, the affected individuals are underrepresented in our sample, thereby leading to conservative estimates of the true effect—i.e., our results would understate the full impact of lead exposure on longevity.

Fig. 1 depicts lead versus non-lead cities in the final sample. Fig. 2 illustrates a density distribution of age-at-death for individuals born in lead and non-lead cities. There are no visually discernible differences in the distribution of age-at-death between individuals born in lead versus non-lead cities. Table 1 provides summary statistics of the final sample for cities with some lead materials in their water system (lead cities, first panel) and cities without any lead compounds in the water system (non-lead cities, second panel). Individuals born in lead and non-lead cities live, on average, 874.5 (72.9) and 875.1 (72.9) months (years). The share of whites in both groups is quite comparable (96 %). The share of Blacks and Hispanics in lead cities (non-lead cities) is 3.5 % and 0.6 % (3.8 % and 2.2 %), respectively. Roughly 4.8 and 3.3 % of lead and non-lead cities have acidic water. Roughly 71 % and 67 % of mothers in lead and non-lead cities are literate, respectively. Moreover, in the sample for mechanism analysis (middle panel), we observe quite comparable statistics for selected outcomes across the two groups.

In Table 2, we document several selected city-level characteristics across both lead and non-lead groups. This data is extracted from historical censuses 1900–1940 and interpolated linearly for inter-decennial years. On average, lead cities have a higher share of whites, a lower share of Blacks, a lower share of Hispanics, and a lower share of other races. Further, both groups reveal almost identical socioeconomic index, urbanization, literacy rate, the share of married, the share of homeowners, and the number of children under five years old in households.

[1] For instance, Health and Retirement Study provides a linked sample of 9,654 people.

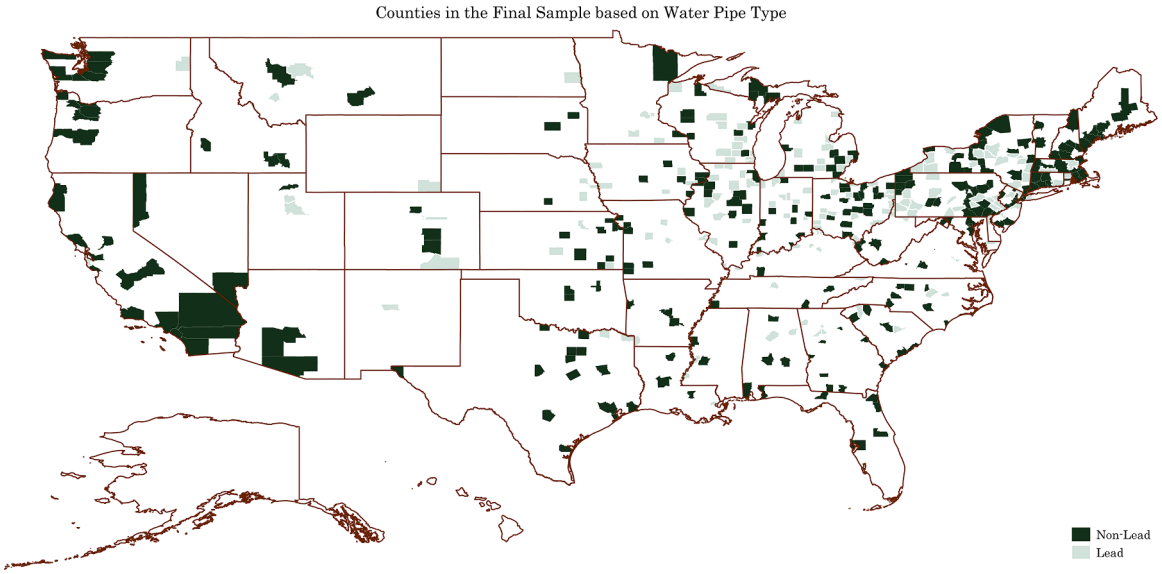

Fig. 1. Distribution of counties in the final sample based on lead status.

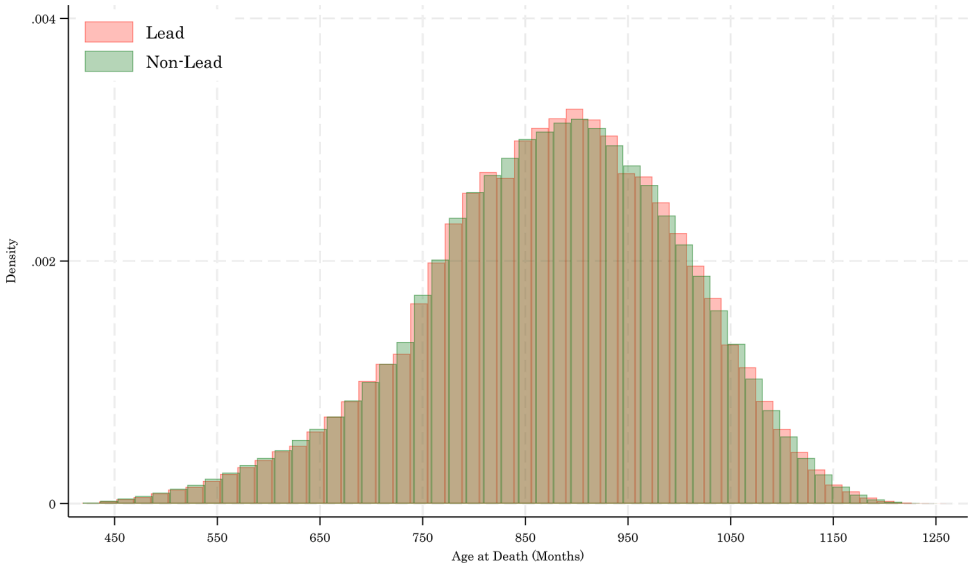

Fig. 2. Density distribution of age-at-death in the final sample based on birth-city lead status.

