Environmental and Resource Economics
https://doi.org/10.1007/s10640-025-01041-6

The Impact of Lead Water Pollution on Birth Outcomes: A Natural Experiment in Scotland

Anthony Higney[1] · **Nick Hanley**[1] · **Mirko Moro**[2]

Received: 21 April 2025 / Revised: 27 August 2025 / Accepted: 10 September 2025
© The Author(s) 2025

Abstract
We explore whether maternal lead exposure affects birthweights and child mortality in a setting where average blood lead levels were extremely high. We analyse two drinking water interventions in Scotland that reduced lead levels in Glasgow and Edinburgh from 1978 onwards. Using a staggered difference-in-differences design we examine administrative data of over 650,000 births between 1975 and 2000. We do not find consistent evidence of any effect leading to an increase in birthweights or a reduction in under-5 mortality. We estimate minimal detectable effects and can rule out even very low changes in birthweight, but we cannot rule out 1–3 deaths prevented per thousand due to the treatments. As our focus is on short-run outcomes around the time of birth, these findings do not rule out the possibility of longer-term impacts from early-life lead exposure. We also suggest our findings indicate future research should further explore the mediating pathways between lead and health outcomes.

Keywords Under-5 mortality · Pollution · Lead · Difference-in-differences

JEL codes I18 · Q53

1 Introduction

An estimated 2.4 million children die within their first year of life globally (UN IGME 2021). A further estimated 2 million are stillborn (UNICEF 2022). High levels of lead in the environment have, historically, been linked with poor health outcomes for children. With an estimated 1-in-3 children having elevated levels of lead in their systems (GBD, 2019), and the global burden of lead estimated to be responsible for as many as 900,000 deaths a

✉ Anthony Higney
Anthony.Higney@glasgow.ac.uk

[1] School of Biodiversity, One Health and Veterinary Medicine, University of Glasgow, Glasgow, Scotland

[2] Division of Economics, University of Stirling, Stirling, Scotland

Published online: 07 October 2025

year (UNICEF, 2020), reducing lead pollution may be one route to prevent infant death and morbidity.

In this paper, we follow Troesken (2006) in examining the impact of lead water pollution on infant health outcomes. Lead may contaminate drinking water through chemical reactions to plumbing. Metal from lead pipes and fixtures can dissolve or erode, increasing lead content in domestic water supplies. This reaction is particularly severe when the water has low mineral content or high acidity, needed to form protective scaling inside pipes, making it especially plumbosolvent and prone to dissolving lead from plumbing systems. The public water supplies in Edinburgh and Glasgow are sourced from upland catchments where the underlying soil and rock chemistry produce naturally soft, acidic water.

This study exploits a uniquely rich empirical setting and dataset to examine the effects of lead exposure on infant outcomes. Scotland's cities' historically high water and blood lead levels—comparable to the worst levels measured today (Ericson et al. 2021)—offer a rare opportunity to study the effects of substantial and prolonged exposure to lead in drinking water. Edinburgh and Glasgow water supplies are characterised by acidic soft water which was especially plumbosolvent, due to the nature of the soil chemistry in upland areas from which water is collected. In 1975, 33% of households in Scotland had water lead levels above 50 µg/l, compared to only 10% in England. Glasgow was badly affected, with 50% of households surveyed having water lead levels above 100 µg/l[1] (Quinn 1985; Potter 1997; Richards and Moore 1984). Due to the high water-lead levels, these cities were among the first to treat their water in the 1970s to raise the pH. This turned out to be inadequate, so a later treatment in the 1980s and 1990s was added. The long-running Glasgow (Watt et al. 1996a) and Edinburgh (Macintyre et al. 1998) lead studies, which meticulously detailed and researched the reductions in the water and blood lead levels over the 1980s and 1990s, indicated the interventions sharply reduced water lead levels and blood lead levels in both cities. Surrounding areas were treated later.

We exploit the plausibly exogenous change in lead exposure these two treatments induced through changes in the water supply compared to surrounding areas. Our analysis uses rich administrative birth and mortality records covering all 650,000 births in Edinburgh, Glasgow, and the surrounding areas between 1975 and 2000. We combine this with precise geographic linkage of water treatment areas based on maternal addresses. Linking the data to home address allows us to capture if a mother and child lived in an area subject to the lead reduction treatment at the time of birth. The combination of large-scale administrative data, natural experiments, and detailed exposure mapping provides a strong basis for credible identification of the relationship between lead exposure and infant health.

We focus on the effect of lead pollution on *birthweight* and *under-5 mortality*. We use birthweight as it is a proxy for a wide range of future life outcomes (see Law 2002; Wilcox 2001; Chatterji et al. 2014; Behrman and Rosenzweig 2004; and Royer 2009). Much previous evidence shows mixed findings of lead pollution on birthweights and on under-5 mortality (for example: Xie et al. 2013; Bornschein et al. 1989, Taylor et al. 2015; Azayo et al. 2009; Golmohammadi et al. 2007; McMichael et al. 1986; Vinceti et al. 2001), with most studies relying on correlational estimates of the relationship between lead pollution and birth outcomes, or low sample sizes. The very few quasi-experimental papers, reviewed in Clay et al. (2024), tend to find an effect, but the quasi-experimental paper with the largest

[1] For context, current regulatory benchmarks vary. In the EU and UK: 10 µg/l; U.S.: 15 µg/l; Canada: 5 µg/l, which situates the high historical levels we study.

sample size (Grönqvist et al. 2020), appendix E) was not included in that review and does not find an effect. In their review of lead and infant health papers, Clay et al. (2024) cite the paucity of evidence and call for more research on this topic. We directly answer that call.

Our research design, based on a robust, staggered difference-in-differences approach, adds to the few papers that use quasi-experimental designs to identify the effect of lead on birth outcomes. Additionally, many existing studies have small sample sizes, while our sample includes over 650,000 births.

Summarising our results, we do not find consistent evidence for an effect of the lead reduction treatment on birthweights or mortality. Our results are similar to the other large sample, European setting paper of Grönqvist et al. (2020), where they do not find evidence for an effect on birth outcomes, but they do on later education outcomes. We also, in a related paper, find effects of the treatment on education, 16 years after the second treatment (Higney et al. 2025), but no consistent evidence for an effect on birth outcomes here. Our results do not match the North American setting papers in the Clay et al. (2024) review, which do find effects on mortality and birthweight. Our analysis of the minimal detectable effect our study can find rules out even very low changes in birthweight, but we cannot rule out 1–3 deaths prevented per thousand due to the treatments. We suggest our findings indicate that future research should explore the mediating pathways between lead and health outcomes to explore these differences in empirical findings of causal links between lead in drinking water and infant/child health.

2 Background

2.1 Lead Pollution and Birth Outcomes

A child is first exposed to lead pollution through the placenta (Dorea and Donangelo 2006), so that a mother's exposure to high levels of lead can in turn expose a foetus to lead pollution. Furthermore, due to increased bone remodelling, previous maternal lead pollution can affect the foetus, as both lead and calcium (chemically similar) are released from the bones at an increased rate during pregnancy (Yurdakök 2012). Maternal and infant lead levels are of similar magnitudes and highly correlated (Al-Saleh et al. 1995), but the relationship between exposure and absorption of lead is complex. For example, it is mitigated by maternal calcium intake (Dorea and Donangelo 2006). Therefore, there are mediators between lead exposure and the damage it may cause.

High levels of lead exposure in the womb are harmful. So much so, that lead oxide was described as being used as an abortifacient by women in the 1800s (Hall and Ransom 1906). In some cases, the amounts of lead ingested were strong enough to cause lead poisoning in the mother (Ransom 1900). However, there are few high-quality causal estimates of the effect of lower but still elevated lead levels that remain common in many parts of the world. Most studies are correlational and find mixed effects. Most of the few quasi-experimental studies are reviewed in Clay et al. (2024) whose review finds lead lowers birthweights and increases infant mortality. However, missing from the review was the largest study, and the only one from Europe: Grönqvist et al. (2020). They use an instrument of local lead moss levels for blood lead levels with a sample of 800,000 children in Sweden and find no effect on birthweights or premature births.

2.2 Lead Plumbing and Water Treatment in Glasgow and Edinburgh

Scotland has seen greatly reduced infant deaths from all causes since 1900 (Fig. 1). Starting in the 1970s, interventions to reduce the amount of lead in drinking water supplies began in Edinburgh and Glasgow, and were improved upon in the 1980s and 90s. This was after infant deaths and stillbirths had already sharply reduced, thanks to improved nutrition, hygiene, and health practices. Therefore, this is a setting where the relatively easy child health gains had already been exhausted, and lead might be thought to account for a larger share of the remaining deaths and pregnancy complications.

Lead piping was widely used in housing in Scotland before being banned for new construction in 1968 (Richards et al. 1980). Lead is malleable, relatively cheap, and has an extremely long life as infrastructure (Feigenbaum and Muller, 2016, and Krebs 2019). Lead piping began to be phased out from the 1970s in Scotland, and all lead water pipes belonging to the mains supplier have been removed. However, in the 1990s as many as 589,000 homes in Scotland were estimated to contain lead pipes (Potter 1997), around 30% of the total. There were also as many as 60,000 water storage tanks made of lead, mostly in Glasgow and Edinburgh (Krebs 2019). These were used because water service was still intermittent in the first half of the 20th century. The tanks allowed households to store and use water during any non-flowing periods.

The reason lead water pipes have not been entirely replaced are twofold: 1) It is expensive, and 2) Homeowners do not know they have lead pipes and that they are responsible for their replacement. Communication pipes are owned by the water supplier and those made of lead have now all been replaced (Akoumianaki 2017). Internal lead piping still exists in many households but has also been gradually replaced and is estimated to only account for 20–30% of the remaining lead pollution in home water supplies in the UK (Akoumianaki 2017). The main pollution burden is thought to come from lead supply pipes (also called service pipes) which connect individual properties to main supply pipes. These are the responsibility of the property owner to replace, but are underground, and therefore difficult to see. An added complication is that property owners may not know they have the responsibility to obtain replacement unleaded pipework, even though grants are available. Today there are estimated to be 273,000 homes out of 2.6 million in Scotland with lead piping (Robertson

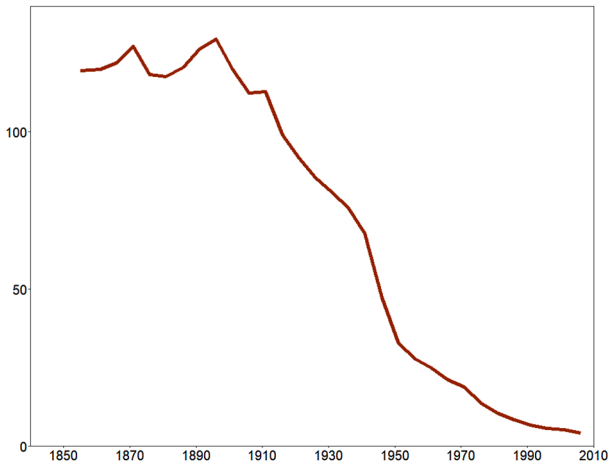

Fig. 1 Deaths within First Year of life, per 1000 births in Scotland source: National Records of Scotland [WWW Document] (2022)

et al. 2020). Watt et al. (2000) estimated as many as 160,000 households out of 300,000 in Glasgow alone had a lead service pipe in 2000.

