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TOXIFIED TO THE BONE:
EARLY-LIFE AND CHILDHOOD EXPOSURE TO LEAD AND MEN'S OLD-AGE MORTALITY

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Toxified to the Bone: Early-Life and Childhood Exposure to Lead and Men's Old-Age Mortality
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ABSTRACT

Several strands of research document the life-cycle impacts of lead exposure during the critical period of children's development. Yet little is known about long-run effects of lead exposure during early-life on old-age mortality outcomes. This study exploits the staggered installation of water systems across 761 cities in the US over the first decades of the 20th century combined with cross-city differences in materials used in water pipelines to identify lead and non-lead cities. An event-study analysis suggests that the impacts are more concentrated on children exposed during in-utero up to age 10. The results of difference-in-difference analysis suggests an intent-to-treat effect of 2.7 months reduction in old-age longevity for fully exposed cohorts. A heterogeneity analysis reveals effects that are 3.5 and 2 times larger among the nonwhite subpopulation and low socioeconomic status families, respectively. We also find reductions in education and socioeconomic standing during early adulthood as candidate mechanism. Finally, we employ WWII enlistment data and observe reductions in height-for-age among lead-exposed cohorts.

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1. Introduction

Following the industrial revolution of late 19th and early 20th century, 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 initiated installing 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 & Currie, 2019; Billings & Schnepel, 2018; Dave & Yang, 2022; Feigenbaum & Muller, 2016; Wodtke et al., 2022). Based on the World Health Organization's recent reports, about 30 percent of the global burden of idiopathic intellectual disability among children and about 4.6 percent of 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 & Edwards, 2021), higher infant mortality rates (Troesken, 2008), and adverse birth outcomes (Bui et al., 2022; Dave & 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), IQ (Nevin, 2000), elevated blood pressure (Farzan et al., 2018), kidney functioning (Skröder et al., 2016), crime (Feigenbaum & Muller, 2016; Reyes, 2007), educational outcomes (Miranda et al., 2007; Sorensen et al., 2019), and old-age Alzheimer's disease (Eid et al., 2016).

Despite the relatively extensive literature on health impacts of lead, little is known about the long-run effects of early-life lead exposure on old-age longevity. This paper aims to fill this gap in the literature. In so doing, we exploit the establishment of lead water pipe systems in US cities during the first decades of the 20^{th} century as a source of exposure to lead through contaminated water. Lead may contaminate water through erosions, dissolving, and certain chemical reactions with minerals carried by water. The leaded water is odorless, tasteless, colorless, and even some standard protocols of detection underestimate the true levels of contaminations (Triantafyllidou et al., 2007). We take advantage of the staggered adoption of water pipe system installations across 761 American cities between 1900-1930 combined with cross-city differences in the materials employed in water pipes. We then explore the longevity consequences among individuals who were exposed to lead-contained water pipes during their in-utero, early-life, and childhood using Social Security Administration (SSA) death records over the years 1975-2005. We find an intent-to-treat effect of 2.7 months reduction in longevity. We implement an event-study analysis and show that while the negative effects are mostly concentrated among children aged 0-12, the impacts are considerably larger for in-utero and prenatal exposures. We also find larger effects among nonwhites and those with low socioeconomic status fathers and well as larger impacts for those in cities with highly acidic or alkaline water source, a condition that facilitates lead leaching into drinking water. We are able to explore a sub-set of potential mechanisms, we find reductions in schooling and height-for-age in young adulthood.

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 exposure to lead and old-age longevity. 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). Further, understanding the long-run costs of lead exposure is important as it justifies the relatively large social costs of interventions (Pfadenhauer et al., 2016). Although the harmful impacts of lead have been known for over a century, the evidence of its long-run effects is limited. Moreover, with an aging water pipe infrastructure in the US, many cities face elevated risk of lead-in-drinking water (Allaire et al., 2018). This has been evident in the case of the recent water crises in Flint and Newark that resulted in lead leaks in urban drinking water (Dave & Yang, 2022; Grossman & Slusky, 2019). Policy concerns about this problem can also be observed in recent expansionary policies of the government. About 1.5 percent of the $1 trillion of the infrastructure bill that was passed on November 2021 had been allocated to replacing lead pipes in the water system. Second, 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 & Gorman, 2004; Karas Montez et al., 2014; Lindeboom et al., 2010; Montez & Hayward, 2011; Noghanibehambari & Fletcher, 2021; Smith et al., 2009; Van Den Berg et al., 2006, 2011). Third, this study contributes to the small literature that examines the impacts of public health interventions in the early 20th century on economic and health outcomes. These studies focus on public health efforts such as water filtration, water chlorination, treating sewage, setting bacteriological standard for milk, vaccination campaigns, and tuberculosis movement (Anderson et al., 2019; Anderson, Charles, & Rees, 2022; Anderson, Charles, McKelligott, et al., 2022; D. Cutler & Miller, 2005). This strand of study usually focuses on short-term outcomes and finds mixed evidence. This study extends this line of research by exploring the effects on longevity, an outcome that is measured many decades after the programs' implementations.

The rest of the paper is organized as follows. Section 2.2 reviews the background and the relevant literature. Section 3 introduces data sources. Section 4 discusses the econometric method. Section 5 addresses several endogeneity concerns. Section 6 reviews the results. In section 7, we discuss the economic significance of the results. Finally, we outline some concluding remarks in section 8.

2. Background

2.1. Water Projects

In the early decades of the 20th 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 that aimed to address various challenges related to water supply and water quality. This wave of water projects was driven by a growing population, urbanization, and the need for better management of water resources. These public health initiatives encompassed a range of measures, such as water filtration, water chlorination, and the establishment of sewage treatment facilities. These water projects implemented various techniques and technologies depending on preexisting local water quality and required scope of treatment to deliver clean water. For instance, Albany, NY implemented double filtration with the Slow Sand Filtration Plant. For this method, the collected raw water was first allowed to settle in large basins for large sediments to settle at the bottom. In the next step, the settled water would pass through sand beds which operated as a natural filter, excluding bacteria and other suspended solids from the water. This plant came into effect in 1899. Later, starting in 1909, water was further treated with calcium hypochlorite, and beginning in year 1916, with liquid chlorine (Logsdon & Lippy, 1982). In Cincinnati, OH, water filtration was implemented using a Rapid Sand Filtration technique (a water purification technique that involves passing water through a bed of sand to remove impurities and particles quickly) in 1907. It then was complemented with water chlorination in 1915.[4]

During these decades, the country saw a diverse array of 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 across the country. 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 its features. Other reasons were their durability, availability, and corrosion resistance. In many cities, an alloy of elements including lead and iron were 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.

[4] We assume the year of first treatment as the year of water project. This assumption is valid as the primary treatments were water filtration with relatively large gains for residents (Logsdon & Lippy, 1982). The following treatments brought additional benefits on top of the initial treatment gains.

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[5].

Medical studies suggest that pollution exposures during pregnancy change epigenetic programming which results in distorted growth path of the fetus (Almond & 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 in a way that increases infants' life-cycle susceptibility to diseases. Dave & 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-point more likely to give birth to low birth weight infants, an increase of 18 percent 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 grams additional birth weight. Wang et al. (2017) examine the association between maternal cord blood lead levels and birth outcomes. They find negative

[5] A broader literature, that we only briefly note, documents the relationship between exposure to other sources of airborne and waterborne pollution and a wide range of short-run and long-run outcomes, including infants' health outcomes, human capital formation, labor market outcomes, and adulthood health (Beach et al., 2016; Brainerd & Menon, 2014; Chay & Greenstone, 2003; Currie et al., 2013, 2014; Ebenstein et al., 2015; Greenstone & Hanna, 2014; Grossman & Slusky, 2019; Jones, 2019; Mettetal, 2019; Sanders, 2012; Smith et al., 2006, 2011, 2012; Zhang & 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 percent of a standard-deviation rise in high school test scores. Fletcher & Noghanibehambari (2022) 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 months reduction in longevity

impacts for 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 & Rosenzweig, 2004; Black et al., 2007; Flouris et al., 2009; Maruyama & 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 US 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 & Schnepel (2018) explore the effects of public health interventions among children with high levels of lead in their blood sample on their outcomes. They find that the negative impacts on education and test scores can be eliminated by interventions such as lead remediation, nutritional assessment, and medical evaluations. 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 percent 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 percent 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 & Frisvold, 2014, 2015; Fletcher & Marksteiner, 2017; Fletcher & Noghanibehambari, 2021; Lleras-Muney, 2022; Lleras-Muney et al., 2020; Lleras-Muney, 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 percent of lead is stored in bones (Rosin, 2009). Given the fact that bone development is disproportionately concentrated during early-life and early 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 blood stream. Therefore, individuals become internally exposed to lead load. Lee et al. (2022) use data from 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 materials of water pipes as the source of lead exposure. They find significant effects on later-life cognition but no effect of 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 a longitudinal data from Bangladesh to assess the association between prenatal lead burden and children 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) use data from the early 20th century US and shows that areas with lead water pipe system revealed 25-50 percent higher infant mortality rates compared with areas with non-lead water pipes. Hollingsworth & 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 in 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 county/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 study, 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.[6] In contrast, our analysis sample contains millions of observations which allows 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.

We extract our city-level water system from replication data of Feigenbaum & Muller (2016). The data reports the year of water system construction for 761 cities in 425 counties. It also adds information about primary materials used for each city/county water pipes. In order to merge water system data with DMF records, we need to infer city/county-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 the Census Linking Project (Abramitzky et al., 2020) to merge the DMF-census-linked data with historical census 1900, 1910, 1920, and 1930. Including the city/county information in 1940, we have at least 1 and at most five city/county identifier for each individual. For instance, for a person born in 1912, we potentially can observe their census city/county in 1920, 1930, and 1940. In case 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-county that is observed for each individual to use as a proxy for city-county of birth and childhood.[7] We then merge DMF with water system

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

[7] If exposure to lead correlates with migration decisions, the measurement error resulting from mis-assignment of individuals due to cross-census linking selection adds bias into our regressions. In Appendix B, we empirically investigate this concern. We examine the association between age-at-earliest-observed-census and our exposure measures and find that exposed individuals are observed at relatively younger ages. We argue that this can be translated into less accuracy for unexposed cohorts, who might have been exposed and migrated out of their city. Mis-assignment leads to considering these individuals as control while they should be treated, hence adding downward bias into our regressions. Furthermore, the 1940 census records state-of-birth. In our final sample, our method is successful in correctly identifying birth state for about 93.1 percent of individuals.

database based on inferred city-county-of-birth. We consider a water system to use lead if there is lead either as the primary material or in combination with any other products.[8] Finally, since water system construction occurred mostly in the first decades of the 20th century, we restrict the sample to birth cohorts of 1880-1930. 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 anthropometric outcomes reported by enlistment agencies. We specifically use information on height of enlistees as an alternative health outcome measured during early adulthood ages. Height is measured at the time of enlistment. In this sample, we focus on cohorts of 1900-1920 to remove outliers due to mismeasurement of age. We also compute and employ height-for-age to account for age differences in height measure at time of enlistment.