4. Econometric method

We start our analysis by documenting cross-sectional correlations between birth-city lead status and later life longevity using the following ordinary least square regression:

$$DA_{icst} = \alpha_0 + \alpha_1 Lead_c + \alpha_2 X_i + \alpha_3 Z_{ct} + \theta_{st} + \varepsilon_{icst} \quad (1)$$

Where the outcome is age-at-death (measured in months) of individual i born in city c in state s and year t. The variable *Lead* is a dummy that equals one if the individual is born in a lead city and zero otherwise. In X, we include race and ethnicity dummies as individual covariates and maternal literacy and paternal socioeconomic index dummies as parental covariates. The matrix Z contains birth-city level covariates listed in Table 2. The parameter θ represents birth-state-by-birth-year fixed effects that absorb cohort convergence in health outcomes across different states and all other time-varying state-specific shocks. Finally, ε is a disturbance term. We cluster standard errors at the birth-city and birth-year level to account for serial and spatial correlations in terms, respectively.

To account for selection caused by unobserved heterogeneity across cities, we employ two-stage least square estimations and exploit the fact that proximity to major lead refineries reduces transportation costs, which provides incentives for local authorities and

Table 1
Summary statistics.

	Lead Cities		Non-Lead Cities	
	Mean	SD	Mean	SD
DMF-Census Data:				
Death Age (Months)	874.4525	125.2236	875.0655	126.3523
Birth-year	1918.3478	9.69	1918.477	9.7149
Death Year	1991.224	8.724	1991.4034	8.7156
White	.964	.1863	.9585	.1995
Black	.0354	.1848	.0379	.1911
Hispanic	.0057	.075	.0219	.1465
Acidic Water	.0479	.2135	.0333	.1795
Log Distance to the Closest Lead Refinery	4.9128	.8197	4.0046	1.3257
Father SEI Missing	.1175	.3221	.1371	.3439
Father SEI below Median	.4602	.4984	.4399	.4964
Father SEI above Median	.4222	.4939	.423	.494
Mother literate	.7128	.4525	.6734	.469
Mother literacy missing	.2297	.4206	.2537	.4351
Observations	783,483		1191,919	
Sample for Mechanism Analysis:				
Years of Schooling	10.6018	2.83	10.7018	2.9414
Years of Schooling < 9	.2781	.4481	.281	.4495
Years of Schooling < 12	.5541	.4971	.545	.498
Socioeconomic index	33.4825	21.6874	35.0934	22.1391
Occupational income score	26.6185	9.2452	26.9556	9.7116
Log wage income	6.7657	.8659	6.8019	.8723
Income percentile	59.6751	31.5305	59.9709	32.0105
Observations	479,544		724,787	
DMF-World War II Enlistment Data:				
AGCT score	77.6849	46.1731	74.1283	48.3585
Observations	8238		13,656	

Table 2
Characteristics of lead and non-lead cities in the final sample.

	Lead Cities		Non-Lead Cities	
	Mean	SD	Mean	SD
Population	67,846.856	137,855.76	80,095.374	380,227.22
Share of whites	.937	.1097	.9092	.139
Share of Blacks	.0622	.1099	.0879	.14
Share of Hispanics	.0032	.0092	.0131	.0545
Share of other races	.0009	.0034	.0029	.0089
Share of females	.5033	.0242	.5055	.0269
Average socioeconomic index	31.4101	4.644	31.4871	4.1679
Female labor force participation rate	.264	.1019	.2806	.1066
Share of married	.5807	.0405	.5796	.0431
Literacy rate	.7064	.3085	.722	.347
Urbanization rate	.99	.08	.994	.0628
Share of institutionalized	.0057	.0214	.0047	.0134
Share of homeowners	.4458	.1198	.4323	.1081
Number of children less than five years old	.3416	.0881	.3267	.0866
White-collar occupations per capita	.0494	.0225	.0521	.024
Farmers per capita	.0072	.0113	.0098	.015
Other occupations per capita	.9401	.0264	.9346	.0302
Observations	9236		12,904	

policymakers to consider leaded materials for water pipe systems. The primary assumption is that the location of lead refineries does not correlate with other characteristics of cities that may contribute to population health outcomes and that their effects operate solely through the availability and use of lead in water systems. This assumption plausibly holds for several reasons. The location of lead refineries primarily depends on proximity to lead ore deposits, such as those containing galena (the primary lead mineral), which were formed over geological timescale long before human settlements. Since lead smelting required significant amounts of fuel, the location decisions also depended on the availability of energy sources such as wood or charcoal. Further, technological improvements and new innovations in the energy sector and in lead processing also interacted with the location of lead refineries. For instance, the innovations of blast furnaces and cupellation for lead smelting during the 19th century made specific locations economically feasible to establish lead refineries. These factors are less likely to correlate with influences of population health and longevity.

We operationalize our two-stage least square estimation using regressions of the following form:

$$Lead_{icst} = \beta_0 + \beta_1 LogDistRef_c + \beta_2 X_i + \beta_3 Z_{ct} + \gamma_{st} + \epsilon_{icst} \quad (2)$$

$$DA_{icst} = \alpha_0 + \alpha_1 \widehat{Lead}_c + \alpha_2 X_i + \alpha_3 Z_{ct} + \theta_{st} + \varepsilon_{icst} \quad (3)$$

Eqs. (2) and (3) represent first-stage and second-stage regressions, respectively. The variable *LogDistRef* measures log of distance (in miles) to the nearest lead refinery. The parameters γ and θ are birth-state by birth-year fixed effects in first and second-stage regressions, respectively. All individual, family, and city controls are as in Eq. (1).