The dangerous combination of naturally acidic water chemistry and lead pipes began to be taken seriously in the 1970s. The UK's Department for the Environment carried out a series of surveys of blood lead levels in the 1970s and 80s. The findings were that "The highest blood lead concentrations were related to plumbosolvent water" and not distance to roads (Quinn 1985 This was particularly problematic in Scotland's two largest cities, where water is sourced from upland catchments with low mineral content. The resulting soft water is also acidic, with low pH levels that increase the solubility of lead in plumbing systems. Unlike hard water, which contains higher concentrations of calcium and carbonate ions that can deposit on the interior of lead pipes and form a protective scale, soft, low-pH water lacks this buffering capacity and is especially prone to dissolving lead (Schock and Lytle 2011; Comber et al. 2011).In 1975 surveys found 33% of households in Scotland had water lead levels above 50 µg/l, compared to 10% in England (Potter 1997). Glasgow was especially viewed as a problem, with 50% of household surveyed having water lead levels above 100 µg/l (Richards and Moore 1984). Average blood lead levels were higher in both cities than in any country today (Fig. 5 and Ericson et al. 2021).

This paper exploits two key chemical interventions to reduce lead in the water supply. We treat these as natural experiments, given the somewhat arbitrary assignment of water treatment to Glasgow and Edinburgh (treatment groups), while leaving many adjacent areas untouched until years later (control groups). These control areas were not only treated later, but also had higher pH levels for their untreated water than the treatment areas. In particular, we rely on this plausibly exogenous variation of lead to identify treatment effects of lead on birth outcomes:

• Treatment 1: the staggered increase in pH levels (reducing acidity and therefore the potential level of lead in drinking water) through lime dosing in Glasgow and Edinburgh. Lime dosing increases the pH and alkalinity of the water, reducing its corrosivity and buffering against further acidification. This helps to limit the dissolution of lead from internal plumbing. This occurred in 1978 for Glasgow, Edinburgh Southwest and (partially) Edinburgh Central, and in 1985 for Edinburgh Northeast. Importantly, these interventions were not carried out in the many surrounding areas of these cities until much later.

• Treatment 2: the staggered dosing with orthophosphate in both cities in 1989 in Glasgow and in 1991 in every area of Edinburgh, to reduce the bioavailability of lead. In practice, orthophosphate reacts with dissolved lead to form insoluble lead-phosphate compounds. These compounds adhere to pipe surfaces, creating a stable layer that inhibits further leaching. Also in this case, this treatment was not implemented in the neighbouring areas until years later.

A full timeline of the treatments is given in Fig. 2.[2] The control group in all cases consists of adjacent areas that did not receive the treatment until later (depicted by the grey areas in Fig. 6). This timeline, along with different treatment groups, provides the scope for the implementation of staggered difference-in-differences methods, described in Sect. 4.

[2] There is no evidence that lime dosing or orthophosphate addition has direct adverse effects on human health at operational levels. The WHO sets no health-based guideline for pH in drinking water; pH is managed solely for purposes of corrosion control, disinfection efficiency, and taste (WHO 2022). Orthophosphate similarly reduces lead bioavailability; its reported trade-offs are operational or environmental—not obstetric (Comber et al. 2011; Hayes and Hydes 2012). Accordingly, any effect of lime dosing and orthophosphate on birth outcomes is assumed to occur indirectly, through reduced plumbosolvency and lower lead exposure.

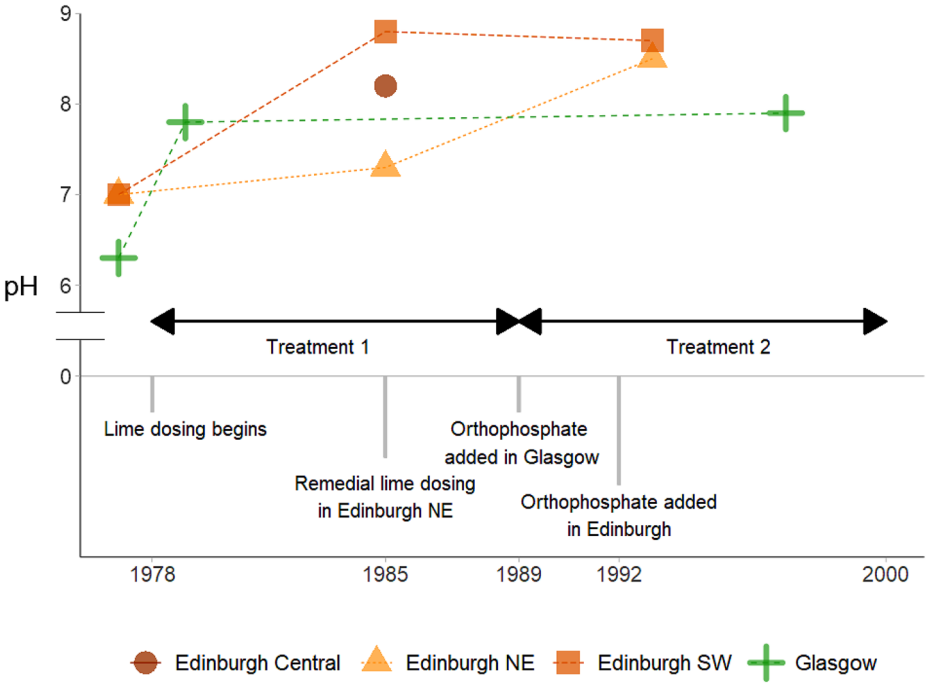

Fig. 2 pH levels in each water supply area. Notes: this chart shows pH levels for the various water supply areas in different years. It also shows when the lime dosing (treatment 1) and orthophosphate dosing (treatment 2) began in each area. Water engineers targeted pH levels to be well above 7 to reduce plumbosolvency. Sources: Macintyre et al. (1998), Richards et al. (1980) and Watt et al., 1996

Treatment 1, lime dosing was undertaken because experiments with the Glasgow water supply in 1973 showed it would raise the pH effectively and thus lower plumbosolvency (Richards et al. 1980). Following this, in both Glasgow and Edinburgh, an investment in an automatic lime-dosing system was considered worthwhile. These began operation in 1978. In Glasgow, which is supplied by Loch Katrine water, the pH was raised from 6.3 to 7.8 after this dosing (Fig. 2). The Glasgow water supply area at this time is mapped in Fig. 3. This map also highlights postcodes with a higher prevalence of lead piping in homes. The distinction between areas with high and low prevalence of lead pipes is based on a survey conducted in Glasgow in the early 1990s which findings are summarised in Watt et al. (1996a). We exploit the difference in high and low lead prevalence areas in Glasgow as a robustness check in the tables A.8 and A.9 in the appendix.

In Edinburgh, the city was supplied with water from two main sources: Alnwickhill, which served the north-eastern part of the city (referred to as Edinburgh NE for simplicity), and Fairmilehead, which catered to the south-western region (Edinburgh SW). The central area of the city (Edinburgh Central) received a combined supply from both these sources. See Fig. 4 for the map of these water supply areas. The water from both these sources was relatively soft, with a pH level of around 7 before dosing. In 1978, a successful lime dosing at Edinburgh SW raised the pH level to above 8. However, similar efforts at Edinburgh NE faced technical difficulties, delaying effective dosing until 1985. As a result, the pH level in Edinburgh NE remained below 8 until after 1985, when it eventually rose to around 8.5.

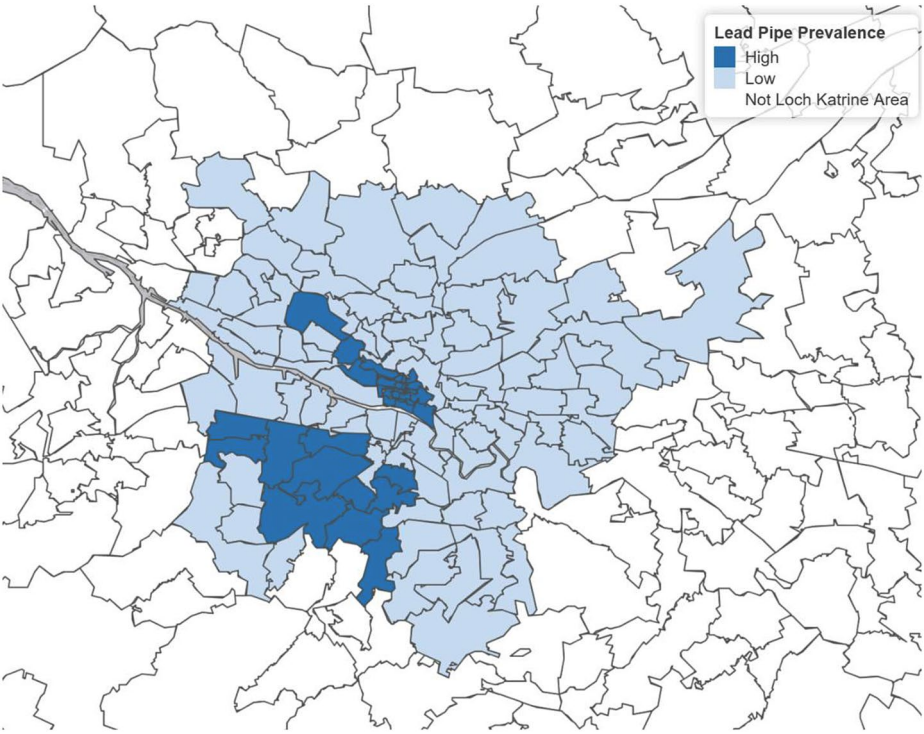

Fig. 3 Historical Loch Katrine Water Supply Area with High and Low Lead Piping Prevalence Note: The areas in dark and light blue represent treated areas that underwent water lead reduction interventions in 1978 and 1989. The heterogeneous effect of these interventions on areas with higher and lower prevalence of lead piping is investigated in the Appendix. Source: Watt et al. (1996a)

Edinburgh Central was supplied jointly by Edinburgh NE and Edinburgh SW and therefore received a partial treatment. However, when measured in 1985 it was found that its pH level exceeded 8, aligning more closely with the pH level observed in Edinburgh SW, the area treated successfully in 1978. The pH levels before and after the lime dosing for each water supply area are depicted in Fig. 2. This data provides a clear comparison of the effectiveness of the treatments in Glasgow and Edinburgh SW in 1978 and in Edinburgh NE in 1985.