Figure 1 depicts lead versus non-lead city/counties in the final sample. Figure 3 depicts the evolution of exposure to waterwork across cohorts in lead and non-lead cities. Table 1 provides summary statistics of the final sample for cities with 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 in lead and non-lead cities live, on average, 891.4 (74.2) and 881.2 (73.4) months (years). Figure 2 illustrates a density distribution of age-at-death in the final sample.

About 97 (96) percent on individuals in our data born in lead cities (non-lead cities) are white. Roughly 11.6 and 4.9 percent of the observations in the lead and non-lead cities are exposed to waterwork installations before age 12. Both lead and non-lead cities have similar measured literacy rates.

[8] For instance, we consider pipes to be leaded if they contain the following combinations: iron and lead, wrought iron and lead, galvanized iron and lead, galvanized iron with lead connection, etc.

4. Econometric Method

We take advantage of two sources of variations. First, the staggered adoption of water system construction across cities and over time. Second, the cross-city differences in water pipe materials, i.e., lead versus non-lead cities. Therefore, we start by comparing the longevity of individuals who were exposed to water system construction projects at different ages in lead versus non-lead cities. Specifically, we implement event study analyses of the following form:

$$DA_{icrt} = \alpha + Lead_c \times \sum_{k=-\underline{T}}^{\overline{T}} \zeta_k I(t_c^* - t = k) + \beta X_i + \theta_{rt} + \lambda_c \times T_t + \varepsilon_{icrt} \quad (1)$$

Where the outcome is age at death of individual i born in city-county c in census-region r and year t. The variable $Lead$ is a dummy that equals one if the individual is born in a lead city and zero otherwise. The parameters ζ represents event-time coefficients. The function $I(.)$ equals one if the argument is true. The argument measures the difference in the city-specific year of water system construction (t_c^*) and birth-year (t), i.e., it calculates the age of individuals at which the waterwork construction started. Since individuals in all age groups could be affected by lead burden, we prefer to compare across age groups rather than specifying a cut-off point. However, we split exposure ages based on a common (and arbitrary) threshold (l) below which effects are primarily concentrated. The literature suggest considerable and long-lasting effects for exposure during the early years of life and childhood (CDC, 2022; Grönqvist et al., 2020; Hornung et al., 2009). Therefore, we set the coefficients of age-at-exposure of 11-12 to be the omitted group. As we will observe in the event-study results, the effects start to appear for age-at-exposure of less than 14 and becomes significant for age-at-exposure of less than 10. Therefore, the omitted group is less likely to be affected by the lead in the water system. Moreover, to have more observations in each event-time, we group event-times into two-year increments.

In X, we include a race dummy as individual covariate and a series of parental dummies for parental controls. These controls include dummies for maternal literacy, paternal socioeconomic status, and a missing indicator for the missing values. The parameter θ represents birth-region-by-birth-year fixed effects that absorb cohort convergence in health outcome across different census regions and all other time-varying region-specific shocks (Goodman-Bacon, 2021b). The county fixed effects (λ) absorb all time-invariant county characteristics. In our preferred specifications, we also include county-specific linear trend in birth year to absorb all county characteristics that evolve linearly over cohorts. Finally, ε is a disturbance term. We cluster standard errors at the city/county of birth level. The regressions are weighted using mean of city-county population over the sample period.

We also implement difference-in-difference regressions in which the primary variable of interest is the share of childhood years (up to age 12) that the individual could have been exposed to post-construction period in lead cities.[9] Specifically, we estimate the following regressions:

$$DA_{icrt} = \alpha + \phi Lead_c \times Exp_{ct} + \varphi Exp_{ct} + \beta X_i + \theta_{rt} + \lambda_c \times T_t + \varepsilon_{icrt} \qquad (2)$$

The variable Exp is the share of childhood exposure to post-water-system-construction. For those who were born seven-and-more years prior to water system, Exp takes a value of zero, assuming minimal impact for older cohorts. For those who were born after the water system initiation, the variable takes a value of one, suggesting a full exposure from prenatal though childhood. For other cohorts, it is calculated as the year they turn seven minus the year of water project, divided by seven. Thus, the primary parameter of interest is ϕ that captures the longevity

[9] We use the empirical analysis event-study of section 6.1 to determine those cohorts for whom the effects start to appear. We then use those exposure ages to build our main independent variable in our difference-in-difference analysis. In Appendix A, we show that the effects are considerably larger when we focus on in-utero exposure rather than childhood exposures.

differential of those who were born in lead cities versus those born in non-lead cities and experienced a full exposure to water construction during their childhood versus those with zero exposure. All other parameters are as in equation 1.

5. Concerns over Endogeneity

5.1. Endogenous Evolution in City-Level Characteristics

One concern in interpreting our results is that local authorities may respond to conditions of cities and counties in their decision to initiate a water project. For instance, an increase in inflow of migrants or rises in fertility that results in a higher population may elevate the social and political debates about public health infrastructures and possible social burden of poor water quality. As we discussed in section 3 and suggested by Feigenbaum & Muller (2016), lead cities were generally wealthier than non-lead cities. The endogenous decisions of local authorities could be different across these two types of cities in unobserved ways.

Another concern is the potential for differential trends in socioeconomic standing and educational levels in lead and non-lead cities that results in differences in local tax collection which is used to finance water system projects. These differential trends my not be differenced-out in our difference-in-difference framework or captured by the fixed effects and trends in our model as they are correlated with differential paths of unobservables. We empirically address this concern by regressing city/county characteristics on the primary measures of water projects as in equation 1. In so doing, we use city/county level characteristics extracted from full-count censuses 1880-1940. We replace cohort (t) with census year in equation 1. We then regress those characteristics on event-time dummies, fixed effects, and trends. To ease interpretation and comparison of effects across outcomes, we standardized all outcomes with respect to mean and standard deviation of the sample. The results are illustrated in different panels of Figure 4 through Figure 6. We do not observe a consistent, robust, and evident pre-trend or post-trend in outcomes. Specifically, there is no discernible differences in lead versus non-lead cities in several years pre and post waterwork in population, share of different races, females (Figure 4), immigrants, married women, literacy rate, number of children (Figure 5), and various measures of socioeconomic score (Figure 6).

A further concern arises due to the aggregation of various lead exposures in specific areas. For instance, it could be the case that manufacturing workers with lead exposure carry lead-contaminated dust to their homes which expose children to lead. However, a simple cross-sectional correlation between lead status of cities and share of manufacturing employment reveals a small and negative association. Therefore, although this could be the case for further exposure to lead, it is not very likely to confound our estimates as we don't observe the co-movement with lead status. Further, it could also be the case that lead-based paint in homes and buildings exacerbate the situation (McFarland et al., 2022). However, the estimates suggest that close to 90% of all homes and buildings built prior to 1970s contained lead in their paints (EPA, 1995). Again, as it is not likely to co-move with our exposure and city-level lead measure and water project, they are not likely to confound our mortality regressions.

5.2. Balancing Tests

Another source of concern is the differential selection into sample based on observable and unobservable characteristics. For instance, if there are differences in whites versus nonwhites in their survival into adulthood (and hence DMF data) and if lead exposure affects survival of these two groups differently, our sample may contain more white people among treated groups and more nonwhites in our control group. Since whites have, on average, higher longevity for unobservable reasons, our lead-longevity estimates underestimate the true relationships. To empirically test the final sample's balance, we regress a series of observable individual and family characteristics on event-time dummies, fixed effects, and trends as in equation 1. We standardized all outcomes to ease cross-panel comparison of effects. The results are reported in Figure 7 through Figure 9. We do not observe any evident pattern of changes in nonwhite across age groups (top-panel of Figure 7). Besides, we do not observe a discernible trend for father's occupational income score (bottom-panel of Figure 7), father's socioeconomic score (top-panel of Figure 8), and maternal literacy (bottom-panel of Figure 8). However, we observe small increases on measures of socioeconomic score and maternal literacy for those who were born 6-7 years post-waterwork in lead versus non-lead cities. Childhood family socioeconomic status and parental education are correlated with higher longevity during old ages (Currie & Rossin-Slater, 2015; Huebener, 2019; Montez & Hayward, 2014; Savelyev et al., 2022). Therefore, to the extent that family socioeconomic status and education allocation of treated groups reveal an increasing trend post-waterwork, we expect that the effects underestimate the true effects. Besides, these effects do not appear across other age groups and do not provide a consistent pattern of selection based on parental characteristics.

5.3. Endogenous DMF-Census Merging

The link between census records and DMF data is primarily based on name commonalities, place-of-birth, and cohort. Hence, they are not characteristics-specific links. However, one may be concerned that the DMF-census links are correlated with city-level lead burden and so our estimations may reflect the underrepresentation of unmerged population (or vice versa). We empirically test this concern using the original population of the 1940. We implement the same method as described in section 3 to search for county-city of birth in historical censuses. We then merge this with records of our final sample. We create a new variable that equals one if merging between original population and final sample is successful and zero otherwise. We then regress this binary variable on measures of lead burden, fixed effects, and trends as in equation 2. We report the results in Table 2, adding more covariates in consecutive columns. In the fully parametrized model of column 3, we observe 15 basis-points reduction in the probability of merging for cohorts born in lead cities versus non-lead cities and an exposure of one versus zero. This change is equivalent to a reduction of about 1 percent relative to the mean of the outcome. This effect is economically small and statistically insignificant. Among non-lead cities, exposure to waterwork is associated with a statistically significant increase in the probability of successful merging. However, this effect is economically small and suggests a 3.7 percent change with respect to the mean of the outcome.

6. Results

6.1. Event-Study Results

The event study results of equation 1 are reported in the top-panel of Figure 10. Compared with cohorts who are 11-12 years old (i.e., per equation 1, k=[11,12]) at the time of waterwork installation, those older aged do not reveal differential longevity.[10] Moreover, we do not observe statistically significant differences in the effects across ages 5-6, 7-8, and 9-10. However, the event-time coefficients start to reveal a declining pattern for those less than 7 years old that becomes statistically significant for age-at-exposure of 3-4 years. We observe considerably larger reductions for those who were exposed during prenatal development (i.e., born in years following waterwork installation).

Since our design is partly dependent on staggered adoption of city-level water projects, one concern is the endogenous influence of OLS-produced coefficients (Callaway & Sant'Anna, 2021; Goodman-Bacon, 2021a; Sun & Abraham, 2021). To explore this concern, we use the estimation

[10] In these event studies, the coefficient for age-at-exposure of 11-12 are eliminated so that these cohorts serve as a contrast group. It should not be translated as the age in which treatment indicators turns on, as it is usually the case in event study analyses.

method developed by Sun & Abraham (2021) and report the event-study results in the bottom-panel of Figure 10. We observe a very similar pattern of effects as the OLS-produced coefficients.