5. Results

5.1. Compositional differences in lead and non-lead status

Certain observed and unobserved characteristics of cities may correlate with their lead status. For instance, progressive state-level taxation might provide additional resources that cover the extra cost of lead implementation compared with its relatively cheaper alternatives. Further, Public health awareness and concerns among city authorities might discourage the use of lead in pipeline infrastructure. These aspects could influence population health and longevity in numerous unobserved ways, confounding our cross-sectional estimates.

Although we acknowledge the unobserved differences between lead and non-lead cities, we are curious to what extent these two groups differ based on observable characteristics. In so doing, we use a city-by-year panel extracted from decennial censuses 1900–1940 and interpolated linearly for inter-decennial years. We regress several observable characteristics on city lead status, conditional on state-by-year fixed effects. These results are reported in Table 3. Lead cities have, on average, a lower population, a higher share of whites, and a lower share of Blacks (columns 1–3). They reveal a slightly lower socioeconomic index but lower female labor force participation and literacy rates (columns 7, 8, and 10). Further, there are more families with children under five years in lead cities than in non-lead cities (column 14). Fewer white-collar occupations per population are recorded in lead cities than in non-lead cities (column 15). There are four noteworthy points in this table. First, the point estimates imply relatively small changes in the outcomes. For example, the coefficient for the share of whites corresponds to a change of approximately 0.7 % relative to the mean. Second, the differences are not consistent—for instance, we observe an increase in the share of whites but a decrease in literacy rates. Third, all point estimates in columns 6–16 are statistically insignificant. Fourth, even among the statistically significant coefficients,

Table 3
Differences in city-level characteristics of lead and non-lead cities.

	Outcomes:			
	Population	Share of whites	Share of Blacks	Share of Hispanics
	(1)	(2)	(3)	(4)
Lead	−2.4282	.0073*	−0.0068*	−0.0023***
	(3.4636)	(0.0041)	(0.004)	(0.0008)
Observations	21,799	21,799	21,799	21,799
R-Squared	.0241	.8715	.8751	.4189
Mean DV	7.511	0.929	0.069	0.011
	Share of other races	Share of females	Socioeconomic index	Female labor force participation rate
	(5)	(6)	(7)	(8)
Lead	−0.0005*	−0.0001	−0.4907	−0.0083
	(0.0002)	(0.0014)	(0.303)	(0.0052)
Observations	21,799	21,799	21,799	21,799
R-Squared	.805	.4419	.3988	.8242
Mean DV	0.003	0.503	32.517	0.304
	Share of married	Literacy rate	Urbanization rate	Share of institutionalized
	(9)	(10)	(11)	(12)
Lead	−0.0013	−0.0011	−0.0002	.0002
	(0.0032)	(0.0041)	(0.0011)	(0.0009)
Observations	21,799	21,799	21,799	21,799
R-Squared	.6466	.8959	.0374	.1
Mean DV	0.573	0.657	0.999	0.005
	Share of homeowners	Number of children less than five years old	Employed in white-collar occupations per capita	Employed in all other non-farm occupations per capita
	(13)	(14)	(15)	(16)
Lead	.025	.0025	−0.0013	.002
	(0.0195)	(0.005)	(0.0013)	(0.0014)
Observations	21,799	21,799	21,799	21,799
R-Squared	.5291	.75	.456	.4254
Mean DV	0.352	0.310	0.052	0.939

Notes. Standard errors, clustered on city and year, are reported in parentheses. All regressions include state-by-year fixed effects and are weighted using city-level population.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

the results suggest that lead cities might exhibit higher longevity due to a greater share of whites and a lower share of Blacks.

One might argue that the selection process of the final sample might add different sociodemographic and socioeconomic compositions among individuals born in lead versus non-lead cities, and those differences are not fully revealed by cross-city comparisons reported in Table 3. We address this concern by examining differences in observable characteristics of individuals in the final sample born in lead versus non-lead cities, conditional on birth-state by birth-year fixed effects. These results are reported in Appendix Table A-1. Those individuals born in lead cities are more likely to be white, less likely to be black, more likely to be from low socioeconomic index families, and more likely to have literate mothers (columns 1, 2, 3, and 6). However, when we compare these estimates relative to the mean of the outcomes, we find quite small implied changes. For instance, the implied percentage changes for non-Hispanic white and mother literate are 0.6 % and 1.4 %, respectively. Further, they do not point to consistent differences as we observe a higher share of whites but also a higher share of families with low socioeconomic status and, simultaneously, higher maternal literacy.

5.2. *Two-stage least square estimations*

We begin by presenting the correlations between birth city lead exposure status and longevity using Eq. (1) in columns 1–2 of Table 4. The point estimates compare the longevity of individuals born within the same census region (column 1) and within the same state (column 2), conditional on individual, family, and city-level covariates. Those born in cities with lead in their water pipeline experience roughly 0.6 months lower longevity.

The results of the first stage regressions of Eq. (2) are reported in columns 3–4 of Table 4. Doubling the distance between birth-city and the closest lead manufacturing refinery is associated with a 21.6 percentage points increase in the likelihood of lead status, off a mean of 0.39. This is a large impact, implying a 53 % increase from the baseline. The second stage regressions of Eq. (3) are reported in columns 5–6. The fully parameterized regression of column 6 suggests that birth-city lead status is associated with 9.7 months lower longevity. This is roughly 16 times larger than the OLS coefficients of column 2. Although the larger magnitude points to underestimated coefficients in OLS regressions, it also provides Local Average Treatment Effects (LATE). It picks up on the variations induced by specific locations of lead refineries and the degree to which cities in the vicinity of these refineries consider their proximity and their decisions about the implementation of lead in pipelines. Therefore, we should exercise caution in interpreting the results as the LATE coefficients only identify treatment effects among the subset of compliers.