These treatments lowered both water and blood lead levels (Fig. 5), but not by enough from the perspective of the government. Treatment 2, orthophosphate dosing, was therefore carried out to further decrease lead in the water supply. Orthophosphate dosing reduces the solubility, and therefore bioavailability, of lead in the water supply (Comber et al. 2011). This is a different mechanism for reducing lead pollution compared to lime treatment, which raises the pH. This was done in Glasgow in 1989 and in all of Edinburgh in 1991. This further reduced blood lead levels (Fig. 5). As the UK government brought in stricter lead-water maximum levels, other areas followed. Eventually, in the 2000s, 95% of the UK's water would be treated with orthophosphate (Hayes and Hydes 2012). The current prescribed limit for lead in drinking water at consumers' taps in the UK is 10 µg/L, following staged

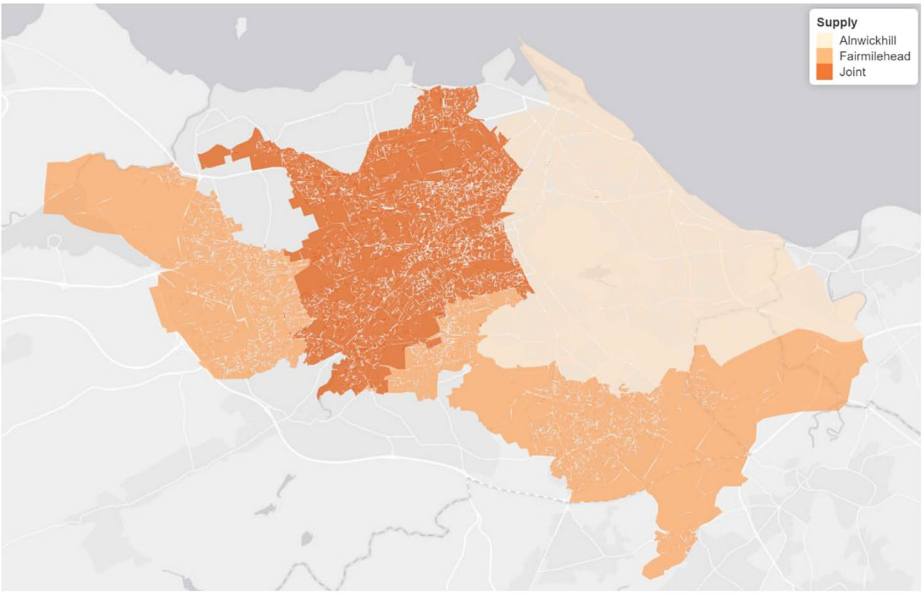

Fig. 4 Historical Water Supply Areas in Edinburgh. Note: The North-East area of Edinburgh (Edinburgh NE) was served by the Alnwickhill water supply. The South-West area of Edinburgh (Edinburgh SW) was served by the Fairmilehead water supply. The central area (Edinburgh Central) is jointly served by both water supplies

reductions from 50 µg/L in 1989 to 25 µg/L in 2003 and 10 µg/L in 2013 (UK Government 1989, 2003, 2016).[3]

3 Data

We use health data from Public Health Scotland (PHS). The data covers all the pregnancy outcomes in Glasgow, Edinburgh, and the surrounding postcode areas of each city for the period 1975 to 2000. This data is from the Scottish Morbidity Records (SMR) and the Death, Birth and Stillbirths Registrations (NRS) Furthermore, from the NRS records, we link live births records with death registrations to identify if a child died before age five.[4]

These data are matched to the historical Scottish Water Supply area maps for the relevant areas by using maternal postcodes for the relevant period. That is, the address of the house at the time of pregnancy is assigned to the postcode and coded as the relevant treatment/

[3] Regulatory limits for lead in drinking water vary internationally and provide useful benchmarks for interpreting our setting. In the United States, the current action level is 15 µg/l under the EPA's Lead and Copper Rule, with a maximum contaminant level goal (MCLG) of zero (UK Government, 2016). In Canada, Health Canada sets a maximum acceptable concentration of 5 µg/l (Health Canada 2019). The European Union, Directive (EU) 2020/2184 retains a limit of 10 µg/l as in the UK, but with the key difference that it mandates a reduction to January 2036, after which it will fall to 5 µg/l (EU 2020).

[4] We would like to thank the Electronic Data Research and Innovation Service (eDRIS) of Scotland for their help with data handling and access. The control areas were the maximum allowed by NHS Scotland of the areas surrounding Glasgow and Edinburgh.

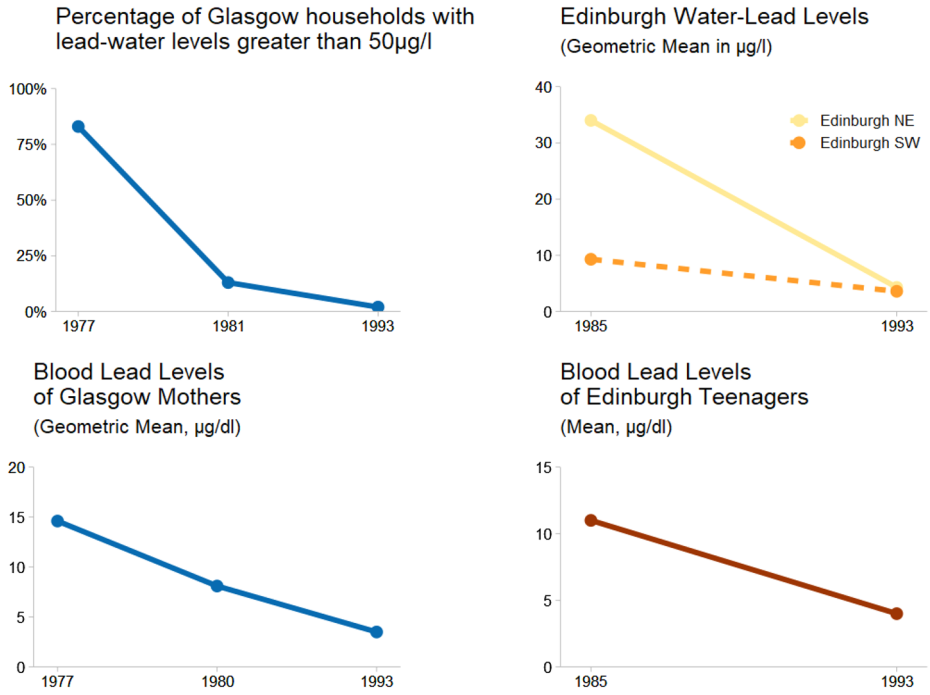

Fig. 5 Water and blood lead levels in Glasgow and Edinburgh sources: Watt et al. (1996a) and Moore (Moore et al. 1998),Macintyre et al. (1998)

control group. There are 139 treated postcode sectors in Glasgow, 74 in the Edinburgh area, and 185 in the control group. The map of water supply areas, and the various treatment groups are included in Figs 3, 4, and 6. Fig. 3 shows the Loch Katrine water supply area during the period the data cover. There is a further split in the Loch Katrine supply area between postcode sectors with relatively high levels of lead piping compared to those with relatively low levels of lead piping, as given in Watt et al. (1996a). In high lead areas, 19% reported lead piping in a survey, while in low lead areas it was 9%. Fig. 4 shows the water supply areas in Edinburgh during the period the data cover. The Fairmilehead source supplied mostly the west of Edinburgh (which we label Edinburgh SW), while Alnwickhill served Edinburgh NE. The "Joint" area, which we label for simplicity Edinburgh Central, is supplied by both water sources during this period. As explained in the methods section, the first treatment of liming was effective in 1978 in Edinburgh SW, but not effective till 1985 in Edinburgh NE. We therefore consider Edinburgh Central which is served by both sources as being treated at the same time as Edinburgh SW in 1978, but exclude it as a robustness check in the appendix.

Our two main outcomes are birthweight and under-5 mortality. We use only single births. Twins, and other multiple births are excluded as their outcomes tend to be very different, with lower birthweights in comparison to single births, as well as different probabilities of complications. However, multiple births are only around 1.5% of all births. Our mortality indicator includes all deaths and recorded non-viable pregnancies, stillbirths and spontaneous abortions. We also link the data with Scottish Morbidity Records so that it includes any

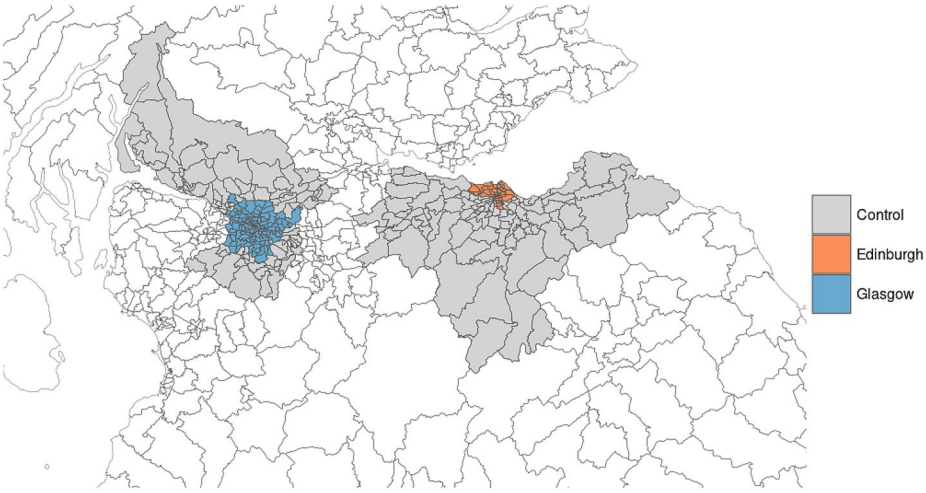

Fig. 6 Map of treatment and control areas

deaths up to age 5. Under-5 mortality is the commonly used definition of child mortality and is the indicator used in Sustainable Development Goal (SDG) target 3.2: "Newborn and child mortality: By 2030, end preventable deaths of newborns and children under 5 years of age, with all countries aiming to reduce neonatal mortality and under-5 mortality".[5] Additional data used as controls at the individual level include the biological sex of the baby, and a series of mother's characteristics such as age, height, and previous obstetric history, such as the number of previous spontaneous abortions, and number of previous pregnancies. At the postcode level, the data is linked to Carstairs scores,[6] which are material deprivation indices that rank different areas by using information from the 1981 census about car ownership, male unemployment, overcrowding and low social class. If a postcode is in the bottom two deciles, we code that postcode with an indicator variable as being in a deprived area. This link to deprivation allows us to control for higher income households undertaking more pollution avoidance behaviour. Table 1 includes descriptive statistics of the variables used.

4 Methods

We use the plausibly exogenous change in water treatments, at different points in time, to identify the effect of lead-water pollution on birth and early life health outcomes. Our main specifications use a staggered difference-in-differences design. We further discuss the estimands, assumptions necessary, and specifications below. The following section is largely

[5] See the SDG targets and indicators here: https://sdgs.un.org/.

[6] These are rankings of areas by material deprivation. The variable takes into account material good ownership, such as car ownership, self-reported class, and unemployment amongst other variables to make an index. The Carstairs scores were originally developed by Carstairs and Morris (1991) and are regularly generated and published by the MRC/CSO Social and Public Health Sciences Unit, the University of Glasgow (https://www.gla.ac.uk/schools/healthwellbeing/research/mrccsosocialandpublichealthsciencesunit/programmes/inequalities/healthinequalities/determinantsofhealthandhealthinequalitiesinscotland/carstairsscores/).

Table 1 Descriptive statistics

Variable	Group	Mean	Median	Std Dev	Obs
Birthweight (grams)					
	Control	**3318**	**3360**	**594**	**353,643**
	Edinburgh	**3320**	**3360**	**595**	**76,498**
	Edinburgh NE	3310	3350	603	26,172
	Edinburgh SW	3372	3410	567	8,315
	Joint	3317	3360	595	42,011
	Glasgow	**3257**	**3300**	**591**	**216,771**
Death Indicator Variable					
	Control	**0.01**	**0**	**0.09**	**353,643**
	Edinburgh	**0.01**	**0**	**0.09**	**76,498**
	Edinburgh NE	0.01	0	0.10	26,172
	Edinburgh SW	0.01	0	0.09	8,315
	Joint	0.01	0	0.09	42,011
	Glasgow	**0.01**	**0**	**0.09**	**216,771**
Proportion Living in Deprived Areas (Carstairs Index)					
	Control	**0.11**	**0**	**0.32**	**353,643**
	Edinburgh	**0.13**	**0**	**0.34**	**76,498**
	Edinburgh NE	0.06	0	0.23	26,172
	Edinburgh SW	0.04	0	0.19	8,315
	Joint	0.20	0	0.40	42,011
	Glasgow	**0.59**	**1**	**0.49**	**216,771**
Total Previous Pregnancies					
	Control	**1.18**	**1**	**1.28**	**353,643**
	Edinburgh	**1.12**	**1**	**1.29**	**76,498**
	Edinburgh NE	1.07	1	1.24	26,172
	Edinburgh SW	1.15	1	1.27	8,315
	Joint	1.15	1	1.31	42,011
	Glasgow	**1.24**	**1**	**1.41**	**216,771**
Mother's Age					
	Control	**27.72**	**28**	**5.29**	**353,643**
	Edinburgh	**28.43**	**29**	**5.46**	**76,498**
	Edinburgh NE	28.25	28	5.32	26,172
	Edinburgh SW	29.93	30	5.24	8,315
	Joint	28.25	28	5.54	42,011
	Glasgow	**26.90**	**27**	**5.58**	**216,771**
Number of Previous Spontaneous Abortions					
	Control	**0.22**	**0**	**0.57**	**353,643**
	Edinburgh	**0.23**	**0**	**0.59**	**76,498**
	Edinburgh NE	0.23	0	0.57	26,172
	Edinburgh SW	0.24	0	0.61	8,315
	Joint	0.23	0	0.59	42,011
	Glasgow	**0.23**	**0**	**0.59**	**216,771**
Male Infant Proportion					
	Control	**0.51**		**0.50**	**353,643**
	Edinburgh	**0.51**		**0.50**	**76,498**
	Edinburgh NE	0.51		0.50	26,172

Table 1 (continued)

Variable	Group	Mean	Median	Std Dev	Obs
	Edinburgh SW	0.52		0.50	8,315
	Joint	0.51		0.50	42,011
	Glasgow	**0.51**		**0.50**	**216,771**

based on the expositions in Athey and Imbens (2022), Wooldridge (2021), and Wooldridge (2010).