To further validate the results, we implement a similar event study analysis in non-lead cities. Specifically, we remove the $lead$ variable in equation 1 and focus on the main effects of event-time coefficients. If the effects are not driven by pre-trend or other city-level factors that are correlated with the waterwork completion, we expect to observe a pattern in the results that, at the very least, do not point to reductions in longevity for early-childhood and in-utero exposures. The results are depicted in Figure 11 for OLS and Sun & Abraham (2021) estimates in the top and bottom panels, respectively. We do not detect a consistent and discernible pattern in coefficients. Moreover, almost all the coefficients are statistically insignificant. Further, in Figure 12, we show the effects in a subsample restricted to lead cities. We observe a quite similar pattern as those reported in Figure 10, which eliminates the concern that temporal changes in the control group (non-lead sample) may influence the overall difference-in-difference results.

6.2. Difference-in-Difference Results

The main results of the paper are reported in Table 3. The first column includes county and city-county fixed effects. We then add region-year-of-birth fixed effects (column 2), city-county trend (column 3), and individual and family controls (column 4). In the fully parametrized specification of column 4, we observe a reduction of 2.7 months of longevity for fully exposed cohorts (versus unexposed cohorts) in lead cities (versus non-lead cities). The main effect of exposure captures the impacts of water projects in non-lead cities. It suggests an insignificant effect of 0.23 months rise in longevity.

We can compare the magnitude of the effects with other early-life influences of later-life longevity. Halpern-Manners et al. (2020) examine the impact of education on longevity using SSA death records data and finds that each additional year of education is associated with roughly 4 months higher age-at-death. Therefore, being born in a lead city (full exposure) is equivalent to about 0.7 years lower educational attainments. Fletcher & Noghanibehambari (2021) examine the impact of college openings on college education and longevity. They document an increase of about 1 year in longevity as a result of college education. Therefore, being born in lead cities may offset about 23 percent of health benefits of college education. Chetty et al. (2016) examine the income-longevity relationship using matched SSA death records and individual tax records. They document that each additional income percentile (an increase of $8,000 from the sample mean, in 2020 dollars) is associated with about 1.6 months higher longevity. Therefore, being born in lead cities have the same effect of 1 percentile lower income during adulthood, roughly $13,500 drop from the median.

6.3. Robustness Checks

In Table 4, we explore the robustness of the main results to alternative specifications. To have a benchmark comparison, we replicate the results of fully parametrized model in column 1. In column 2, we interact county-of-residence in 1940 effects with county-of-birth effects to control for endogenous migration decisions and potential long-run influence of neighborhood choice (Derenoncourt, 2022). The estimated effects increase by about 11 percent compared to those of column 1.

In column 3, we add State-Economic-Area (SEA) of birth by year-of-birth fixed effects. SEA constitute a group of counties that are in a commuting zone and are economically interconnected. Therefore, we identify effects across SEAs in which at least one city is a lead-city and the other(s) is a non-lead-city. The identification variation comes from comparing longevity of individuals with differential exposure to water system change in lead versus non-lead cities who were born within the same SEA and year.[11] Hence, this specification controls for all unobserved confounding influences at the SEA-year level. We observe a reduction of 3.2 months for fully exposed cohorts in lead cities.

In column 4, we allow for the main effects of city-of-birth to vary across different sociodemographic groups by interacting county fixed effects with race and parental covariate dummies. The estimated effects are very similar to column 1. Several studies point to the influence of season-of-birth in life-cycle health outcomes (Flouris et al., 2009; Vaiserman, 2021; Vaiserman et al., 2002). We control for this potential confounder by adding birth-month dummies interacted with birth-year fixed effects. The results, reported in column 5, are only slightly smaller than the main results. There is also evidence of the seasonality patterns in mortality, specifically in relation to seasonality in temperature and pollution (Marti-Soler et al., 2014; Simmerman et al., 2009). In column 6, we control for this by adding death-month dummies. The estimated effects are almost identical to the main results.

One concern in our analysis is the truncated nature of DMF data. The data is truncated from left and right, making the sample prone to selection bias. We implement Heckman (1979) estimate to account for potential issues of truncation. Specifically, this method first estimates an equation in which the outcome is successful merging with the original cohorts of 1940 (as in section 5.3) as a function of observables and fixed effects. It then calculates an Inverse Mills ratio to account for selection into the final sample (from the original population) based on observables. Finally, this ratio is added to the regressions of longevity as an additional control. The results are reported in

[11] The final sample contains 281 SEAs. In 30 of these SEAs, we can identify the effects as there are variations by lead pipes across different cities within those SEAs. That counts to 907,604 observations that identify the effects of column 3.

column 7. The estimated effects are considerably larger than those of column 1, suggesting that truncation might lead to understated impacts.

Next, we explore the sensitivity to alternative functional forms. In column 8, we replace the outcome with the log of age-at-death. The interaction term suggests a drop of about 0.36 percent, quite similar to 0.3 percent decrease from the mean implied by column 1. Finally, we show that the results are robust and remain significant when we cluster standard errors at the city level and when we use two-way clustering at city-county and region-year level (columns 9 and 10, respectively).

6.4. Heterogeneity across Subsamples

In Table 5, we explore the heterogeneity of the results by interacting a nonwhite dummy and a dummy for father's socioeconomic score less than median with right-hand side variables. In column 1, the triple-interaction term suggests that the nonwhites in treated groups reveal 9.6 months lower longevity, almost 3.5 times the effect size on whites in row 9 (-1.4). Moreover, we observe a positive, statistically significant, and relatively large coefficient of exposure among nonwhites (8.6), suggesting potential benefits of water projects among this subpopulation. We also observe a larger impact among people whose fathers had lower than median socioeconomic score, of about 5.4 months. This evidence is in line with the literature that suggests larger impacts of lead burden among minorities and children of disadvantaged populations (Grönqvist et al., 2020; Hollingsworth et al., 2022; Wodtke et al., 2022). Moreover, we observe positive and significant increase in longevity of about 6.3 months for non-lead city exposure to water projects, suggesting long-term benefits of water infrastructure change and access to clean water.

Another source of heterogeneity is related to the solubility of lead in water. Lead solubility in water depends on water pH (Kim et al., 2011). A higher concentration of lead can be leached into the water system if water is highly acidic (pH≤6.5) or highly alkaline (pH≥8.5) (Ferrie et al., 2012; Lee et al., 2022). We use city-level pH data in 1940 reported by Lohr & Love (1954a) and Lohr & Love (1954b) to infer whether the water is acidic or alkaline. We interact a dummy indicating acidic/alkaline water with the right-hand side variables. We report the results in column 3. The triple interaction term suggests 15.6 months lower longevity among those in cities with acidic/alkaline water.

6.5. Candidate Mechanisms

Several strands of research suggest that early-life 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 & 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 & 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. Since our main sample covers cohorts of 1880-1930, several cohorts have not yet completed their education in 1940. To overcome this issue, we use censuses 1960 and 1970 to examine mechanism channels. We use city/county of observation in 1960 and 1970 as a proxy for city/county of birth. To mitigate migration bias, we limit the sample to individuals whose state-of-birth is the same as state-of-residence at the time of the census. We implement regressions similar to equation 2 and report the results in columns 1-3 of Table 6. We observe an increase in the probability of having less than high school education and less than 12 years of schooling by about 2.1 and 3.3 percentage-points, off a mean of 0.26 and 0.52, respectively (columns 1 and 2). Further, we observe a reduction of 0.5 units in occupational income score, off a mean of 29.8.

To further complement this section, we use data from World War II enlistment linked to DMF and explore the effects on anthropometric outcomes as measured and reported by enumerators. This data is extracted from Goldstein et al. (2021) and covers a fraction of people in our final sample. Before examining health outcomes, we explore the probability of being in the enlistment data as a function of exposure to lead during childhood. Column 4 in Table 6 suggests a 1.1 percentage-points reduction in the likelihood of being in the WWII enlistment data, off a mean of 0.13 (~8.7%), suggesting some scope for pre-enlistment impacts on health. Next, we examine the effects on health-related measures. Specifically, we focus on height as it is a strong predictor of other health measures, including mortality (Bozzoli et al., 2009; Crimmins & Finch, 2006; Deaton, 2007; Deaton & Arora, 2009; Spijker et al., 2012). The results suggest reductions in height. Fully exposed cohorts in lead cities reveal a reduction of about 0.48 inches in height, off a mean of 68 (column 4). To account for the influences of age in height, we also calculate height-for-age. We standardize the variable with respect to mean and standard deviation of the sample. We estimate that treated groups reveal a reduction of 5.1 percent of a standard-deviation of heigh-for-age (column 5).

7. Discussion

The results of this study provide intent-to-treat estimates across the whole population and suggest a lower bound of the true effects. This is more evident as we observe larger effects when we look at the population at higher risks such as nonwhites who are more likely to live in urban areas with a higher exposure to the new waterwork.

In the US, life expectancy increased from 39.4 in 1880 to 53.2 in 1930 (O'Neill, 2021). The negative intent-to-treat effects of a full exposure to lead in drinking water during childhood offsets about 1.6 percent of the overall health benefits that resulted in rises in life expectancy across cohorts of 1880-1930. In the original 1940 census, cohorts who were born post-waterwork and in lead cities represent about 9.64 million people. Using the estimated effects of Table 3 for these cohorts, we calculate roughly 2.17 million 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 estimates of Value of Statistical Life (VSL). Some studies suggest using a VSL of about $10 million (in 2020 dollars) (Kniesner & Viscusi, 2019; Viscusi, 2018). Our final sample is based on individuals survived to age 45. The average life expectancy at age 45 in the US is roughly 34.5 years. Hence, the difference between the average longevity of treated cohorts and the average US life expectancy of survivors to age 45 is roughly 3.6 years. Based on these back-of-an-envelope calculations, we reach a Value of Statistical Life Year (VSLY) of about $3.05 million. Using this VSLY and the marginal effect of Table 3, we calculate a monetary equivalent loss of about $686K for each person.[12] Therefore, the loss in treated cohorts' life-years is equivalent to $6.6 trillion.[13] 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 early-life lead burden (Clay et al., 2014; Roy & Edwards, 2021; Troesken, 2008).

[12] This is calculated by interacting the VSLY (3.05 M) with the effect of main results in years (2.7 ÷ 12)

[13] We quantify this using the calculation of VSLY based on VSL extracted from Colmer (2020). The average US life expectancy conditional on survival up to 45 is 79.5 years. The average longevity of treated cohorts is 76 years. We use this difference, an arbitrary but common discount rate of 3 percent, and a VSL of $10 million in the following formula: $VSLY = \frac{rVSL}{1-(1+r)^{-L}}$, where r is the discount rate and L remaining life years of average individual in the sample. To reach the final cost estimation, we use the marginal effect of Table 3 (3.05 months), total number of treated cohorts (9.64 million), and the estimated VSLY ($2.975 M), as follows: $\left(\frac{2.7}{12}\right) \times 9.64M \times 3.05M = 6.6T$

8. Conclusion

Despite considerable efforts in improving 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. Between 1900-1950, many American cities installed water systems from pipes that contained lead. Some cities even mandated the use of lead due to its durability. 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 contain lead levels above the standard thresholds set by the American Academy of Pediatrics (NRDC, 2021). With aging water pipes, 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 the vulnerable populations.