One concern in interpreting these results is the potential violation of the exclusion restriction when using distance to refinery as an instrument. Refineries may have contributed to lead pollution in drinking water or air. Additionally, they were more likely to be constructed in areas with greater market access—such as those with higher percentages of railroads or proximity to larger cities. Market access, in turn, may influence city growth through improvements in land value, healthcare access, and job opportunities. Since these "side effects" of the distance-to-refinery instrument are also linked to health and later-life longevity, it is possible that the exclusion restriction does not fully hold. That is, proximity to lead refineries may influence longevity not only through their effect on lead pipe usage but also through other pathways such as local pollution or market access, which would compromise the instrument's validity even if the first-stage relationship is strong.

To assess the extent to which these intermediary factors might bias our IV estimates, we employ the bounding approach developed by Conley et al. (2012). This method allows for a non-zero correlation (γ) between the instrument and the outcome. If the instrument perfectly satisfies the exclusion restriction, then γ equals zero. For cases where γ deviates from zero, the method generates lower and upper bounds for the 2SLS-IV coefficient, based on the researcher's priors about the plausible range of γ.

To better inform our assumptions about γ, we draw on existing literature. Donaldson and Hornbeck (2016) show that railroad expansions in the late 19th and early 20th centuries increased agricultural land values by about 60 %. Noghanibehambari and Fletcher (2024) argue that the Dust Bowl of the 1930s led to a 30 % reduction in agricultural land values and a 0.85-month reduction in longevity for those exposed in early life. Based on this, the upper bound of the direct influence through the "market access" channel is approximately 1.7 months. On the lower end, in our OLS regressions with and without the IV, the coefficient changes by <0.2 months. Although the literature on early-life exposure to airborne lead is limited, this suggests a plausible lower bound for γ of about -0.2. As shown in the bottom panel of Table 4, the resulting bounds for γ imply IV estimates ranging from approximately -18.5 to -4.8 months.[2]

One way to understand the magnitude of the estimated effect of Table 4 is to compare it with the documented effects of other shocks on longevity in studies that employ similar data, time periods, historical settings, and outcomes. For instance, Noghanibehambari and Fletcher (2023) investigate the effects of early life exposure to the Dust Bowl of the 1930s on old-age male longevity and find a reduction of about 2.5 months for individuals whose fathers are farmer and have higher exposure to the topsoil erosion due to the Dust Bowl. Since the Dust Bowl represented an unprecedented environmental catastrophe with large and long-lasting shocks to agricultural income and land value, the fact that the effect of lead exposure is about 3.8 times larger than exposure to the Dust Bowl is quite significant and policy-relevant.

Our results are also comparable to those of other studies examining the effects of exposure to toxic substances and hazardous chemicals on later life outcomes. For instance, Fletcher and Noghanibehambari(2024) investigate the impacts of early-life exposure to agrichemicals and pesticides on later-life male longevity using the emergence of cicadas between 1920–1940 as a measure that

[2] We note that the bounding IV estimates are calculated based on the specification in column 5, which includes region-by-year fixed effects. This choice is primarily due to non-convergence issues encountered when using the more saturated state-by-year fixed effects model in column 6.

Table 4
Two-stage least square estimations of lead status on old-age longevity.

	Ordinary Least Square, Outcome: Age-at-Death (Months)		*First Stage Outcome: Lead Status*		*Second-Stage Outcome: Age-at-Death (Months)*	
	(1)	(2)	(3)	(4)	(5)	(6)
Log distance to the closest lead refinery			.198***	.216***		
			(0.007)	(0.006)		
Lead	−0.506***	−0.613***			−7.729***	−9.701***
	(0.194)	(0.202)			(0.941)	(0.976)
Observations	1975,402	1975,397	1975,402	1975,397	1975,402	1975,402
R-Squared	.404	.405	.301	.45	.403	.404
Mean DV	874.822	874.822	0.397	0.397	874.822	874.822
First-Stage F-Statistics					825.689	1260.775
Birth region by birth-year F.E.	✓	✓	✓	✓	✓	✓
Controls	✓	✓	✓	✓	✓	✓
Birth-state by birth-year F.E.		✓		✓		✓
Bounding IV Estimates of Conley et al. (2012)						
Range of γ	Lower Bound	Upper Bound				
[0,0]	−9.5	−5.8				
[−0.1,0.1]	−10.1	−5.4				
[−0.3,0.3]	−11.1	−4.4				
[−0.6,0.6]	−12.7	−2.9				
[−0.9,0.9]	−14.2	−1.4				
[−0.2,1.7]	−18.5	−4.8				

Notes. Standard errors, clustered on birth-city and birth-year, are reported in parentheses. Controls include dummies for race and ethnicity, dummies for paternal socioeconomic index, dummies for maternal literacy, and birth-city covariates, including average socioeconomic index, female labor force participation rate, share of married, literacy rate, average occupational prestigious score, urbanization rate, and share of females.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

increases pesticide use in the agricultural sector. They document a reduction of about one year in the longevity of those whose fathers are employed in the agricultural sector. This effect is comparable to the effects of lead exposure, documented in Table 4.

5.3. Heterogeneity

Table 5 reports a series of heterogeneity analyses that explore whether the estimated effects of early-life lead exposure on later-life longevity vary across different demographic groups and environmental conditions. To provide a clearer picture of the results across subsamples, we organize the findings into four panels. Panel A presents the ordinary least squares (OLS) estimates. Panel B reports the reduced-form estimates of the effect of the instrument—minimum distance to the nearest lead refinery—on longevity. Panel C shows the first-stage results, capturing the relationship between the instrument and lead exposure status. Finally, Panel D presents the second-stage regression results. We discuss these findings in detail below.