4.1 Estimands

Our main results focus on three estimands. First, the average effect of water treatment (and therefore lead reduction) at time t on the group which began treatment at time r.

We write this τ_{rt} and define it formally below.

$$\tau_{rt} = E\left[y_{it}\left(r\right) - y_{it}\left(0\right) d_{ir} = 1\right], r = q, \ldots, T; t = r, \ldots, T. \tag{1}$$

Where $y_{it}\left(r\right)$ is the outcome for child i at time t given their water supply began treatment at time r, and $r \leq t$, and $y_{it}\left(0\right)$ is the unobserved counterfactual outcome for child i at time t where they have not yet received treatment, q is the first period where any cohort is treated, and d_{ir} is a cohort indicator which equals 1 if individual i is in treatment group r. Simply, τ_{rt} is the average treatment effect on the treated (ATT) for that treatment cohort in that year.

Our second estimand is the average treatment for a specific treatment group, for all the years of treatment in our data.

$$\bar{\tau}_r = E\left[\tau_{rt}\right], r = q, \ldots, T; t = r, \ldots, T. \tag{2}$$

Which we estimate as:

$$\widehat{\bar{\tau}}_r = \frac{\sum_{t=r}^{T} \hat{\tau}_{rt}}{(T - r + 1)} \tag{3}$$

Our third estimand, is the overall average for all groups and all years, which we estimate with:

$$\hat{\tau} = \sum_{t=r}^{T} \hat{\tau}_r \times w_r \tag{4}$$

Where w_r is simply a weight that equals the proportion of treated units that are in group r.

4.2 Difference-in-Differences Design Models

Our main results are from models relying on difference-in-differences designs. In the baseline, reduced form model, lead levels, given by the variable $Lead_{it}$, are assumed to affect the birth outcome as shown in (5). The effect of lead is given byT.

$$y_{ijt} = c_j + g_t + \boldsymbol{x_i}\boldsymbol{\beta} + (\mathrm{T} \times Lead_{it}) + u_{it} \tag{5}$$

where y_{ijt} is the outcome for individual i, in postcode *j,* at time t. There is a time-invariant postcode-level effect, c_j, a time trend in outcome, g_t, and a vector of other variables that affect the outcome, $\boldsymbol{x_i}$, which vary by individual. The final term u_{it} is the error term.

This model cannot be estimated for a number of reasons, not least because lead exposure of each individual at each time is unknown. Even if known, other variables may co-vary with lead and the outcome, leading to biased estimates due to endogeneity. We could estimate a two-way fixed effects model using a $post \times treatment$ indicator but, given the staggered timing of the intervention between Glasgow, Edinburgh SW, and Edinburgh NE, this could lead to the effect not being identified, due to the "forbidden comparisons" problem (Goodman-Bacon 2021, Callaway and Sant'anna 2021).

However, given the plausibly exogenous change in lead exposure outlined in Sect. 2, we can identify the effect of the lead reduction if we are willing to accept certain assumptions. Following Wooldridge (2021), the main parallel trends assumption we rely on is a conditional common trends assumption.

4.2.1 Conditional Common Trends, Staggered Treatment (CCTS)

Following Athey and Imbens (2022) and Wooldridge (2021), we define the outcome for the never-treated group as $y_{it}(\infty)$ (omitting the postcode signifier *j* for clarity). Given this, we formally state the CCTS assumption as:

$$E\left[y_{it}(\infty) - y_{i1}(\infty)\, d_{ir}, x_{ij}\right] = E\left[y_{it}(\infty) - y_{i1}(\infty)\, x_{ij}\right], r = q, \ldots, T; t = 2, \ldots, T. \tag{6}$$

The assumption states that for every cohort the trend in outcome if never treated is unrelated to being in any treatment cohort, after conditioning on the covariates. This can be tested to a degree, see Sect. 5.

Under-5 mortality is a binary outcome and it is likely the CCTS assumption is violated. Therefore we replace this assumption when estimating the effect on under-5 mortality.

4.2.2 Conditional Parallel Relative Trends, Staggered Treatment (CPRTS)

$$\frac{E\left[y_{it}(\infty)\,|d_{ir},\ x_i\ \right]}{E\left[y_{i1}(\infty)\,|d_{ir},\ x_i\ \right]} = \frac{E\left[y_{it}(\infty)\,|x_i\right]}{E\left[y_{i1}(\infty)\ |x_i\ \right]},\ t = 2, \ldots, T,\ r = q, \ldots, T \tag{7}$$

The ratio of average outcome if never-treated at time *t* compared to the first period average outcome only depends on the covariates. There is no selection into or out of treatment.

4.3 Estimation

There are now several estimation methods to deal with difference-in-differences when there is staggered adoption (see Roth et al., 2023 for a recent review). Following Harmon (2023), these can be divided into two groups: "subgroup" types and "imputation" types. "subgroup"

types use a particular subset of the data to construct a "clean" control group for a particular treatment group. For example, all not yet treated units before the treatment for a particular cohort starts, or all never-treated units. "imputation" types instead will impute counterfactuals with either inverse probability weighting or regression adjustment. For our main estimates, we use the Wooldridge (2021) two-way Mundlak estimation method, which is an "imputation" type. We primarily estimate using the Wooldridge (2021) two-way Mundlak because it can simultaneously handle a repeated cross-section and the use of a quasi-maximum likelihood logistic model that is needed due to the CPRTS assumption. However, as a robustness check we also include alternative "subgroup" type estimators in the appendix. Note that these alternatives use the CCTS assumption for mortality rather than the CPRTS assumption used for the two-way Mundlak and both cannot be true at the same time.

For birthweights we use a linear model and the CCTS assumption and estimate with a two-way Mundlak regression with robust errors clustered at postcode sector level following Wooldridge (2021).

$$
\begin{aligned}
y_{it} = \eta +, \boldsymbol{x_i \kappa} &+ \sum_{r=q}^{T} \lambda_r d_{ir} + \sum_{r=q}^{T} \zeta_r (d_{ir} \times, x_i) \\
&+ \sum_{s=2}^{T} \theta_s f s_t + \sum_{s=2}^{T} (f s_t \times \boldsymbol{x_i})\, \boldsymbol{\pi_t} \\
&+ \sum_{r=q}^{T} \sum_{s=r}^{T} \tau_{rt} \left(d_{ir} \times p_{irt} \times f s_t\right) \\
&+ \sum_{r=q}^{T} \sum_{s=r}^{T} \left(d_{ir} \times p_{irt} \times f s_t \times \dot{\boldsymbol{x}}_{\boldsymbol{ir}}\right) \boldsymbol{\rho_{rt}} + u_{it}
\end{aligned}
\tag{8}
$$

Where η is the intercept and fs_t are indicators for every time period that equal 1 when $s = t$. The d_{ir} variable is the cohort indicator as in the CCTS assumption. Next, p_{irt} is a post-treatment indicator. It equals 1 for every period after that group first received treatment. Formally, $p_{irt} = 1 \forall t > q_r - 1$, where q_r is the period which the group first received treatment. For example, given $t = 1, 2, 3$ and group 1 was first treated in period 2, then $q_1 = 2$. If group 2 first received treatment in period 3 then $q_2 = 3$. Finally, $\dot{x}_{ir}$ is the deviation from the cohort average for individual i.

If we wish to allow for more heterogeneity in time trends, we can model the time trend as simply $d_{ir} \times t$. This allows for heterogeneity in time trends and also allows us to test the common trends assumption with a Wald test jointly on all the coefficients of $d_{ir} \times t$.

For under-5 mortality, we use the CPRTS assumption and estimate with a quasi-maximum likelihood logistic two-way Mundlak regression.

$$
\begin{aligned}
E\left[y_{it} d_{ir}, \boldsymbol{x_i}\right] = & \boldsymbol{\Lambda}[\eta+, \boldsymbol{x_i \kappa} + \sum_{r=q}^{T} \lambda_r d_{ir} + \\
& \sum_{r=q}^{T} \zeta_r (d_{ir} \times, x_i) + \sum_{s=2}^{T} \theta_{\mathrm{s}} f s_t + \\
& \sum_{s=2}^{T} (f s_t \times x_i) \pi_t + \\
& \sum_{r=q}^{T} \sum_{s=r}^{T} \tau_{rt} \left(d_{ir} \times p_{irt} \times f s_t\right) + \\
& \sum_{r=q}^{T} \sum_{s=r}^{T} (d_{ir} \times p_{irt} \times f s_t \times \dot{x}_{ir}) \rho_{rt}
\end{aligned} \tag{9}
$$

Where $\boldsymbol{\Lambda}$ represents the logistic function. The treatment effect estimated is an average partial effect (APE) of being treated. That is, we estimate the model and then take the coefficients applicable for a particular year and cohort. We take the expected value of the values with the treatment variable minus the values without including the treatment variable. We obtain standard errors for the APE with bootstrapping.

5 Results

We first plot the mean birthweight for Glasgow, Edinburgh and the control group of surrounding areas in Fig. 7. In all groups there is a clear upward trend. The Edinburgh average closely tracks the control group, while the Glasgow average remains below both at all times, and there is no convergence even after the 1978 and 1989 treatment. Both the Edinburgh and Glasgow averages are more volatile than the control group. The trends appear similar for all groups. There is no clear treatment effect to be seen in the raw averages, but this may be due to differences in group characteristics that affect the treatment effect. We explore this in sect. 5.1.

Similarly, in Fig. 8 we plot under-5 mortality rates for each group. The trends are again similar, but the percentage for Glasgow and Edinburgh is more volatile. No clear treatment effect is visible in the raw data, but once again this may be due to heterogeneity in group characteristics and therefore selection bias. We move on now to the difference-in-differences estimation.