In this paper, we explored the long-lasting impacts of lead in water pipes on longevity. We exploited the staggered adoption and installation of water systems across US cities combined with the differences in pipeline materials to identify exposed cohorts in cities with lead in their water pipes. We examined the effects of early-life and childhood exposure to lead in water on old-age longevity using Social Security Administration death records linked with the full-count 1940-census. We found intent-to-treat reductions of about 2.7 months in old-age longevity. We showed that the effects are larger among nonwhites and those in lower socioeconomic status families. We provided evidence that reductions in educational attainments and early adulthood occupational income scores are likely mechanisms. Moreover, we used World War II enlistment data and found reductions in height-for-age, an important predictor of later-life general health.

Although we used lead service lines is the measure of exposure, we should note that significant efforts have been made to lower population-level lead exposure, such as 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 level across the US 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 to 22 million people are still using lead-containing service lines in the US (Cornwell et al., 2016).

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Tables

Table 1 - Summary Statistics

	Lead Cities		Non-Lead Cities	
	Mean	SD	Mean	SD
DMF-Census Data:				
Death Age (Months)	912.0818	125.4844	909.9363	126.3487
Birth Year	1912.0573	10.4409	1912.3983	10.5828
Death Year	1988.0733	8.626	1988.2368	8.6753
White	.9678	.1764	.9588	.1986
Black	.0305	.1721	.0373	.1895
Other	.0016	.0402	.0038	.0619
Hispanic	.0092	.0955	.0162	.1264
Mother Education < HS	.3391	.4734	.3	.4583
Mother Education = HS	.0914	.2882	.1235	.329
Mother Education > HS	.017	.1294	.0233	.1508
Mother Education Missing	.5525	.4972	.5532	.4972
Father SEI 1^{st} Quartile	.1151	.3192	.1034	.3045
Father SEI 2^{nd} Quartile	.1044	.3058	.1079	.3103
Father SEI 3^{rd} Quartile	.0995	.2994	.1016	.3021
Father SEI 4^{th} Quartile	.0789	.2696	.0836	.2767
Father SEI Missing	.6021	.4895	.6035	.4892
Exposure	.4518	.4977	.4096	.4918
Exposure × Lead	.2727	.3824	0	0
Acidic/Alkaline Water ($pH\leq 6.5$ or $pH\geq 8.5$)	.3696	.4827	.1529	.3599
Observations	2063657		353072	
Sample for Mechanism Analysis:				
Years of Schooling < 8	.2648	.4413	.2769	.4474
Years of Schooling < 12	.5173	.4997	.511	.4999
Occupational Income Score	29.7742	9.5442	30.0438	9.6473
Observations	312657		62423	
DMF-World War II Enlistment Data:				
Height (inch)	68.0169	3.2682	68.0677	3.2953
Height-for-Age (Standardized)	0	.998	.0002	1.0123
Observations	253047		41391	

Table 2 - Exploring Potential Endogenous Merging

	Outcome: successful merging between original 1940 population and the final sample		
	(1)	(2)	(3)
Exposure×Lead	-.01322***	-.00343***	.0015
	(.00184)	(.00118)	(.00153)
Exposure	.00574***	.00564***	.00411***
	(.00134)	(.00094)	(.00103)
Observations	23858091	23858091	23858091
R-Squared	.01482	.02276	.02298
Mean DV	0.107	0.107	0.107
County-City FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Region-Year of Birth FE		✓	✓
County-City by Birth-Year Trend			✓

Notes. Standard errors, clustered on county-city, are in parentheses. Regressions are weighted using county-city-level population. The data covers birth cohorts of 1880-1930.
*** p<0.01, ** p<0.05, * p<0.1

Table 3 - Main Results

	Outcome: Age at Death (Months)			
	(1)	(2)	(3)	(4)
Exposure×Lead	-1.56141**	-2.88429***	-2.73644***	-2.66289***
	(.76432)	(.72739)	(.9744)	(.9717)
Exposure	-.85409	-.66274	.22672	.23613
	(.58404)	(.58087)	(.61462)	(.6126)
Observations	2416729	2416729	2416729	2416729
R-Squared	.39543	.39552	.39566	.39587
Mean DV	890.906	890.906	890.906	890.906
County-City FE	✓	✓	✓	✓
Birth Year FE	✓	✓	✓	✓
Region-Year of Birth FE		✓	✓	✓
County-City by Birth-Year Trend			✓	✓
Family Controls				✓

Notes. Standard errors, clustered on county, are in parentheses. Regressions are weighted using county-level population. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.
*** p<0.01, ** p<0.05, * p<0.1

Table 4 - Robustness Checks

	Column 5 Table 3	Adding 1940-County by Birth-County FE	Adding State-Economic-Area by Birth-Year FE	Adding City by Individual/Family Dummies	Adding Birth-Month-by-Birth-Year FE
	(1)	(2)	(3)	(4)	(5)
Exposure×Lead	-2.66289***	-2.95403***	-3.13963**	-2.62118***	-2.57956***
	(.9717)	(.96663)	(1.35625)	(.98016)	(.96907)
Exposure	.23613	.47379	1.25375	.23349	.21238
	(.6126)	(.62393)	(.92077)	(.61458)	(.61265)
Observations	2416729	2383407	2416729	2416645	2416729
R-Squared	.39587	.3993	.3964	.39597	.3965
Mean DV	890.906	890.771	890.906	890.907	890.906
	Adding Death-Month FE	(Heckman, 1979) Estimate	Outcome: Log Age at Death	SE Clustered at the City Level	Two-Way Clustering at County-City and Region-Year Level
	(6)	(7)	(8)	(9)	(10)
Exposure×Lead	-2.67721***	-3.78227***	-.0036***	-2.66289**	-2.66289***
	(.97318)	(1.08979)	(.00121)	(1.10074)	(.9717)
Exposure	.26448	0.96684	.00036	.23613	.23613
	(.61208)	(0.72974)	(.00071)	(.75753)	(.6126)
Observations	2416729	23,858,091	2416729	2416729	2416729
R-Squared	.39619	----	.38812	.39587	.39587
Mean DV	890.906	890.906	6.782	890.906	890.906

Notes. Standard errors, clustered on county (except for column 10), are in parentheses. Regressions are weighted using county-level population. All regressions include county-city FE, county-city trend, region-year-of-birth FE, individual controls, and family covariates. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

*** p<0.01, ** p<0.05, * p<0.1

Table 5 - Heterogeneity of the Results across Sociodemographic Groups

	Outcome: Age at Death (Months)		
	(1)	(2)	(3)
Exposure×Lead×Nonwhites	-9.58796*		
	(5.14915)		
Exposure×Nonwhites	8.62077*		
	(4.68018)		
Nonwhites	-7.0569***		
	(.79992)		
Exposure×Lead×Father SEI<Median		-5.4839*	
		(3.16585)	
Exposure× Father SEI<Median		6.3033**	
		(3.05695)	
Father SEI<Median		-5.93842***	
		(.41762)	
Exposure×Lead×Acidic/Alkaline Water			-15.62797***
			(5.81613)
Exposure× Acidic/Alkaline Water			11.70862**
			(5.92459)
Exposure×Lead	-2.65199***	-2.64711***	-5.47069
	(.97188)	(.97196)	(3.51398)
Exposure	.24948	.29297	3.89759*
	(.61282)	(.61438)	(2.27357)
Observations	2416729	2416729	834105
R-Squared	.39587	.39587	.39234

Notes. Standard errors, clustered on county-city, are in parentheses. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, region-year-of-birth FE, individual controls, and family covariates. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.
*** p<0.01, ** p<0.05, * p<0.1

Table 6 - Potential Mechanisms and Mediatory Outcomes

	Outcomes:					
	Years of Schooling < High School	Years of Schooling < 12	Occupational Income Score	Being in the Enlistment Data	Height	Height-for-Age (STD)
	(1)	(2)	(3)	(4)	(5)	(6)
Exposure×Lead	.02131*	.03375**	-.49433**	-.01096*	-.48845**	-.05147**
	(.01215)	(.01452)	(.21914)	(.00649)	(.24834)	(.02068)
Exposure	-.00866	-.01325	.32602*	-.00728*	-.00443	-.00033
	(.00913)	(.0115)	(.16697)	(.00392)	(.04429)	(.00372)
Observations	375080	375080	360617	2416729	294438	294438
R-Squared	.12336	.09038	.05979	.1154	.03639	.92186
Mean DV	0.267	0.516	29.819	0.125	67.923	0.072

Notes. Standard errors, clustered on county-city, are in parentheses. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, region-year-of-birth FE, individual controls, and family covariates. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data for columns 1-3 covers birth cohorts of 1880-1930 observed in 1960-1970 censuses. The data for columns 4-5 covers birth cohorts of 1900-1920 who died between 1975-2005.
*** p<0.01, ** p<0.05, * p<0.1