Heterogeneity by Age of Water Pipeline - Older pipelines are more susceptible to corrosion, and while it is true that corrosion can sometimes result in the formation of protective mineral scales that limit lead leaching, this protective effect is not guaranteed and depends on water chemistry and maintenance practices. In many historical cases—particularly in the absence of corrosion control treatments—aging pipes experienced degraded protective layers, leading to increased lead release over time (Edwards and Dudi, 2004; EPA, 2024; Renner, 2009). Our empirical strategy tests whether cities with older pipeline systems saw stronger negative effects of lead exposure, under the hypothesis that degradation of pipe coatings over time resulted in greater lead leaching into drinking water. We use the date of water system installation extracted from Baker (1897) and calculate the age of the water system based on the year of installation and the year the individual was born. We replicate the analysis in subsamples based on the age of the water system, distinguishing between cities with below-median (younger pipelines) and above-median (older pipelines) pipeline age (columns 2–3). We observe larger coefficients in cities with older pipelines, consistent across the OLS, reduced-form, and 2SLS estimates.

Heterogeneity by Car Density – To examine whether the health impacts of lead in water were exacerbated by concurrent exposure to airborne lead, we exploit variation in automobile density across states in 1940 using historical vehicle registration data from Rahman (2024). During this period, leaded gasoline was widely used, and emissions from vehicles were a major source of ambient lead exposure (Hernberg, 2000). In so doing, we stratify the sample by above- and below-median state-level car density and report the results in columns 4–5 of Table 5. In the subsample of states with low automobile density, we find a negative but smaller and less precise effect of birth-city lead status on longevity: a reduction of approximately 6 months. In contrast, in the high car density subsample, the effect is much larger and statistically significant, with a reduction of about 11 months in longevity. These findings are consistent with the hypothesis that dual exposure—from both lead-contaminated drinking water and airborne lead emissions from vehicles—may have amplified the long-run health effects.

Heterogeneity by Water Chemistry – Acidic water may react with protective coatings and other oxide layers on the inner surface of lead pipe fixtures. Such reactions allow the materials to break down and enable lead to leach into the water supply. Acidic water can also corrode plumbing materials that have partial lead. The more the water is acidic, the faster the corrosion. Chemical reactions caused by corrosion result in the lead dissolving into the water supply. Therefore, one expects to observe higher correlations between

Table 5
Heterogeneity analysis.

	Full Sample	Younger Pipeline	Older Pipeline	Above-Median Car Density in 1940	Below-Median Car Density in 1940	Non-Acidic Water	Acidic Water	Nonwhite	White	Illiterate mothers	Literate mothers	Third-Higher Generation Immigrant	Second Generation Immigrant
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
Panel A. OLS, Outcome: Age-at-Death (Months)													
Lead	−0.613***	−0.232	−1.581***	−1.749***	.828***	−0.938***	.571	1.645*	−0.682***	−0.142	−0.802***	−0.091	−0.853***
	(0.202)	(0.29)	(0.294)	(0.342)	(0.231)	(0.229)	(0.472)	(0.952)	(0.206)	(0.322)	(0.231)	(0.299)	(0.247)
Observations	1975,397	958,386	1017,005	883,376	1092,021	1701,150	274,245	77,583	1897,695	614,257	1361,014	626,663	1348,670
R-Squared	.405	.29	.292	.402	.407	.406	.399	.447	.4	.493	.274	.358	.348
Panel B. Reduced Form, Outcome: Age-at-Death (Months)													
Log Minimum Distance to Refinery	−2.1***	−1.799***	−2.805***	−2.845***	−0.156	−2.301***	2.893***	−0.748	−2.141***	−2.001***	−2.137***	−1.182***	−2.471***
	(0.205)	(0.255)	(0.381)	(0.289)	(0.275)	(0.212)	(0.718)	(0.853)	(0.208)	(0.26)	(0.234)	(0.258)	(0.222)
Observations	1975,397	958,386	1017,005	883,376	1092,021	1701,150	274,245	77,583	1897,695	614,257	1361,014	626,663	1348,670
R-Squared	.405	.29	.292	.402	.407	.406	.399	.447	.4	.494	.275	.358	.348
Panel C. First Stage Outcome: Lead													
Log Minimum Distance to Refinery	.216***	.195***	.264***	.244***	.025**	.228***	−0.028	.175***	.218***	.216***	.219***	.207***	.219***
	(0.006)	(0.009)	(0.009)	(0.007)	(0.01)	(0.006)	(0.031)	(0.009)	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)
Observations	1975,397	958,386	1017,005	883,376	1092,021	1701,150	274,245	77,583	1897,695	614,257	1361,014	626,663	1348,670
R-Squared	.45	.435	.563	.485	.365	.529	.422	.5	.449	.464	.445	.408	.473
Panel D. Second Stage Outcome: Age-at-Death (Months)													
Lead	−9.701***	−9.2***	−10.639***	−11.655***	−6.133	−10.08***	−102.48	−4.281	−9.814***	−9.26***	−9.737***	−5.717***	−11.265***
	(0.977)	(1.29)	(1.573)	(1.347)	(10.911)	(0.982)	(115.918)	(4.895)	(0.984)	(1.253)	(1.094)	(1.228)	(1.078)
Observations	1975,397	958,386	1017,005	883,376	1092,021	1701,150	274,245	77,583	1897,695	614,257	1361,014	626,663	1348,670
Test of Equality of Coefficients in the Two Subsamples:													
χ^2		4.84		44.94		48.23		2.68		0.25		29.45	
$prob > \chi^2$		0.02		0.00		0.00		0.10		0.62		0.00	

Notes. Standard errors, clustered on birth-city and birth-year, are reported in parentheses. All regressions include birth-state by birth-year fixed effects and individual/family/city controls. Controls include dummies for race and ethnicity, dummies for paternal socioeconomic index, dummies for maternal literacy, and birth-city covariates, including average socioeconomic index, female labor force participation rate, share of married, literacy rate, average occupational prestigious score, urbanization rate, and share of females.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

lead status and health outcomes in cities with more acidic water if the negative associations are driven by lead exposure (Ferrie et al., 2012; Kim et al., 2011). This method of identifying the effects of lead exposure has been employed in previous studies across various contexts (Clay et al., 2014; Feigenbaum and Muller, 2016; Troesken, 2008). We use city-level pH data in 1954 reported by Lohr and Love (1954a) and Lohr and Love (1954b) to infer whether the water is acidic (i.e., pH<7). We then stratify the sample based on acidic versus non-acidic water and replicate the analysis. These results are reported in columns 6–7 of Table 5. Although the overall 2SLS coefficient for cities with acidic water is substantially large, the large standard errors limit any meaningful interpretation or comparison. Moreover, the small and statistically insignificant coefficient in the first-stage regression suggests that the instrument does not perform well in this subsample.