5.1 Two-Way Mundlak Regressions

Table 2 presents estimates for the effect of the treatment 1 on both birthweights (columns 1 and 2), and mortality (columns 3 and 4). Standard errors are clustered by postcode sector for the ATTs in columns 1 and 2. For the APEs in Columns 3 and 4 we use a bootstrap for the standard errors. In column 1 we see the overall average effect on birthweights, without covariates, which has a small negative point estimate. This is the opposite sign from what we would expect, but the estimate is not statistically significant. The group level estimates

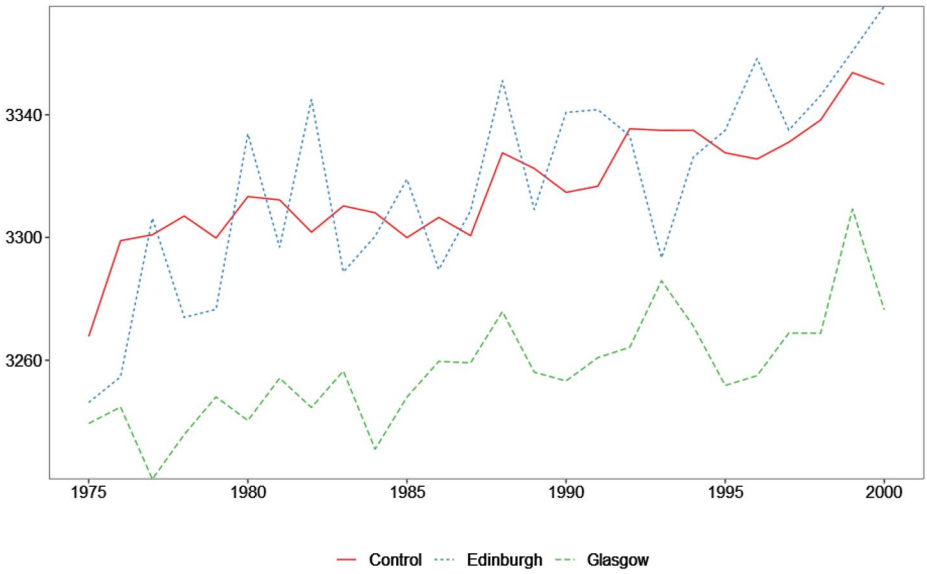

Fig. 7 Average birthweight by treatment cohort, grams notes: chart shows the mean birthweight in grams of each birth in Edinburgh, Glasgow and the control group

Fig. 8 Under-5 mortality rate by treatment cohort notes: chart shows the total under-5 mortality rates, the probability of all deaths and recorded non-viable pregnancies, including stillbirths and spontaneous abortions, for all births in each birth in Edinburgh, Glasgow and the control group

Table 2 Effect of treatment 1 by cohort (two-way mundlak method)

Dependent Variable		Birthweights	Birthweights	Under-5 Mortality	Under-5 Mortality
		(1)	(2)	(3)	(4)
		ATT	*ATT*	*APE*	*APE*
Overall	*Estimate*	−3.5	−14.5***	−0.0017**	−0.0017
	SE	(1.9)	(2.5)	(0.0008)	(0.0012)
	MDE	4.75	6.25	−0.0020	−0.0030
Glasgow	*Estimate*	−9.2***	−19.2***	−0.0030***	−0.0018
	SE	(2.2)	(3.1)	(0.0010)	(0.0015)
	MDE	5.5	7.75	−0.0025	−0.0038
Edinburgh SW	*Estimate*	13.5***	−4.7	0.0030***	−0.0002
	SE	(4.8)	(4.4)	(0.0010)	(0.0009)
	MDE	12.0	11.0	−0.0025	−0.0023
Edinburgh NE	*Estimate*	13.7**	10.3	0.0001	−0.0040***
	SE	(5.8)	(5.9)	(0.0013)	(0.0014)
	MDE	14.5	14.8	−0.0033	0.0035
Observations - Overall		618,108	612,483	646,893	641,004
Clusters - Overall		398	398	398	398
Area Covariates		*No*	*Yes*	*No*	*Yes*
Mother Covariates		*No*	*Yes*	*No*	*Yes*
Infant Gender Fixed Effect		*No*	*Yes*	*No*	*Yes*
Baseline Mean		3253.3	3253.3	0.0079	0.0079
(Standard Deviation)		(548.3)	(548.3)	(0.0884)	(0.0884)

Notes: Table shows cohort specific treatment effects from lime dosing using two-way Mundlak regressions. ATT = Average Treatment on the Treated estimate. APE = Average Partial Effect estimate. Birthweights is the birthweight of the child in grams. Under-5 Mortality is the probability of all deaths and recorded non-viable pregnancies, including stillbirths and spontaneous abortion. Birthweight regression estimates are rounded to 1 decimal place, mortality estimates are rounded to 3 decimal places. SE = standard error. For birthweight regressions we use errors clustered by postcode sector, for mortality we use bootstrapped errors. MDE = Minimal detectable effect, calculated as 2.5*SE. We calculate MDE for an increase in birthweight and a decrease in mortality. Significance Codes: ***: 0.01, **: 0.05, *: 0.1

are significant, but Glasgow has a small negative point estimate, while both the Edinburgh water areas have positive estimates. Column 2 includes covariates in the model, as described in Sect. 3. The overall estimate is again negative, the opposite sign from expected, while it is statistically significant. This is mainly driven by the negative estimate from Glasgow. This time the Edinburgh SW estimate is also negative, but not significant, while the Edinburgh NE estimate is again positive. The significance of some estimates and not others, and the differing signs of the estimates may mean that the design is underpowered to detect the true effect. We therefore include an estimate of the minimal detectable effect (MDE). This is estimated as 2.5 times the standard error (Rainey 2025). This estimates the smallest effect size that the study had 80% power to detect (in the positive direction for birthweight and negative for mortality). The smallest birthweight effect size in the Clay et al. (2024) review is 24 g. this is well within the MDE for all our estimations. Therefore, if our study is not well-powered enough to detect the effect, the effect in this setting must be much smaller than that found in the studies in Clay et al. (2024).

In column 3 we show the estimates for the effects on under-5 mortality without including covariates. The overall average partial effect is negative, as we would expect, and significant at the 5% confidence level. However, when we add covariates in column 4 it is not significant. We see a similar pattern for the Glasgow group, as this is the main cause of the sign for the overall estimate. The Edinburgh SW estimate is positive in column 3, implying the lead reduction increased mortality, but becomes negative and not significant in column 4 when we add covariates. The Edinburgh NE estimate is negative in both columns but only significant with covariates. When we look at the MDE, we estimate we had 80% power to detect effect sizes of −0.0038 to −0.0023, i.e. 2–4 fewer deaths in every thousand births due to the lead reduction.

In Table 3, we only examine years for which all of the treatment areas already had treatment 1, but not yet treatment 2. Glasgow was treated in 1989, and all Edinburgh water areas in 1991. In columns 1 and 2 we see the estimates of the effect on birthweight of treatment 2. All point estimates are small and not significant, save the Edinburgh estimate in column 2 where we include covariates, but this is negative. The MDE is at most 18 g.

In column 3, we estimate the effect on mortality without covariates. The overall estimate is positive, but not significant. The Glasgow estimate is negative and not significant, while the Edinburgh effect has the opposite from the expected sign and is significant. In column 4, when we include covariates, the overall APE becomes negative but remains not significant. The Glasgow estimate remains negative but is significant, while the Edinburgh point estimate remains positive but is no longer significant. The MDE varies from −0.0015 to −0.0028.

5.2 Pre-Trends

The CCTS and CPRTS (or parallel trends) assumptions are crucial for identification of an effect in our difference-in-differences design. We therefore examine these closely in this section. First, we plot event studies. These are estimated in a similar way to Eq. (8) and (9), but we include the pre-treatment years as well for each cohort. In Fig. 9, we show event study estimates of treatment 1 using two-way Mundlak regressions for the effect on birthweight. The cohort-year interactions are shown as years relative to treatment, so Edinburgh NE, which was treated later, has more pre-treatment periods than Glasgow and Edinburgh

Table 3 Effect of treatment 2 by cohort (two-way mundlak method)

Dependent Variable		Birthweights	Birthweights	Under-5 Mortality	Under-5 Mortality
		(1)	(2)	(3)	(4)
		ATT	*ATT*	*APE*	*APE*
Overall	*Estimate*	1.7	−3.3	0.0006	−0.0006
	SE	(3.4)	(3.1)	(0.0004)	(0.0006)
	MDE	8.5	7.8	−0.0010	−0.0015
Glasgow	*Estimate*	3.7	2.7	−0.0004	−0.0013**
	SE	(3.1)	(3.0)	(0.0005)	(0.0006)
	MDE	7.8	7.5	−0.0013	−0.0015
Edinburgh	*Estimate*	−1.4	−12.3**	0.0020***	0.0005
	SE	(7.2)	(6.2)	(0.0007)	(0.0011)
	MDE	18.0	15.5	−0.0018	−0.0028
Observations - Overall		362,598	287,326	362,687	359,071
Clusters - Overall		391	391	391	391
Area Covariates		*No*	*Yes*	*No*	*Yes*
Mother Covariates		*No*	*Yes*	*No*	*Yes*
Infant Gender Fixed Effect		*No*	*Yes*	*No*	*Yes*
Baseline Mean		3288.4	3288.4	0.0087	0.0087
(Standard Deviation)		(586.4)	(586.4)	(0.0927)	(0.0927)

Notes: Table shows cohort specific treatment effects from orthophosphate dosing using two-way Mundlak regressions. ATT = Average Treatment on the Treated estimate. APE = Average Partial Effect estimate. Birthweights is the birthweight of the child in grams. Under-5 Mortality is the probability of all deaths and recorded non-viable pregnancies, including stillbirths and spontaneous abortion. Birthweight regression estimates are rounded to 1 decimal place, mortality estimates are rounded to 3 decimal places. SE = standard error. For birthweight regressions we use errors clustered by postcode sector, for mortality we use bootstrapped errors. MDE = Minimal detectable effect, calculated as 2.5*SE. We calculate MDE for an increase in birthweight and a decrease in mortality. Significance Codes: ***: 0.01, **: 0.05, *: 0.1

SW. We can see, for every year and cohort, the 95% intervals cover zero. This can be taken as evidence there are no pre-trends, but the pre-treatment point estimates for Edinburgh NE do tend to rise just before treatment, indicating there could be a pre-trend for that cohort. We investigate further by performing the test of model assumptions suggested by Wooldridge (2021) for the 2- way Mundlak. This is a test of both the common trends and no anticipation assumptions. It is a Wald test where we include in the regression a continuous time variable (i.e from 1 to T) in the regression and perform a test of joint null effects on each cohort and time interaction. We reject the hypothesis of no anticipation or no common trends if the Wald test fails and finds the coefficients to be jointly statistically significant. In the case of the birthweight regression without covariates the p-value for the test is 0.01, so we reject the hypotheses of no anticipation or common trends, suggesting pre-trends. However, when we condition on covariates we do not reject the conditional no anticipation and conditional common trends assumptions (p-value 0.17).

Figure 10 plots the trends for treatment 2. We see again all the coefficients for Glasgow have intervals that cover zero, and all but one estimate for Edinburgh covers zero, with none of those pre-treatment. Again, this suggests no pre-trends. The Wald test leads to us not rejecting common trends and no anticipation without covariates (p-value 0.66) or with them (p-value 0.77).