Figures

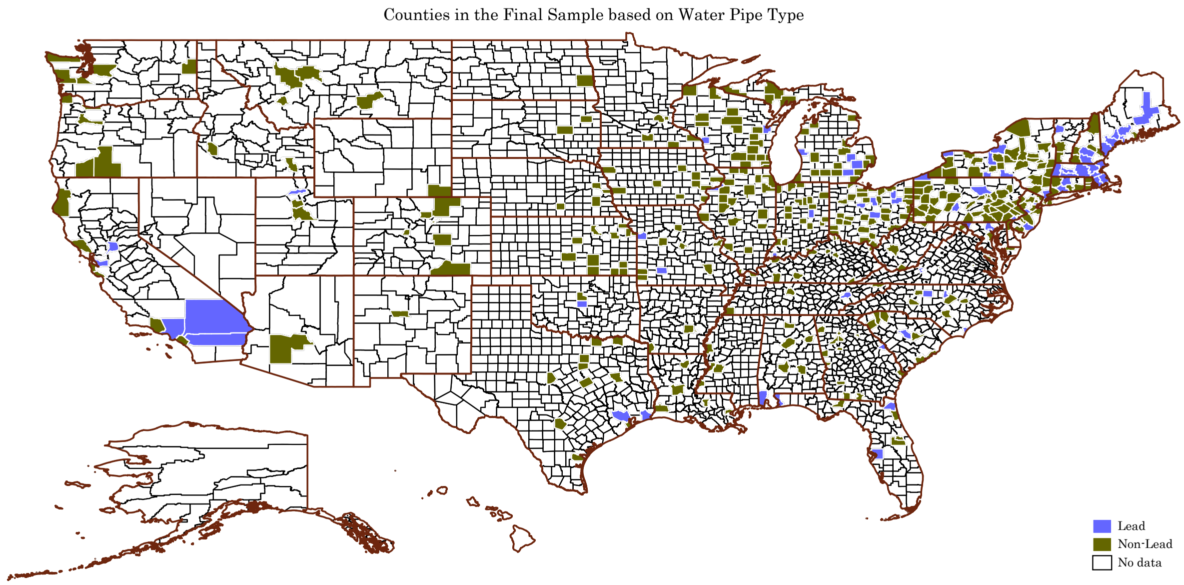

Figure 1 - Distribution of Counties in the Final Sample

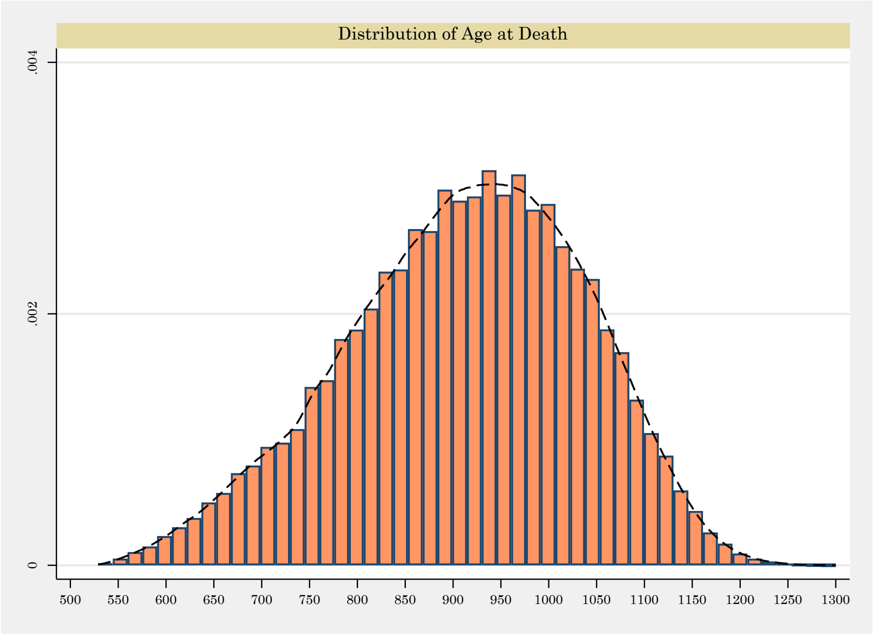

Figure 2 - Distribution of Age at Death (Months) in the Final Sample

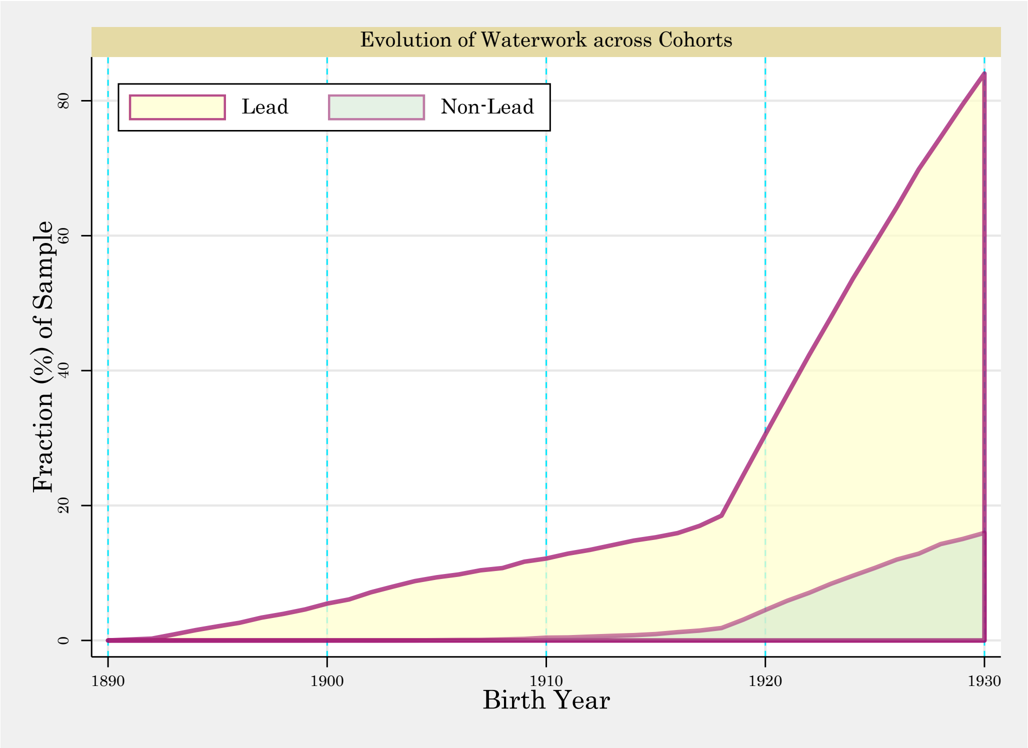

Figure 3 - Evolution of Exposure to Waterwork in Lead and Non-Lead Cities

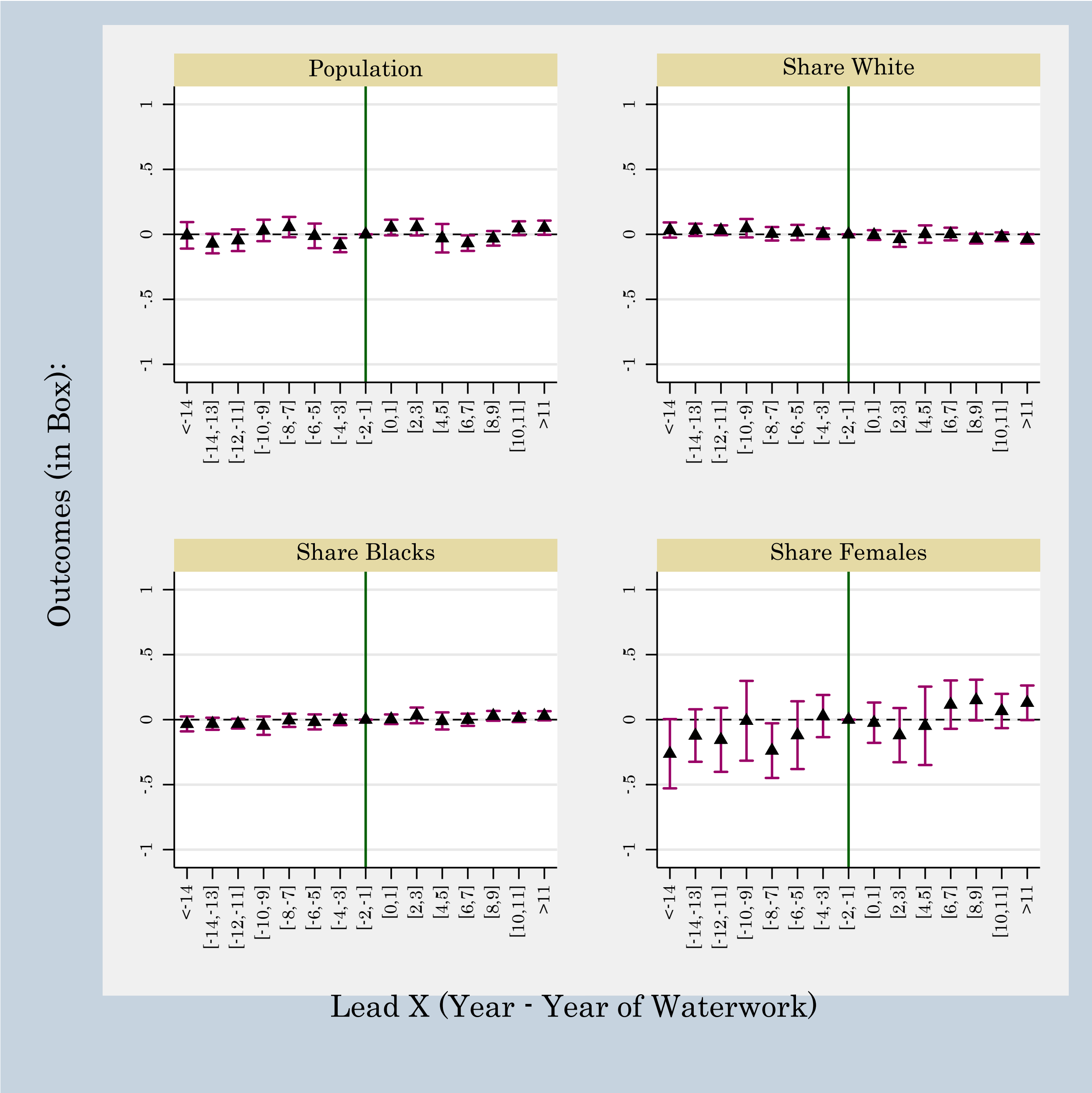

Figure 4 - Lead Pipe Installation and Changes in County-City Characteristics

Notes. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, and region-year-of-birth FE. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

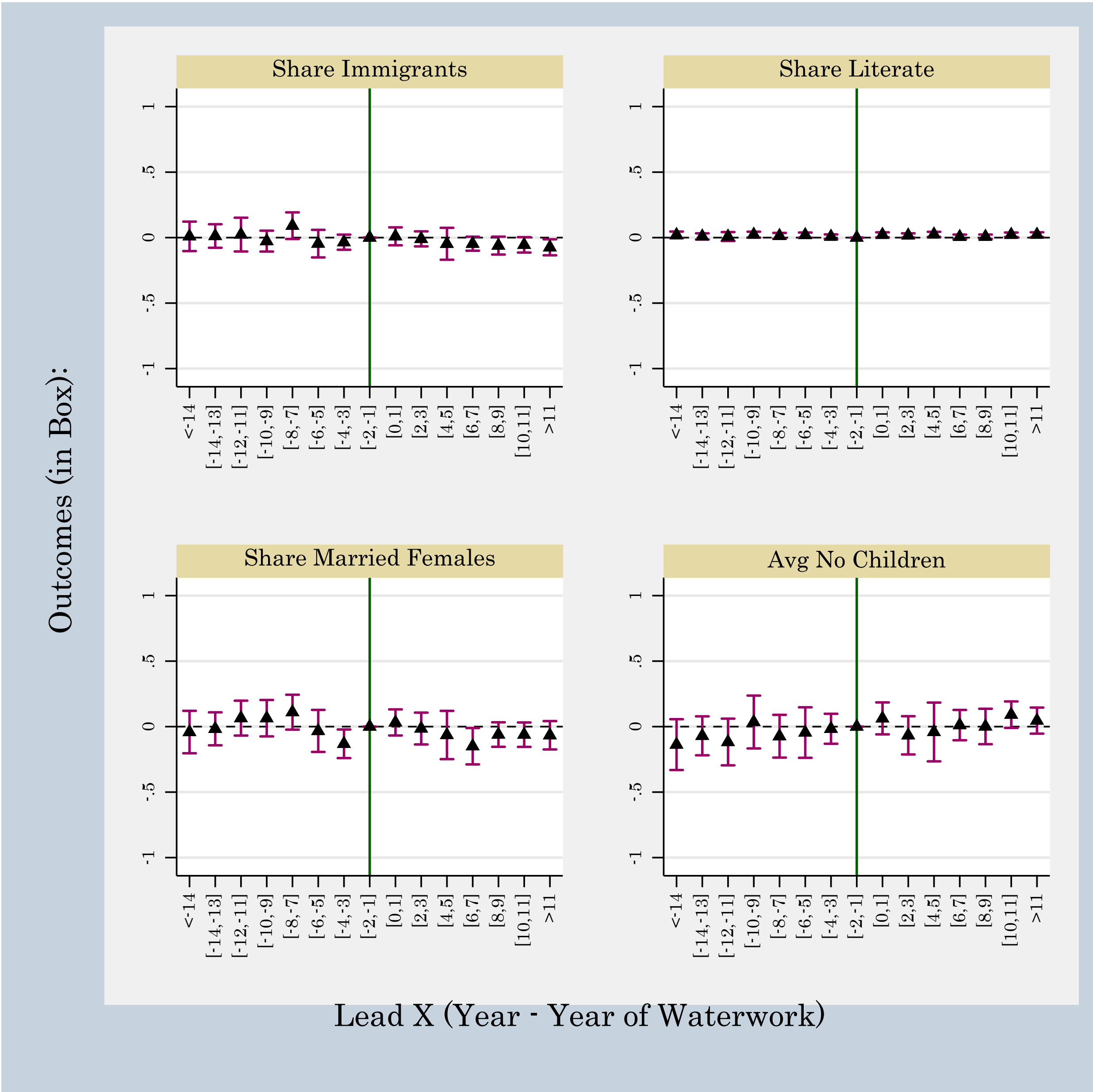

Figure 5 - Lead Pipe Installation and Changes in County-City Characteristics

Notes. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, and region-year-of-birth FE. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