Sociodemographic Heterogeneity – In columns 8–9 of Table 5, we examine the heterogeneity in two-stage least square estimations based on race. Among whites, we observe a reduction of 9.8 months in longevity. However, we observe an insignificant decrease of 3.8 months among Blacks. Additionally, we cannot statistically rule out the difference between the coefficients of columns 8 and 9.

Between 1900–1940, black infant mortality rates were substantially higher than white infant mortality rates (Eriksson et al., 2018). Additionally, there is evidence that lead exposure during the early decades of the 20th century in the U.S. was associated with increases in infant mortality rates (Clay et al., 2014). Further, there is evidence that at considerably high infant mortality regimes, selection pressures dominate scarring effects to influence long-term outcomes (Bozzoli et al., 2009; Nobles and Hamoudi, 2019). Therefore, one argument for the smaller association among Blacks is that the higher infant mortality regime experienced by Blacks during this period enables a degree of selection dominance over scarring, hence the overall impact becomes smaller than that of whites who experience lower selection pressures.

Moreover, we observe quite comparable coefficients among individuals with literate and illiterate mothers (columns 10–11). This fact suggests that lead-longevity associations are possibly primarily driven by biological mechanisms and reveal a lower degree of interaction with other social determinants of health.

Heterogeneity by Immigrant Generational Status – In the next two columns, we stratify the sample based on immigrant generational status. Specifically, we separate second-generation immigrants—individuals born in the U.S. with at least one foreign-born parent—from third or higher-generation immigrants, whose parents were also U.S.-born. We find that the negative effect of early-life exposure to leaded water is considerably larger among second-generation immigrants, with an estimated reduction in longevity of approximately 11.3 months. Among third and higher-generation individuals, the effect is smaller at 5.7 months, though still negative and meaningful. This pattern may reflect differences in household financial vulnerability, living conditions, or occupational exposures (Bullard, 2018; Collins and Zimran, 2019; Cutler and Miller, 2005). Second-generation immigrant families may have faced more crowded or economically constrained environments, potentially increasing susceptibility to the health impacts of environmental toxins.

Heterogeneity by Gender – A growing body of research suggests that the health effects of environmental toxin exposure may vary by gender, with male individuals often exhibiting greater vulnerability (Almeida Lopes et al., 2017; Froehlich et al., 2009; Jedrychowski et al., 2009; Wright et al., 2008). While the DMF data only includes mortality for male individuals, we explore gender-based heterogeneity using the Social Security Administration Numident data, which includes both men and women. However, the Numident data has an important limitation: it only covers death records from 1988 to 2005, a substantially narrower window than the DMF (1975–2005). This constraint is particularly binding in our setting. When we restrict the DMF sample to 1988–2005, the estimated effect of early-life lead exposure on longevity drops by about 82 %, from 9.6 months to 1.7 months (Appendix Table B-2, Column 1).

Despite this limitation, the Numident data yields a similar estimate for male individuals, suggesting consistency with our DMF-based results (Appendix Table B-2, Column 2). In contrast, when we turn to female individuals in the Numident sample, the estimated effect drops by an additional 57 %, suggesting possible gender differences in vulnerability to early-life lead exposure

Table 6
Two-stage least square estimations to examine the robustness to additional controls and alternative functional forms.

	Adding more city level controls	Outcome: log age-at-death	Outcome: age-at-death > 65	Outcome: age-at-death > 70	Outcome: age-at-death > 75	Distance from Lead Distillery > 30 Miles	Distance from Lead Distillery > 60 Miles
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Lead	−9.624***	−0.011***	−0.026***	−0.032***	−0.032***	−5.79***	−11.178***
	(1.279)	(0.001)	(0.004)	(0.004)	(0.004)	(1.999)	(2.004)
Observations	1975,402	1975,402	1975,402	1975,402	1975,402	1406,585	1311,422
R-Squared	.404	.403	.255	.29	.257	.406	.406
Mean DV	874.822	6.763	0.756	0.591	0.404	872.452	872.472
First-Stage F-Stat	1109.884	1260.775	1260.775	1260.775	1260.775	110.887	94.017

Notes. Standard errors, clustered on birth-city and birth-year, are reported in parentheses. The outcome in the first column is age-at-death measured in months. All regressions include birth-state by birth-year fixed effects and individual/family/city controls. Controls include dummies for race and ethnicity, dummies for paternal socioeconomic index, dummies for maternal literacy, and birth-city covariates, including average socioeconomic index, female labor force participation rate, share of married, literacy rate, average occupational prestigious score, urbanization rate, and share of females. Column one adds to these covariates by including share of different age groups, share of different race groups, share of homeowners, share of institutionalized population, share of immigrants, share of different occupation groups, and school attendance rate.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

(Appendix Table B-2, Column 3). Although we interpret these findings cautiously due to sample constraints, they suggest that the adverse longevity effects of lead exposure may be stronger among men.