In Fig. 11 we show event study APEs for each cohort year for the effect on mortality of treatment 1. All pre-treatment intervals cover zero, suggesting no clear evidence of pre-

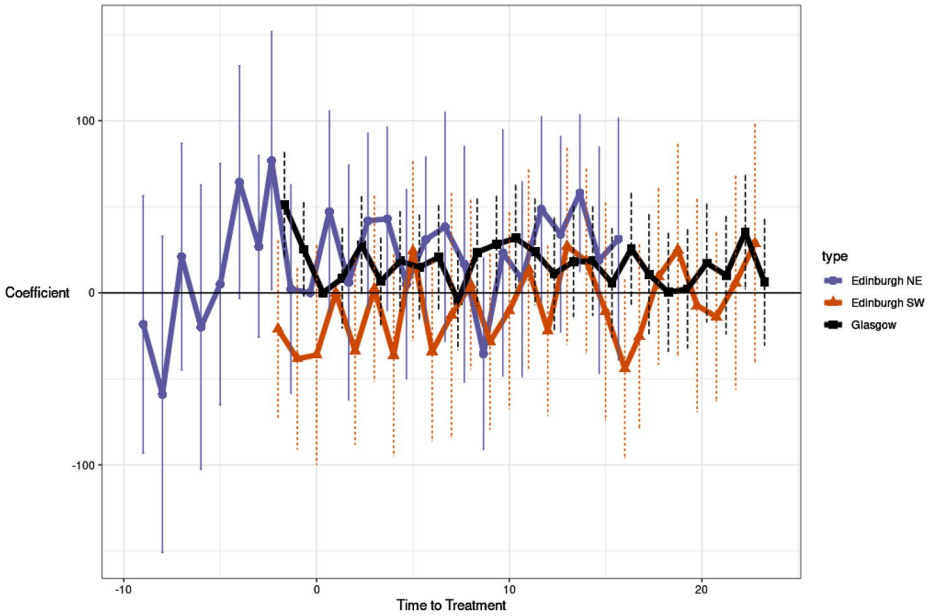

Fig. 9 Effect of treatment 1 on birthweights (event study with two-way mundlak estimator) notes: table shows estimated treatment effects on different groups of lead reduction due to lime-dosing in Glasgow and Edinburgh compared to a never-treated control group. The horizontal axis labelled as 'time to treatment' shows the years relative to treatment, as a result the pre-treatment period differs across each area. We use standard errors clustered by postcode sector. In all cases the estimates are the average treatment on the treated for that year on that group birthweights is the birthweight in grams for a child

trends. Glasgow has mostly negative point estimates after treatment while Edinburgh SW and Edinburgh NE mostly positive. The two Edinburgh water treatment areas have almost all intervals covering zero while Glasgow does have some intervals that are statistically significant. When we look at the effect on mortality of treatment 2 in Fig. 12, we see perhaps some evidence for a negative pre-trend in Edinburgh. Point estimates in both Glasgow and Edinburgh tend to be negative but all except one post-treatment interval cover zero. Again, we conclude there is no strong evidence for an effect on under-5 mortality in these event studies. Estimates for Edinburgh are typically positive, while Glasgow shows a reduction in deaths after treatment 1. When we carry out the Wald test for pre-trends and no anticipation we do not reject the hypotheses of no anticipation or common trends without covariates (p-value 0.72), or with covariates (p-value 0.26).

Figure 12 plots the event study of mortality effects after treatment 2. Glasgow does not show evidence for pre-trends or an effect, but Edinburgh does. We again perform a Wald test and do not reject the hypotheses of no anticipation or common trends without covariates (p-value 0.73), or with covariates (p-value 0.19).

As a robustness check, we include results from a matrix completion difference-in-difference estimation in appendix tables A.10 and A.11. Matrix completion can be thought of as a generalization of synthetic control methods (Athey et al. 2021) and is used to impute the counterfactual trend based on latent patterns in the pre-treatment values of all units. Iden-

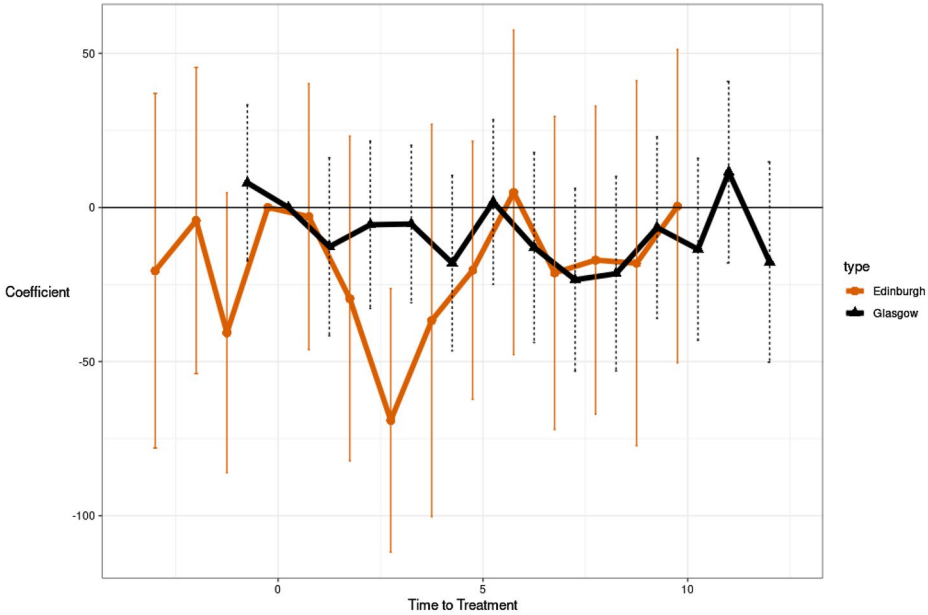

Fig. 10 Effect of treatment 2 on birthweights (event study with two-way mundlak estimator) notes: table shows estimated treatment effects on different groups of lead reduction due to orthophosphate in Glasgow and Edinburgh compared to a never-treated control group. The horizontal axis, labelled as 'time to treatment'. shows the years relative to treatment, as a result the pre-treatment period differs across each area we use standard errors clustered by postcode sector. In all cases the estimates are the average treatment on the treated for that year on that group birthweights is the birthweight in grams for a child

tification therefore relies on weaker assumptions than the parallel trends assumption in (6). The results in the appendix are very similar to the main results here.

5.3 Further Robustness Checks

In the online appendix, we carry out a wide variety of further robustness checks. These confirm our main results. These include matrix completion estimates that use the data to generate a fitted counterfactual for the treated group, rather than just the control units counterfactual, alternative staggered difference-in-differences estimators, comparing high and low lead areas in Glasgow, and alternative two-way Mundlak specifications. The results are qualitatively similar in all cases.

6 Discussion and Conclusions.

We examined the effect of policies to reduce lead pollution in drinking water on health outcomes, (birthweights and under-5 mortality) in Glasgow and Edinburgh in the 20th century. We use a setting with plausibly exogenous staggered treatments, and therefore utilise a difference-in-differences design that accounts for the staggered nature of the treatment. Across a variety of specifications, and with multiple robustness checks, we do not find

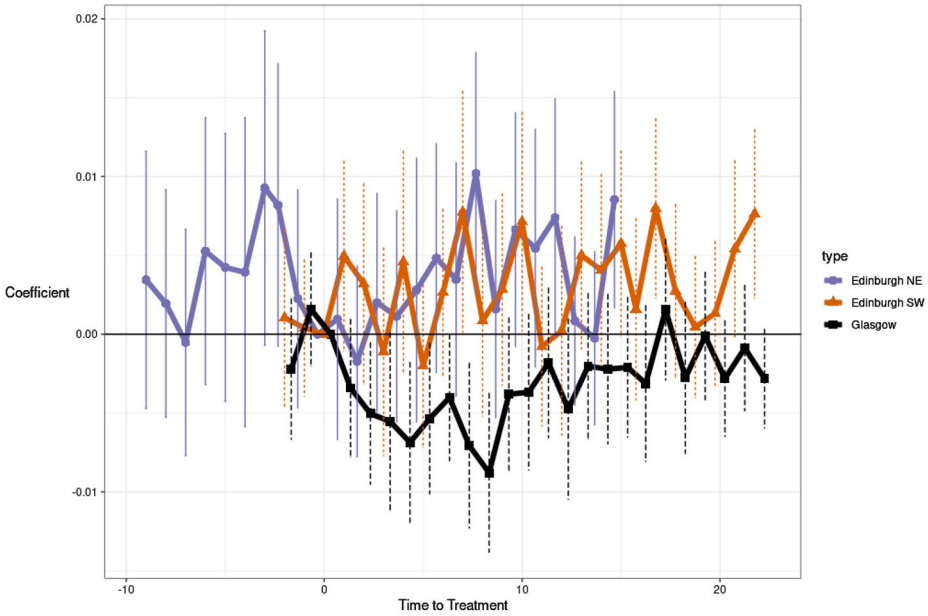

Fig. 11 Effect of treatment 1 on under-5 mortality (event study with two-way mundlak estimator) notes: table shows estimated treatment effects on different groups of lead reduction due to lime-dosing in Glasgow and Edinburgh compared to a never-treated control group. The horizontal axis labelled as 'time to treatment' shows the years relative to treatment, as a result the pre-treatment period differs across each area we use bootstrapped standard errors. In all cases the estimates are the average partial effect for that year on that group. Mortality is the probability of all deaths and recorded non-viable pregnancies, including stillbirths and spontaneous abortion for under-5s

consistent evidence of a beneficial effect of lead water pollution reduction on infant health outcomes. These findings contribute to the existing literature on the impact of lead and birth outcomes, using a much larger sample, and plausibly exogenous variation in lead, and high levels of lead.

Our study has two main limitations. Firstly, the treatment variation is at a postcode sector level rather than an individual level and we observe most of our sample only once. This means that our confidence intervals are less precise than if the treatment was exogenously applied at an individual level. However, this is common in the literature that examines the effect of lead with quasi-experiments, because lead cannot be ethically given as part of a randomised control trial. We therefore must rely on coarser treatment variation. Secondly, as stated above our MDE means we cannot rule out socially significant effects on mortality, although we can rule this out for birthweight. Note, however, that both limitations are common to most studies in this field, and our setting nonetheless offers unusually strong and plausibly exogenous variation in lead exposure.

It is worth thinking about our results in light of the plausible mechanisms laid out in Sect. 2. It may simply be that there is no effect on these health outcomes at the level of lead in drinking water in our setting. It is possible that birthweight and under-5 mortality are not the most sensitive indicators of lead toxicity. Lead may be more commonly associated with long-term developmental effects and behavioural problems, which are difficult to detect in birth or early childhood outcomes. This conclusion is consistent with the best evidence

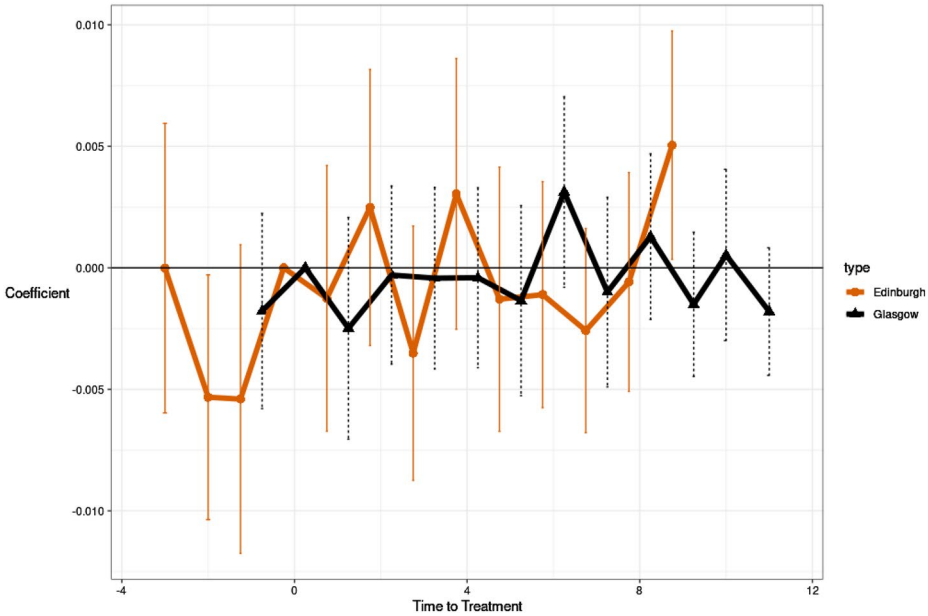

Fig. 12 Effect of treatment 2 on under-5 mortality (event study with two-way mundlak estimator) notes: table shows estimated treatment effects on different groups of lead reduction due to orthophosphate in Glasgow and Edinburgh compared to a never-treated control group. The horizontal axis labelled as 'time to treatment' shows the years relative to treatment, as a result the pre-treatment period differs across each area we use bootstrapped standard errors. In all cases the estimates are the average partial effect for that year on that group. Mortality is the probability of all deaths and recorded non-viable pregnancies, including stillbirths and spontaneous abortion for under 5s

provided by the other large quasi-which find no effect on the most acute outcomes using administrative data from Sweden (Grönqvist et al. (2020).