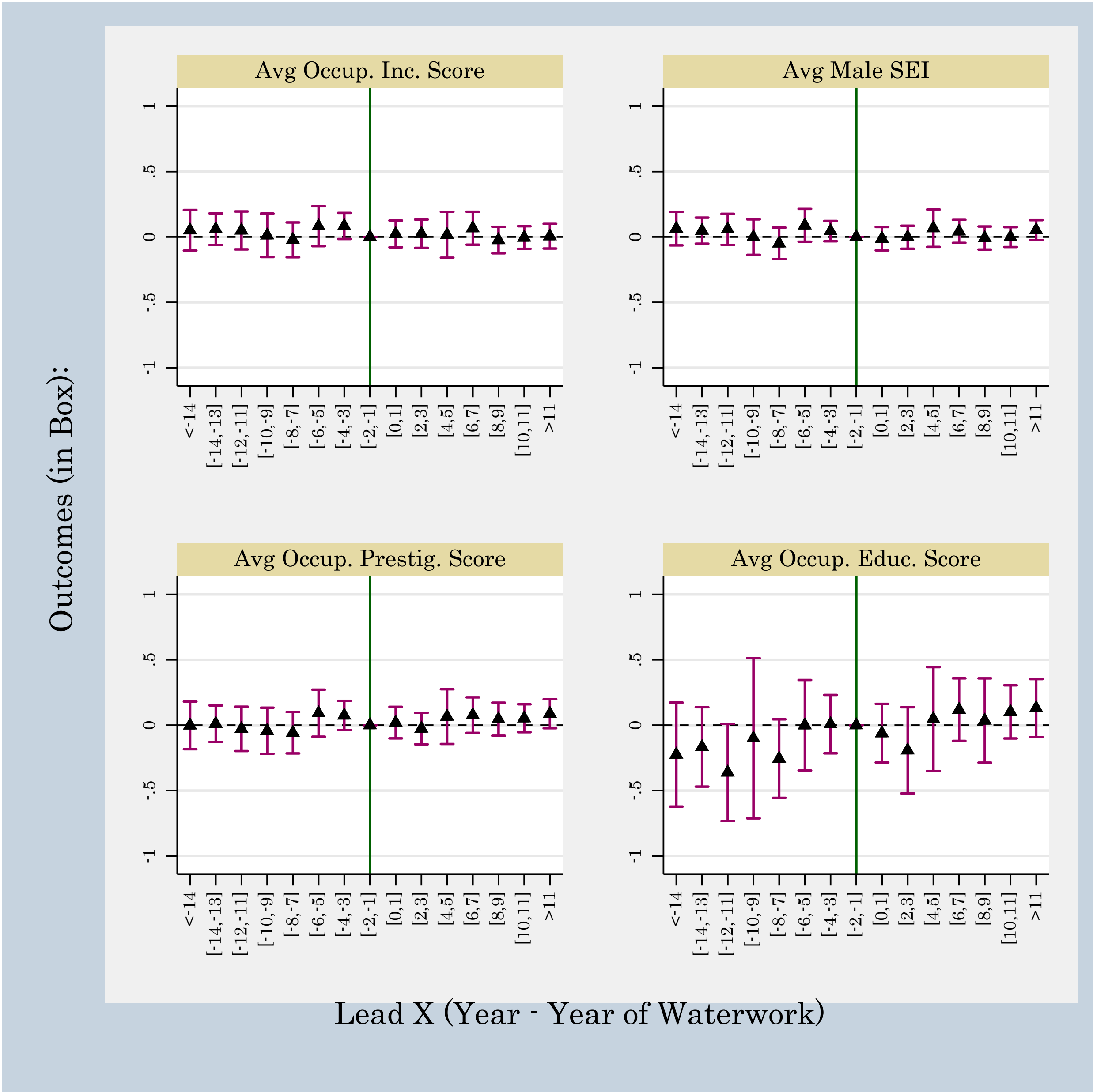

Figure 6 - Lead Pipe Installation and Changes in County-City Characteristics

Notes. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, and region-year-of-birth FE. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

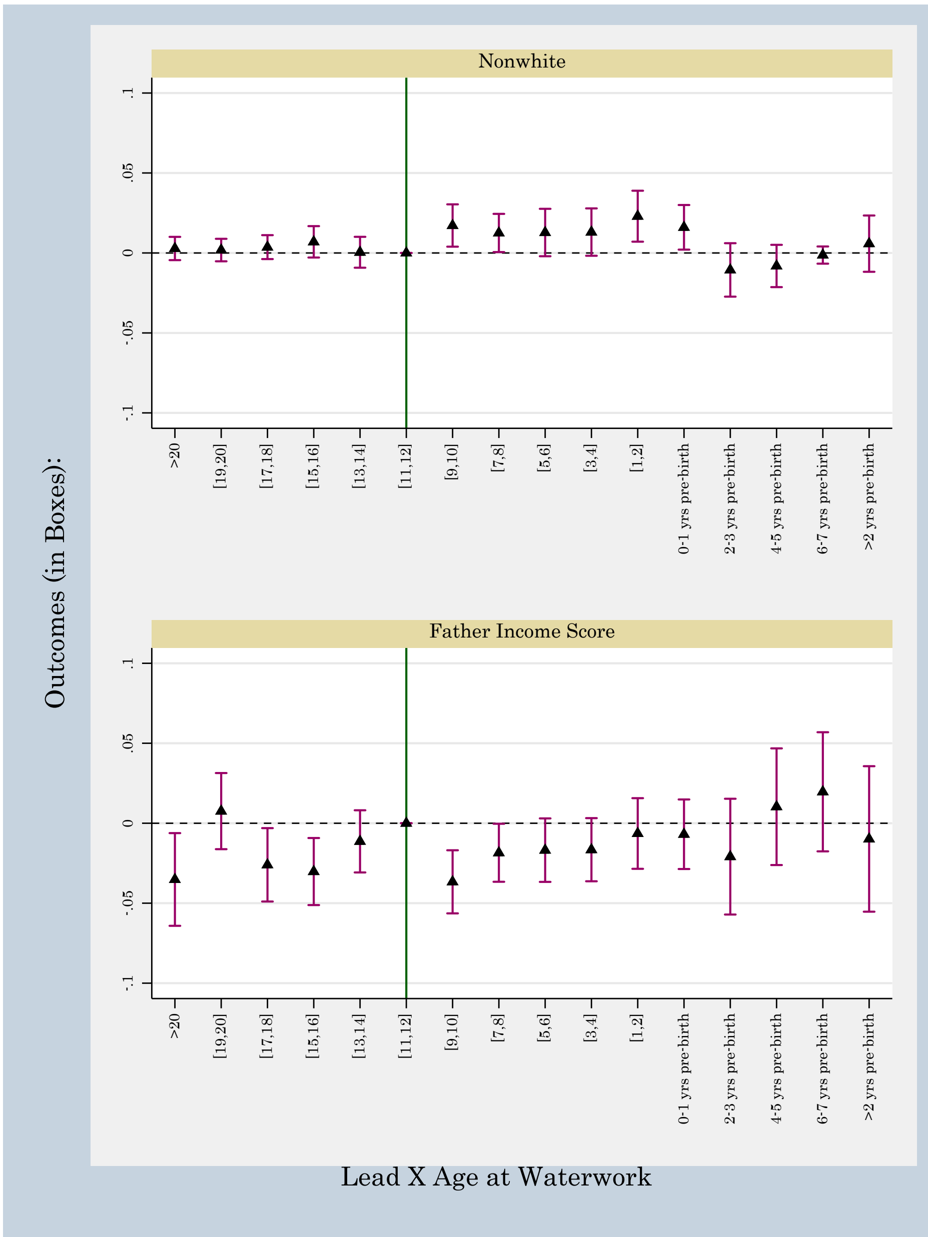

Figure 7 - Lead Pipe Installation and Changes in Children's Sociodemographic Characteristics in the Final Sample

Notes. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, and region-year-of-birth FE. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