5.4. Robustness checks

In Table 6, we examine the robustness of the two-stage least square results to alternative specifications and functional forms. In column 1, we add a wide array of additional city-level covariates, including the share of different age groups, share of different race groups, share of homeowners, share of institutionalized population, share of immigrants, share of different occupation groups, and school attendance rate. The estimated coefficient is almost identical to that of the main results.

In column 2, we replace the outcome with log age-at-death. Birth-city lead status is associated with a 1.1 % increase in age-at-death, almost identical to the implied percentage change of the coefficient in column 4 of Table 4. In columns 4–6, we replace the outcome with dummy variables indicating longevity beyond 65, 70, and 75, respectively. Compared with the mean of the outcomes, the estimated coefficients imply a change of roughly 3.4 %, 5.4 %, and 7.9 %, respectively. The effects become larger as individuals reach older ages, suggesting the latent impacts of lead exposure during earlier years of life.

In the last two columns of Table 6, we conduct a robustness check by restricting the sample to cities located >30 and 60 miles away from the nearest lead refinery. This approach functions as a type of donut-hole analysis, aimed at ruling out the possibility that our main results are driven by direct environmental pollution (e.g., airborne or waterborne lead contamination) emanating from proximity to the refineries themselves. If the observed reductions in longevity were primarily due to direct exposure from living near a lead manufacturing site, we would expect the estimated effects to diminish as we exclude cities closer to the refineries. However, the results indicate otherwise, with estimated coefficients that are comparable to the main results and remain both statistically significant and economically meaningful.

To address potential selection bias in our analysis sample driven by cross-census linking as well as census-DMF linking, we implement an inverse probability weighting (IPW) strategy (Bailey et al., 2020; Halpern-Manners et al., 2020). We begin with the full-count 1940 Census and restrict the data to male cohorts born between 1900 and 1940 in cities included in our final sample. We then link this dataset to our analysis sample using individuals' unique *histid* identifiers and create a binary indicator for successful linkage. Next, we estimate a probit regression in which the dependent variable is the linkage indicator and the explanatory variables include individual-level characteristics (race and ethnicity, maternal literacy, and paternal socioeconomic index), city-level controls, and birth state by birth year fixed effects. This regression predict the probability that an individual with a given set of characteristics appears in the final sample. We then use the inverse of this predicted probability as a weight in our main regression specifications. Appendix Table B-1 reports the results of this weighted analysis. Both the first-stage and second-stage estimates remain consistent with our main results.

5.5. Additional analyses

Early versus Mid-Life Exposure – As mentioned in section 2.2, lead is primarily stored in bones and teeth. Since bone development is concentrated in childhood years, children with high exposure accumulate more lead in their bones and teeth. In old age, as bone density decreases, this lead is released into the bloodstream, causing internal exposure. This biological mechanism implies the relevance of exposure during earlier years of life compared to adulthood years for the latent impacts on old-age longevity.

In a similar line of argument, studies that examine the long-term effects of environmental exposures usually point to the relevance of certain critical ages during early life and childhood (Almond et al., 2018; Almond and Currie, 2011). One potential implication is that we may observe discernible differences in lead-longevity correlations based on the age group of exposure. However, city-level lead status does not change over time in our sample period. One solution is to look at the sample of individuals who migrate from the city of birth to another city with a different lead status. To implement such comparisons, we focus on the subsample of migrants who belong to birth cohorts of 1920–1940. Therefore, we have information on lead status in 1940 (early adulthood exposure) as well as lead status at birth (early life exposure). We implement regressions similar to Eq. (1) and report the results in Appendix Table A-2. For this subsample, the difference in longevity based on birth-city lead status is about 2.6 months, conditional on controls and fixed effects (column 1). In column 2, we add 1940 city fixed effects to compare individuals born in different lead-status cities who migrated to the same city. The estimated coefficient is almost identical to that of column 1, suggesting that the destination city characteristics do not confound the estimates of column 1. In column 3, we compare lead status in birth-city and early-adulthood-city for individuals who migrated to another city with a different lead status than their birth-city. We observe a considerably larger coefficient for birth-city than the 1940 city lead status, implying the relevance of early-years exposures rather than later childhood/early adulthood exposures.

6. Candidate mechanisms

Several strands of research suggest that early-life and childhood exposure to pollution, and specifically lead burden, may affect skill formation, human capital accumulation, and labor market outcomes (Beach et al., 2016; Currie et al., 2014; Sanders, 2012; Sorensen et al., 2019; Taylor, 2022; Zhang and Xu, 2016). On the other end, studies point to the influence of income, socioeconomic status, and educational attainments in determining old-age mortality outcomes (Cutler et al., 2006; Fletcher, 2015; Lleras-Muney, 2005; Mazumder, 2008; Meghir et al., 2018; Miller and Bairoliya, 2021). Therefore, we would expect to observe changes in the trajectory of education and socioeconomic status as mediatory pathways between early-life lead exposure and later-life longevity. We focus on individuals above age 19 and examine their characteristics in the 1940 census employing two-stage least-square estimations of Eqs. (2)

and (3). The results are reported in Table 7. We observe reductions in education, measures of socioeconomic status and occupational standing, and wage income. Birth-city lead status is associated with 0.13 lower years of schooling, a 28.2 % reduction in any college education, an 10.5 % reduction in the socioeconomic index, a 3.5 % reduction in occupational income score, a 12 % reduction in wage income, and a drop in income percentile of about 1.5 units. Finally, in column 7, we use World War II enlistment data and examine the effects on AGCT score as a proxy for cognitive and aptitude abilities. We find that birth-city lead status is associated with a 4.6 units lower AGCT score, off a mean of 75.5. This effect is equivalent to a reduction of about 6.1 % with respect to the mean.