However, there are also alternative explanations.

The first possibility is that an effect does exist, but even with our sample size, given the low annual incidence of child deaths – often less than 1% – it may not be sufficient to detect it with precision. Our estimates of the MDE imply we had 80% power to estimate quite small effects on birthweight, but the same cannot be said for mortality, where we had 80% power to detect a reduction of 1–3 deaths per thousand due to the lower lead levels. This could indicate that the water treatments did lower mortality, but by less than the study was powered to detect.

Secondly, an effect may be present, but only at levels of lead exposure higher than those observed in our sample. It may be the level of exposure in our sample was simply too low to produce a detectable average effect. Lead pollution at high levels causes very obvious and extreme health problems, but at lower levels it is much harder to see acute lead poisoning symptoms. However, the water and blood lead levels in our sample, especially for Glasgow, are considerably higher than in other studies that do find an effect.

A third possibility is that the literature on lead and child mortality may be affected by publication bias. The lead-crime literature, for example, has been shown to suffer from publication bias (Higney et al. 2022). It can be the case that even if every paper estimated an unbiased causal effect, in the presence of publication bias we would still be left with a

bias in the literature. It is beyond the scope of this paper to estimate if there is publication bias for the lead pollution and birth outcomes literature, but it is a problem across empirical science and cannot be ruled out here.

Fourthly, it is possible that lead exposure has a negative effect on children's health outcomes, but a mediating factor reduces this impact. The recent review of Clay et al. (2024) shows most quasi-experimental studies find an effect. However, all the quasi-experimental studies which find an effect are from North America, while our paper and Grönqvist et al. (2020) do not find an effect and both use data from Northern Europe. One potential difference is nutrition, specifically high milk intake. The UK and Northern Europe has some of the highest milk consumption rates in the world (FAO, 2022), more than two and a half times the global average (FAO [WWW Document] 2022). In the 1930s, the National Milk Scheme in Scotland promoted milk consumption and provided targeted subsidies for mothers of children under five years old. This, along with the provision of free milk in schools for much of the twentieth century (Krebs 2019), led to a significant increase in milk consumption. Studies have shown that high milk intake is associated with lower blood lead levels (Chuang et al. 2004). Thus, it is possible that better nutrition, particularly high milk consumption, may have played a role in reducing the impact of lead on under-5 mortality in Northern Europe compared to elsewhere.

Finally, lime dosing and orthophosphate treatment are operationally simple and far less costly than requiring households to replace lead plumbing. Even if the health benefits are modest, these interventions may still be highly cost-effective. A formal cost-benefit analysis could help quantify the value of promoting such low-cost public health measures. We suggest that future research should investigate the mediating pathways between lead and harmful outcomes, to better understand effective treatments.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10640-025-01041-6.

Acknowledgements This research was funded by the Economic and Social Research Council (ESRC), grant number 2117499. We thank Jeffrey Wooldridge, Nils Braakmann, Markus Gehrsitz, Danny Campbell and Hector Gutierrez Rufrancos for their comments on this paper. We also thank participants at the European Association of Environmental and Resource Economists (EAERE) conference and the Scotland and Northern England Conference in Applied Microeconomics. Any remaining errors are our own.

Author Contributions Conceptualization: Anthony Higney, Nick Hanley, Mirko Moro. Methodology: Anthony Higney, Nick Hanley, Mirko Moro. Formal analysis: Anthony Higney. Writing – original draft: Anthony Higney. Writing – review & editing: Nick Hanley, Mirko Moro

Declarations

Ethics Approval and Consent to Participate Project received ethics approval from the University of Stirling ethics panel 22,082,017.

Conflict of Interest The authors declare that they have no conflict of interest.

Open Access This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted

by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by/4.0/.

References

2000) Public health implications of new guidelines for lead in drinking water: a case study in an area with historically high water lead levels. In: Watt GCM, Britton A, Gilmour HG. Food Chem. Toxicol, vol 38. Moore, M.R., Murray, G.D., Robertson, S.J, pp S73–S79. https://doi.org/10.1016/S0278-6915(99)00137-4

Akoumianaki I (2017) Lead in drinking water: public health, mitigation, and economic perspectives. CD2016_03. Available online at: https://doi.org/crew.ac.uk/publications

Al-Saleh I, Khalil MA, Taylor A (1995) Lead, erythrocyte protoporphyrin, and hematological parameters in normal maternal and umbilical cord blood from subjects of the Riyadh region, Saudi Arabia. Arch Environ Health 50:66–73. https://doi.org/10.1080/00039896.1995.9955014

Angell NF, Lavery JP (1982) The relationship of blood lead levels to obstetric outcome. Am J Obstet Gynecol 142:40–46. https://doi.org/10.1016/S0002-9378(16)32282-7

Athey S, Bayati M, Doudchenko N, Imbens G, Khosravi K (2021) Matrix completion methods for causal panel data models. J Am Stat Assoc 116(536):1716–1730. https://doi.org/10.1080/01621459.2021.1891924

Athey S, Imbens GW (2022) Design-based analysis in difference-in-differences settings with staggered adoption. J Econom 226:62–79. https://doi.org/10.1016/J.JECONOM.2020.10.012

Azayo MM, Manji K, Kalokola F (2009) Lead levels in women at delivery at the Muhimbili National hospital: a public health problem. J Trop Pediatr 55:138–139. https://doi.org/10.1093/TROPEJ/FMN085

Behrman JR, Rosenzweig MR (2004) Returns to birthweight. Rev Econ Stat 86:586–601. https://doi.org/10.1162/003465304323031139

Bellinger DC (2005) Teratogen update: lead and pregnancy. Birth defects res. Part a clin. Mol. Teratol 73:409–420. https://doi.org/10.1002/BDRA.20127

Bornschein RL, Grote J, Mitchell T, Succop PA, Dietrich KN, Krafft KM, Hammond PB (1989) Effects of prenatal lead exposure on infant size at birth. Lead Expo. Child Dev 307–319. https://doi.org/10.1007/978-94-009-0847-5_18

Brubaker CJ, Schmithorst VJ, Haynes EN, Dietrich KN, Egelhoff JC, Lindquist DM, Lanphear BP, Cecil KM (2009) Altered myelination and axonal integrity in adults with childhood lead exposure: a diffusion tensor imaging study. Neurotoxicology. https://doi.org/10.1016/j.neuro.2009.07.007

Callaway B, Goodman-Bacon A, Sant'anna PHC (2021) Difference-in-differences with a continuous treatment. arXiv. https://arxiv.org/abs/2107.02637

Callaway B, Sant'anna PHC (2021) Difference-in-differences with multiple time periods. J Econom 225:200–230. https://doi.org/10.1016/J.JECONOM.2020.12.001

Calonico S, Cattaneo MD, Farrell MH (2020) Optimal bandwidth choice for robust bias-corrected inference in regression discontinuity designs. Econom J 23(2):192–210. https://doi.org/10.1093/ectj/utz022

Carstairs V, Morris R (1991) Deprivation and health in Scotland. Aberdeen University Press, Aberdeen

Cattaneo MD, Titiunik R (2022) Regression discontinuity designs. Annual Review Of Economics 14(1):821–851

Cecil KM, Brubaker CJ, Adler CM, Dietrich KN, Altaye M, Egelhoff JC, Wessel S, Elangovan I, Hornung R, Jarvis K, Lanphear BP (2008) Decreased brain volume in adults with childhood lead exposure. PLOS Med. https://doi.org/10.1371/journal.pmed.0050112

Chatterji P, Kim D, Lahiri K (2014) Birth weight and academic achievement in childhood. Health Econ 23:1013–1035. https://doi.org/10.1002/HEC.3074

Chuang HY, Tsai SY, Chao KY, Lian CY, Yang CY, Ho CK, Wu TN (2004) The influence of milk intake on the lead toxicity to the sensory nervous system in lead workers. Neurotoxicology 25:941–949. https://doi.org/10.1016/J.NEURO.2004.04.003

Clay K, Hollingsworth A, Severnini E (2024) The impact of lead exposure on fertility, infant mortality, and infant birth outcomes. Rev Environ Econ Policy 18(2):301–320. https://doi.org/10.1086/730854

Clay K, Troesken W, Haines M (2014) Lead and mortality. Rev Econ Stat 96:458–470. https://doi.org/10.1162/REST_A_00396

Comber S, Cassé F, Brown B, Martin J, Hillis P, Gardner M (2011) Phosphate treatment to reduce plumbosolvency of drinking water also reduces discharges of copper into environmental surface waters. Water Environ J 25:266–270. https://doi.org/10.1111/J.1747-6593.2010.00219.X

Dave DM, Yang M (2022) Lead in drinking water and birth outcomes: a tale of two water treatment plants. J Health Econ 84:102644. https://doi.org/10.1016/J.JHEALECO.2022.102644.

Dorea JG, Donangelo CM (2006) Early (in uterus and infant) exposure to mercury and lead. Clin Nutr 25:369–376. https://doi.org/10.1016/J.CLNU.2005.10.007

Doucouliagos H, Stanley TD (2009) Publication selection bias in minimum-wage research? A meta-regression analysis. Br. J. Ind. Relations 47:406–428. https://doi.org/10.1111/J.1467-8543.2009.00723.X

Edwards M (2014) Fetal death and reduced birth rates associated with exposure to lead-contaminated drinking water. Environ Sci Technol 48:739–746. https://doi.org/10.1021/ES4034952/SUPPL_FILE/ES4034952_SI_001.PDF

Ericson B, Hu H, Nash E, Ferraro G, Sinitsky J, Taylor MP (2021) Blood lead levels in low-income and middle-income countries: a systematic review. The Lancet Planet Health 5(3):e145–e153. https://doi.org/10.1016/S2542-5196(21)00281-3. PMID: 33713615

EU (2020) Directive (EU) 2020/2184 of the European Parliament and of the council of 16 December 2020 on the quality of water intended for human consumption. Off J Eur Communities. https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A32020L2184

Falcón M, Viñas P, Luna A (2003) Placental lead and outcome of pregnancy. Toxicology 185:59–66. https://doi.org/10.1016/S0300-483X(02)00589-9

FAO [WWW Document] (2022) https://www.fao.org/faostat/en/#data/FBS (accessed 12.31.22.