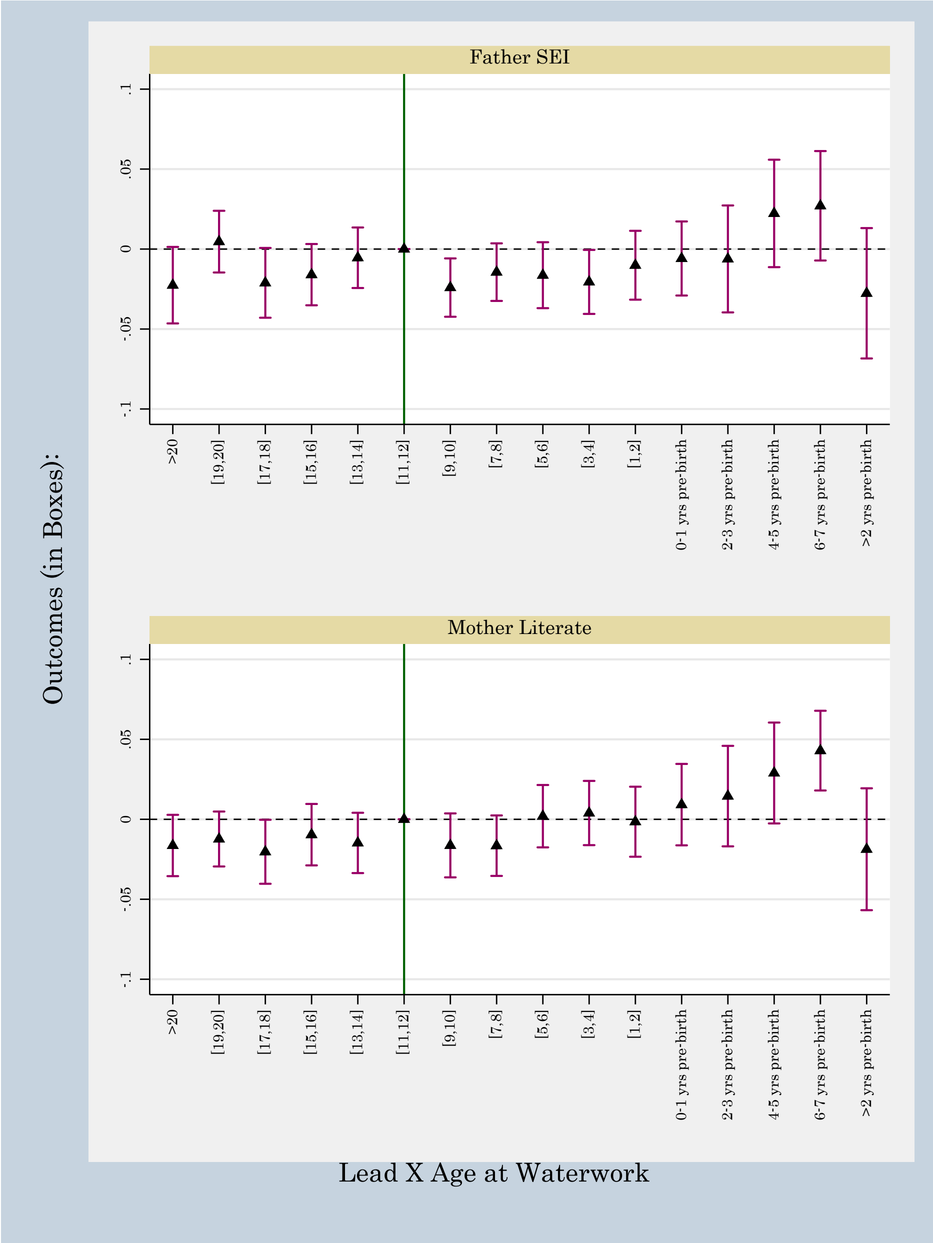

Figure 8 - Lead Pipe Installation and Changes in Children's Sociodemographic Characteristics in the Final Sample

Notes. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, and region-year-of-birth FE. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

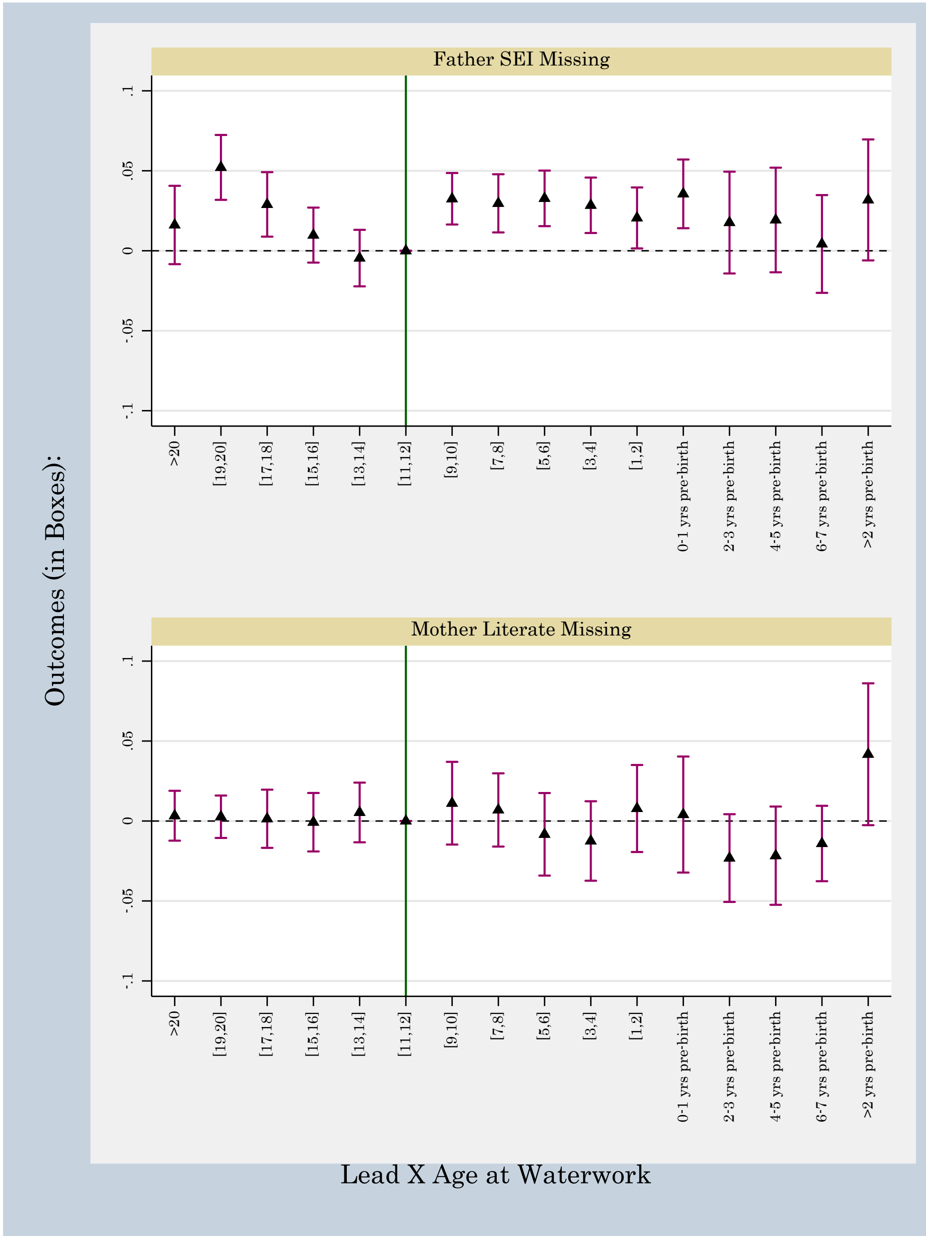

Figure 9 - Lead Pipe Installation and Changes in Children's Sociodemographic Characteristics in the Final Sample

Notes. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, and region-year-of-birth FE. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

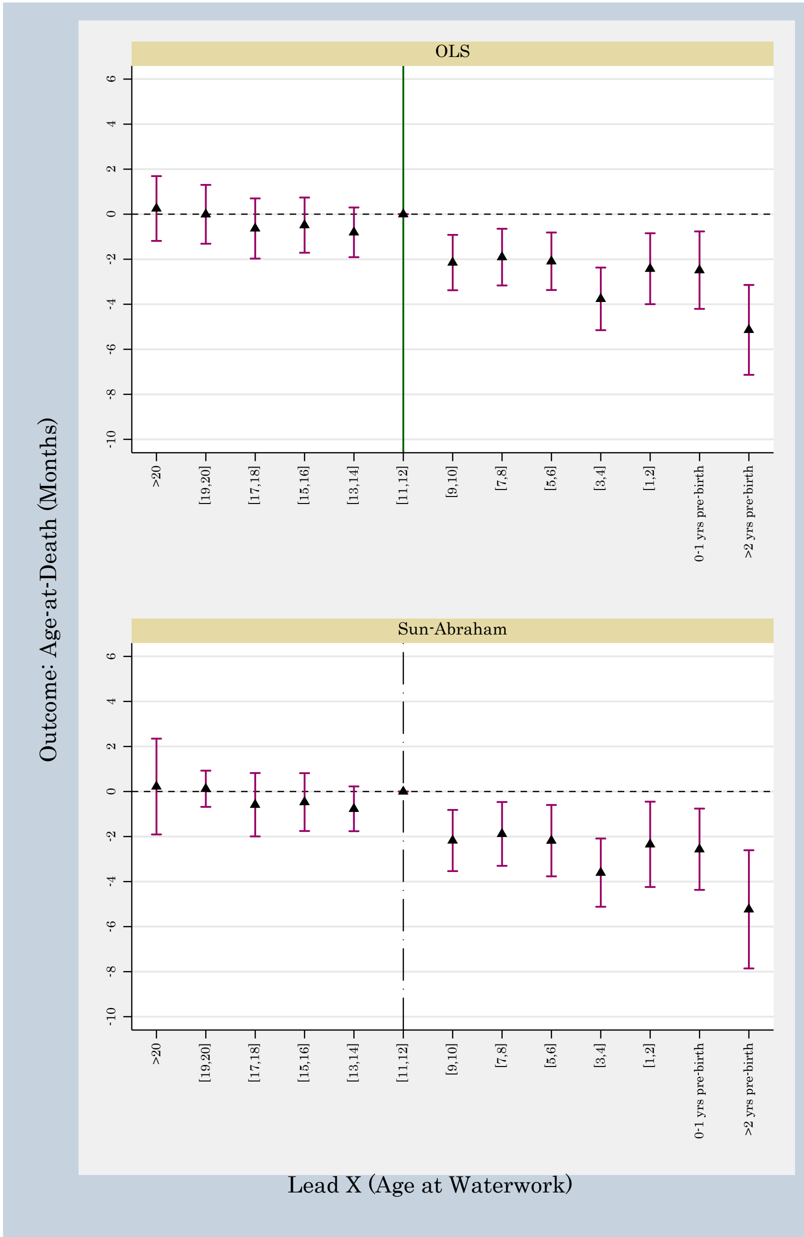

Figure 10 – Event-Study Results of Lead Pipe Installation and Longevity

Notes. The coefficient for age-at-exposure of 11-12 are eliminated so that these cohorts serve as a contrast group. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, region-year-of-birth FE, individual controls, and family covariates. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