Halpern-Manners et al. (2020) examine the effects of education on longevity and document that each additional year of schooling is associated with about 3.4 months of higher longevity. Therefore, the reduction in years of schooling observed in column 1 implies a drop in longevity of about 0.5 months. This is only 4.6 % of the overall effects of lead status and longevity in column 4 of Table 4. Fletcher and Noghanibehambari (2023) document that college education is associated with about 1.3–2.7 years of higher longevity. Based on column 2 of Table 7 and their estimates, one can calculate that birth-city lead status is associated with 4.3–9.1 months lower longevity. This is about 44–93 % of the observed effects in Table 4. Therefore, although reductions in human capital are a likely mechanism, decreases in attaining higher levels of education are possibly a more important channel of impact.

Chetty et al. (2016) examine the association between income and longevity using the universe of death records and tax returns in the U.S. and document that each additional income percentile is associated with about 1.7–2.2 months higher longevity. Therefore, the coefficient of column 6 in Table 7 implies 2.5–3.3 months increases in longevity. This is about 26–34 % of the observed reduced form effects of column 6 of Table 4.

7. Conclusion

Despite considerable efforts to improve water quality, many Americans are still at risk of lead in their drinking water. This is primarily due to materials used in water system pipelines. There are estimates that between 10–13 million service lines are based on leaded materials (Cornwell et al., 2016). Roughly half of the country's drinking water contains lead levels above the standard thresholds set by the American Academy of Pediatrics (NRDC, 2021). With aging water pipes, the dissolution of lead and water contamination has become a public health threat. Therefore, it is of policy relevance to examine the full costs of lead exposure, specifically among vulnerable populations.

In this paper, we explored the long-lasting impacts of exposure to lead in water pipes on later-life longevity. Overall, we documented the negative influences of lead exposure on later-life old-age longevity. The fact that the negative associations are larger in cities with acidic water and older pipeline systems contributes to our claim that reductions in longevity are driven by lead exposure in water systems. These estimates suggest that birth city lead status is associated with reductions in longevity of about 1.6–1.8 months. Further, using the sample of migrants, we found a substantially larger negative influence of lead status of birth-city rather than city of residence in later life, implying the relevance of exposure during earlier years of life. Our preferred estimation strategy relied on two-stage least square estimations that exploited proximity to lead refineries which lowers the cost of lead transportation as the instrument. We found that birth-city lead status is associated with roughly 9.6 months lower longevity. This effect is quite robust across alternative specifications and functional forms. Further, we provided empirical evidence to show that a significant portion of these reductions in later life longevity are driven by reductions in education, specifically higher educational levels.

In the U.S., male life expectancy increased from 46.3 in 1900 to 60.8 in 1940 (O'Neill, 2021). The negative intent-to-treat effects of full exposure to lead in drinking water offsets about 5.5 % of the overall health benefits that resulted in rises in life expectancy across cohorts of 1900–1940.[3] In the final sample, cohorts who were born in lead cities counted as 783,483 individuals. Using the estimated effects of the paper across different identification strategies, we calculate roughly 626.8 thousand life-years lost due to the use of lead in water pipes in the early part of the 20th century. We can monetize this value by incorporating the Value of Statistical Life (VSL) estimates. Some studies suggest using a VSL of about $10 million (in 2020 dollars) (Kniesner and Viscusi, 2019). The average longevity in the final sample is 72.9 years. A simple calculation suggests each life year's value is around $137.2 thousand. Using the estimated life years lost in the value of each statistical life, we can estimate a loss of roughly $85.9 billion (in 2020 dollars) due to reductions in longevity as a result of exposure to lead in earlier years of life. We should note that this effect does not capture the life years lost due to fetal deaths, infant mortality, and premature mortality as a result of the early-life lead burden (Clay et al., 2014; Roy and Edwards, 2021; Troesken, 2008).

Although we used lead service lines as the measure of exposure, we should note that significant efforts have been made to lower population-level lead exposure, such as the Safe Drinking Water Act of 1974 and the Lead and Copper Rule of 1991. There are estimates that the efforts since 1970 resulted in a reduction of about 94 % in blood lead levels across the U.S. population aged 1 to 74 (Dignam et al., 2019). Through these efforts, a substantial portion of net service lines have been replaced. The estimates, however, suggest that between 15 and 22 million people are still using lead-containing service lines in the U.S. (Cornwell et al., 2016).

CRediT authorship contribution statement

Jason Fletcher: Writing – review & editing, Supervision, Funding acquisition, Conceptualization. **Hamid Noghanibehambari:** Writing – original draft, Formal analysis.

[3] In this section, we use the coefficient of column 6 Table 4, i.e., 9.7 months.

Table 7
Two-stage least square estimation results to examine mechanism channels.

	Outcomes:						
	Years of schooling (1)	Any college education (2)	Socioeconomic index (3)	Occupational income score (4)	Log wage income (5)	Income percentile (6)	AGCT score (7)
Lead	−0.133***	−0.049***	−3.639***	−0.939***	−0.12***	−1.501***	−4.621***
	(0.042)	(0.004)	(0.247)	(0.088)	(0.009)	(0.251)	(1.692)
Observations	1204,331	1224,343	1062,210	1084,501	945,083	1160,296	21,894
R-Squared	.144	.08	.137	.107	.291	.188	.278
Mean DV	10.662	0.173	34.451	26.821	6.787	59.972	75.467
%Change	−1.248	−28.197	−10.562	−3.501	−1.769	−2.503	−6.123
First-Stage F-Stat	749.976	755.672	746.940	751.464	737.970	759.641	411.844

Notes. Standard errors, clustered on birth-city and birth-year, are reported in parentheses. All regressions include birth-state by birth-year fixed effects and individual/family/city controls. Controls include dummies for race and ethnicity, dummies for paternal socioeconomic index, dummies for maternal literacy, and birth-city covariates, including average socioeconomic index, female labor force participation rate, share of married, literacy rate, average occupational prestigious score, urbanization rate, and share of females. The sample is restricted to individuals aged >18.
*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Data availability

Data will be made available on request.

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