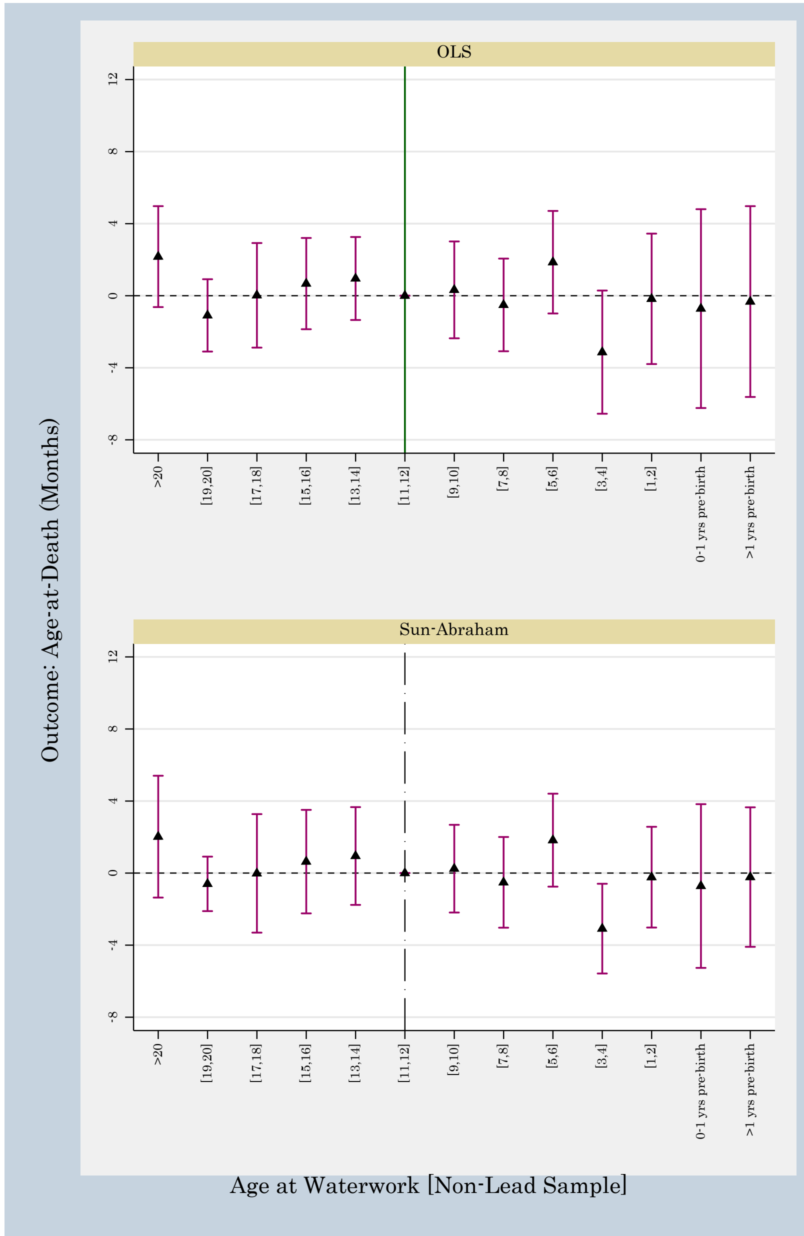

Figure 11 – Placebo Event-Study Results of Lead Pipe Installation in Non-Lead Cities and Longevity

Notes. The coefficient for age-at-exposure of 11-12 are eliminated so that these cohorts serve as a contrast group. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, region-year-of-birth FE, individual controls, and family covariates. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

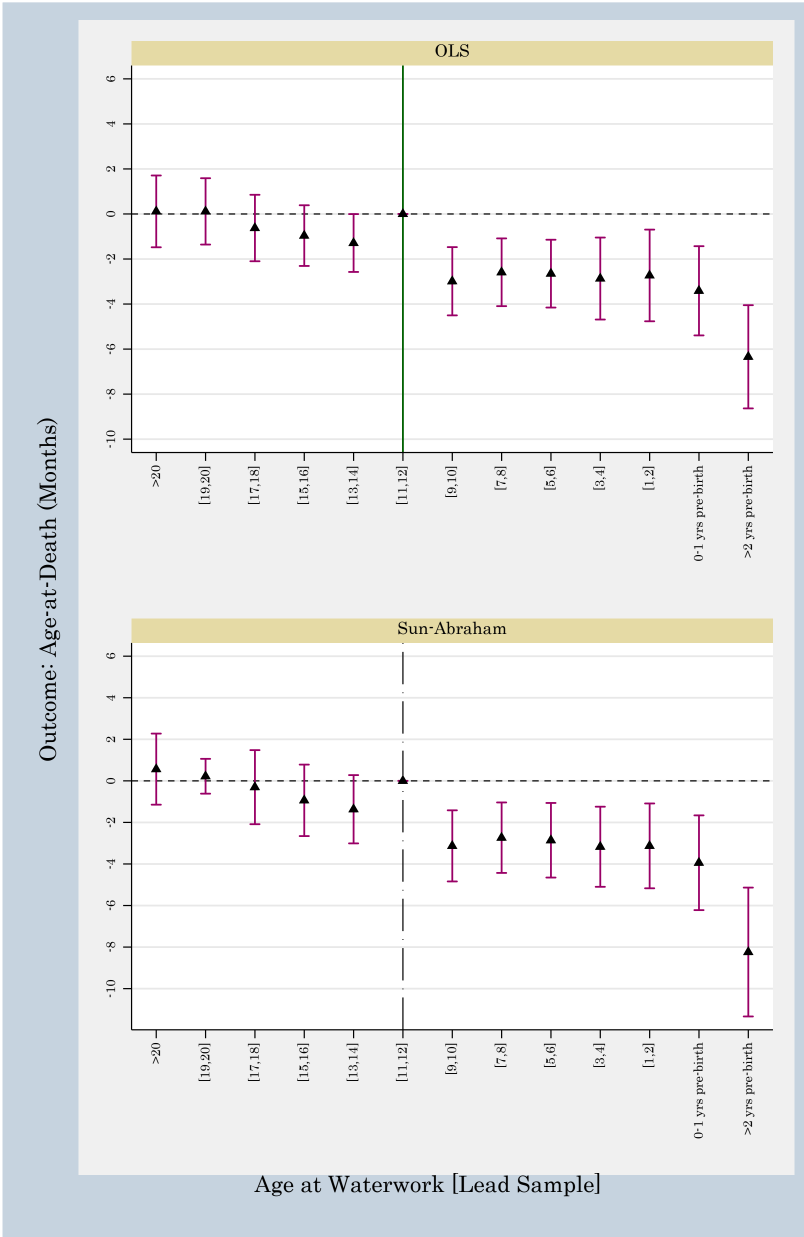

Figure 12 –Event-Study Results of Lead Pipe Installation in Lead Cities and Longevity

Notes. The coefficient for age-at-exposure of 11-12 are eliminated so that these cohorts serve as a contrast group. Point estimates and 95 percent confidence intervals are depicted. Standard errors are clustered on county-city. Regressions are weighted using county-city-level population. All regressions include county-city FE, county-city trend, region-year-of-birth FE, individual controls, and family covariates. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

Appendix A

In the difference-in-difference analysis of the main results, we defined the share of childhood up to age 12 that the individual was exposed to waterwork as the primary independent variable. This selection is to capture the combined effects of exposures during in-utero, early-life, and childhood. In this appendix, we examine the effects of exposure for the period of in utero. In so doing, we replicate the exposure measure in equation 1 with a dummy variable indicating that birth year is equal or greater than the year of waterwork. Regression results are reported in Appendix Table A-1. The results suggest an increase of about 67 percent when exposure starts from in utero.

Appendix Table A-1 – Replicating the Main Results for In-Utero Exposures

	Outcome: Age at Death (Months)		
	(1)	(3)	(5)
Share of Exposure Up to Age Z $\times$Lead	-2.14401	-4.49446**	-4.49884**
	(1.56334)	(1.99681)	(1.99337)
Share of Exposure Up to Age Z	2.30631	1.87989	1.88437
	(1.43176)	(1.78011)	(1.77663)
Observations	2416729	2416729	2416729
R-Squared	.39551	.39566	.39587
Mean DV	890.906	890.906	890.906
County-City FE	✓	✓	✓
Birth Year FE	✓	✓	✓
Region-Year of Birth FE	✓	✓	✓
County-City by Birth-Year Trend		✓	✓
Family Controls			✓

Notes. Standard errors, clustered on county, are in parentheses. Regressions are weighted using county-level population. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

*** p<0.01, ** p<0.05, * p<0.1

Appendix B

In the paper, we use cross-census linking rules to infer county and city of birth. One concern in using the cross-census rules is that selection into the linked sample could be correlated with the treatment, hence confounding the estimates with measurement errors. Moreover, it is likely that this measurement errors increases as cohorts age, considering the fact that they are mechanically more likely to migrate as they get older. We can empirically test this concern using the age-at-earliest-observed-census as the outcome to estimate differences in age at observation as a function of our exposure measures and other controls. We report the results in Appendix Table B-1. The coefficient of waterwork exposure suggests a small and insignificant effect of 0.01 years, off a mean of 15.5. Those who experienced exposure to waterwork in lead cities are 0.2 years younger when we observe them in historical censuses compared with control cohorts. Although this correlation is statistically significant, it is economically small. Moreover, the negative sign suggests slightly higher accuracy in assigning place of birth for exposed cohorts. In the same line of reasoning, less accuracy for unexposed cohorts is concerning if some of these cohorts were born/raised in another city with lead exposure and moved to non-lead cities. Since these people are treated but our regressions consider them as control groups, it is likely that the measurement error underestimate the true effects.

Another way to understand the direction and magnitude of this bias, is to use county and city of residence in 1940 (as reported in the 1940 census) as a proxy for place of birth. We implement the same sample selection and empirical method as in Table 3 and report the results in Appendix Table B-2. In the full specification of column 4, we observe a coefficient that is roughly 15 percent smaller than the effects in the main results. This suggests that measurement errors due to mi-assignment of county-city of birth likely induces a downward bias, and that the bias is relatively small.

Appendix Table B-1 - The Association between Lead Exposure and Age at the First Census

	Outcome: Age at the First Observation Census			
	(1)	(2)	(3)	(4)
Exposure×Lead	-.20962	.01139	-.20199	-.24381**
	(.16142)	(.15285)	(.12972)	(.11202)
Exposure	.81716***	.94954***	-.01052	-.01045
	(.17431)	(.16438)	(.09424)	(.08064)
Observations	2416729	2416729	2416729	2416729
R-Squared	.2597	.2612	.27227	.4389
Mean DV	15.548	15.548	15.548	15.548
County-City FE	✓	✓	✓	✓
Birth Year FE	✓	✓	✓	✓
Region-Year of Birth FE		✓	✓	✓
County-City by Birth-Year Trend			✓	✓
Family Controls				✓

Notes. Standard errors, clustered on county, are in parentheses. Regressions are weighted using county-level population. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.

*** p<0.01, ** p<0.05, * p<0.1

Appendix Table B-2 - Replicating the Main Results Using County-City of 1940 as a Proxy of County-City of Birth

	Outcome: Age at Death (Months)			
	(1)	(2)	(3)	(4)
Exposure×Lead	-1.98482***	-1.69224**	-2.45666***	-2.28411**
	(.65717)	(.71932)	(.92848)	(.92757)
Exposure	.276	.38801	1.0736*	1.14764*
	(.54199)	(.56134)	(.61289)	(.61283)
Observations	3339233	2975275	2975275	2975275
R-Squared	.44079	.41394	.41403	.41425
Mean DV	906.002	894.447	894.447	894.447
County-City FE	✓	✓	✓	✓
Birth Year FE	✓	✓	✓	✓
Region-Year of Birth FE		✓	✓	✓
County-City by Birth-Year Trend			✓	✓
Family Controls				✓

Notes. Standard errors, clustered on county, are in parentheses. Regressions are weighted using county-level population. Individual controls include race and ethnicity dummies. Family controls include dummies for father's socioeconomic index, maternal education, and a missing indicator for the missing values. The data covers birth cohorts of 1880-1930 who died between 1975-2005.
*** p<0.01, ** p<0.05, * p<0.1