Feigenbaum JJ, Muller C (2016) Lead exposure and violent crime in the early twentieth century. Explor Econ Hist 62:51–86. https://doi.org/10.1016/j.eeh.2016.03.002

Gazze L, Heissel L, ISSN 0095–0696 (2021) Infrastructure upgrades and lead exposure: do cities face trade-offs when replacing water mains? Journal Of Environmental Economics And Management 108:102472. https://doi.org/10.1016/j.jeem.2021.102472

Gazze L, Persico C, Spirovska S (2021) The long-run spillover effects of pollution: how exposure to lead affects everyone in the classroom. SSRN Electron J. https://doi.org/10.2139/SSRN.3842757

GBD (2020) Global Burden of Disease (GBD 2019) | Institute for health metrics and evaluation [WWW Document]. Glob. Burd. Dis. - IMHE. https://www.healthdata.org/gbd/2019. Accessed 26 Jan 2022

Gechter M, Meager (2022) Combining experimental and observational studies in meta-analysis: a debiasing approach. Working paper

Goldenberg RL, Culhane JF (2007) Low birth weight in the United States. Am J Clin Nutr 85(2):584S–590S. https://doi.org/10.1093/ajcn/85.2.584S

Golmohammadi T, Ansari MR, Nikzamir A, Abhari RS, Elahi SB (2007) The effect of maternal and fetal lead concentration on birth weight: polluted versus non-polluted areas of Iran. Medicine (baltimore) 74–78

Goodman-Bacon A (2021) Difference-in-differences with variation in treatment timing. J Econom 225:254–277. https://doi.org/10.1016/J.JECONOM.2021.03.014

Grönqvist H, Nilsson JP, Robling P-O (2020) Understanding how low levels of early lead exposure affect Children's life trajectories. J Polit Econ 128(9):3376–3433. https://doi.org/10.1086/708725

Grossman DS, Slusky DJG (2019) The impact of the flint water crisis on fertility. Demography 56:2005–2031. https://doi.org/10.1007/S13524-019-00831-0

Hall A, Ransom WB (1906) PLUMBISM FROM THE INGESTION OF DIACHYLON AS AN ABORTIFACIENT. Br Med J 1:428. https://doi.org/10.1136/BMJ.1.2356.428

Hansard HC Deb (1990 May 23) Lead in water (Scotland) [Hansard] (173):cc389–96. https://api.parliament.uk/historic-hansard/commons/1990/may/23/lead-in-water-scotland

Hayes CR, Hydes OD (2012) UK experience in the monitoring and control of lead in drinking water. J Water Health 10:337–348. https://doi.org/10.2166/WH.2012.210

Health Canada (2019) Guidelines for Canadian drinking water quality: guideline technical document - lead. Government of Canada. WWW Document.https://www.canada.ca/en/health-canada/services/publications/healthy-living/guidelines-canadian-drinking-water-quality-guideline-technical-document-lead.html (accessed 08.06.25)

He Q, Ji X (2021) The labor productivity consequences of exposure to particulate matters: evidence from a Chinese National panel survey. Int J Environ Res Public Health 18:12859. https://doi.org/10.3390/IJERPH182312859

Higney A, Hanley N, Moro M (2022) The lead-crime hypothesis: a meta-analysis. Reg Sci Urban Econ 97:103826. https://doi.org/10.1016/j.regsciurbeco.2022.103826

Higney A, Hanley N, Moro M, Gasteen A (2025) The impact of lead pollution on human capital formation: size of dose matters. Working paper. Available at: https://anthonychigney.github.io/home/assets/images/leadEducation20220129.pdf

Hollingsworth A, Rudik I (2021) The effect of leaded gasoline on elderly mortality: evidence from regulatory exemptions. Am Econ J Econ Policy 13:345–373. https://doi.org/10.1257/POL.20190654

Krebs C (2019 2019) Lead poisoning and intelligence: a search for cause and effect in the Scottish mental surveys. J. Environ. Public Health. https://doi.org/10.1155/2019/8980604

Law CM (2002) Significance of birth weight for the future. Arch Dis Child - Fetal Neonatal Ed 86:F7–F8. https://doi.org/10.1136/FN.86.1.F7

Macintyre C, Fulton M, Hepburn W, Yang S, Raab G, Davis S, Heap M, Halls D, Fell G (1998) Changes in blood lead and water lead in Edinburgh. An eight year follow-up to the Edinburgh lead study. Environ Geochem Health 20:157–167. https://doi.org/10.1023/A:1006593225113/METRICS

Marcus DK, Fulton JJ, Clarke EJ (2010) Lead and conduct problems: a meta-analysis. J Clin Child Adolesc Psychol. https://doi.org/10.1080/15374411003591455

McMichael AJ, Vimpani GV, Robertson EF, Baghurst PA, Clark PD (1986) The port pirie cohort study: maternal blood lead and pregnancy outcome. J Epidemiol Community Heal 40:18–25. https://doi.org/10.1136/JECH.40.1.18

Moore MR, Goldberg A, Fyfe WM, Richards WN (1981) Maternal lead levels after alterations to water supply. Lancet 318:203–204. https://doi.org/10.1016/S0140-6736(81)90384-6

Moore MR, Robertson SJ, Gilmour WH, Murray GD, Britton A, Low RA, Watt GCM (1998) Decline of maternal blood lead concentrations in Glasgow. J Epidemiol Community Heal 52:672–673. https://doi.org/10.1136/JECH.52.10.672.

National Records of Scotland [WWW Document] (2022) https://www.nrscotland.gov.uk/statistics-and-data/statistics/statistics-by-theme/vital-events/general-publications/vital-events-reference-tables/2020/list-of-data-tables#section4 (accessed 12.31.22.

Parker JM, Wilby RL (2013) Quantifying household water demand: a review of theory and practice in the UK. Water Resour Manage 27(4):981–1011. https://doi.org/10.1007/s11269-012-0190-2

Potter S (1997) Lead in drinking water. Science and Environment Section House of Commons Library

Quinn MJ (1985) Factors affecting blood lead concentrations in the UK: results of the EEC blood lead surveys, 1979-1981. Int J Epidemiol 14:420–431. https://doi.org/10.1093/IJE/14.3.420

Rainey C (2025) Power rules: practical statistical power calculations. In: OSF preprints 5am9q. Center for Open Science

Ransom WB (1900) Diachylon as an abortifacient. Br Med J 2:201

Richards WN, Britton A, Cochrane A (1980) Reducing plumbosolvency-the effect of added lime on the Loch Katrine supply to Glasgow. Journal Of The Institute Of Water Engineers And Scientists 1979(34):315–333

Richards WN, Moore MR (1984) Lead hazard controlled in Scottish water systems. Journal (american Water Works Association) 76(8):60–67. http://www.jstor.org/stable/41272146

Robertson G, Wilson A, Papastathopoulos Y, Graham M, Eades L, Chatzisymeon E (2020) Estimating the number of homes in Scotland with internal lead piping. WWW Document. https://www.dwqr.scot/media/1f1n3kv4/ueh-007-19-project-management-doc-048-final-project-report-20-november-2020.pdf (accessed 12.31.22).

Royer H (2009) Separated at Girth: US twin estimates of the effects of birth weight. Am Econ J Appl Econ 1:49–85. https://doi.org/10.1257/APP.1.1.49

Schock MR, Lytle DA (2011) Internal corrosion and deposition control. In:(ed) Water quality and treatment: a handbook on drinking water, 6th edn. McGraw-Hill, pp 20.1–20. 109

Scottish Water (2021) Your guide to water pipework. WWW Document. https://www.scottishwater.co.uk/-/media/ScottishWater/Document-Hub/Your-Home/Pipework-in-your-home/090821SWYourGuideToWaterPipework2021v1pageslr.pdf (accessed 12.31.22)

Sun L, Abraham S (2021) Estimating dynamic treatment effects in event studies with heterogeneous treatment effects. J Econom 225(2):175–199. https://doi.org/10.1016/j.jeconom.2020.09

Taylor CM, Golding J, Emond AM (2015) Adverse effects of maternal lead levels on birth outcomes in the ALSPAC study: a prospective birth cohort study. BJOG An Int J Obstet Gynaecol 122:322–328. https://doi.org/10.1111/1471-0528.12756

Troesken W (2006) The great lead water pipe disaster. Cambridge, MA: MIT Press. ISBN 978-0-262-20167-4

UK Government (1989) The water supply (water quality) regulations 1989 (SI 1989/1147). https://www.legislation.gov.uk/uksi/1989/1147/made

UK Government (2003) The water supply (water quality) (amendment) regulations 2003. SI 2003/1416. https://www.legislation.gov.uk/uksi/2003/1416/made

UK Government (2016) The water supply (water quality) regulations 2016 (SI 2016/614), Schedule 1. https://www.legislation.gov.uk/uksi/2016/614/schedule/1U.S. EPA. (2025, January 7). Lead and Copper Rule. [WWW document] https://www.epa.gov/dwreginfo/lead-and-copper-rule. Accessed 8 June 2025

UNICEF (2020) The toxic truth: children's exposure to lead pollution undermines a generation of potential | UNICEF [WWW Document]. https://www.unicef.org/reports/toxic-truth-childrens-exposure-to-lead-pollution-2020. Accessed 31 Dec 2022

UNICEF (2022) A neglected tragedy: The global burden of stillbirths - UNICEF DATA [WWW Document]. https://data.unicef.org/resources/a-neglected-tragedy-stillbirth-estimates-report/. Accessed 31 Dec 2022

UN IGME (United Nations Inter-agency Group for Child Mortality Estimation) (2021) [WWW Document]. https://www.who.int/publications/m/item/levels-and-trends-in-child-mortality-report-2021. Accessed 31 Dec 2022

Vinceti M, Rovesti S, Bergomi M, Calzolari E, Candela S, Campagna A, Milan M, Vivoli G (2001) Risk of birth defects in a population exposed to environmental lead pollution. Sci Total Environ 278:23–30. https://doi.org/10.1016/S0048-9697(00)00885-8.

Vlasak T, Jordakieva G, Gnambs T, Augner C, Crevenna R, Winker R, Barth A (2019) Blood lead levels and cognitive functioning: a meta-analysis. Sci Total Environ. https://doi.org/10.1016/j.scitotenv.2019.03.052.

Watt GCM, Gilmour WH, Moore MR, Murray GD, Britton A, Robertson SJ, Womersley J (1996a) The Glasgow 93 lead study. Great Glasgow Health Board, Internal Report

Watt GCM, Gilmour WH, Moore MR, Murray GD, Britton A, Robertson SJ, Womersley J (1996b) Is lead in tap water still a public health problem? An observational study in Glasgow. BMJ 313:979–981. https://doi.org/10.1136/bmj.313.7063.979

Watt GCM, Gilmour WH, Moore MR, Murray GD, Britton A, Robertson SJ, Womersley J (1996) The Glasgow 93 Lead Study. Great Glasgow Health Board, Internal Report

WHO (1970) European standards for drinking-water, 2nd edn. World Health Organization. https://apps.who.int/iris/handle/10665/40025

WHO (2010) Preventing disease through healthy environments: a global assessment of the burden of disease from environmental risks. WWW Document. https://www.who.int/publications/i/item/9789241565196 accessed 12.31.22.

WHO (2016) The public health impact of chemicals: knowns and unknowns. WWW Document. https://www.who.int/publications/i/item/WHO-FWC-PHE-EPE-16-01 (accessed 12.31.22.

WHO (2021) Exposure to lead: a major public health concern. WWW Document. https://www.who.int/publications/i/item/9789240037656, 2nd (accessed 12.31.22.

WHO (2022) Guidelines for drinking-water quality: fourth edition incorporating the first and second addenda. WWW Document. https://www.who.int/publications/i/item/9789240045064 (accessed 08.06.25.

Wibberley DG, Khera AK, Edwards JH, Rushton DI (1977) Lead levels in human placentae from normal and malformed births. J Med Genet 14:339. https://doi.org/10.1136/JMG.14.5.339.

Wilcox AJ (2001) On the importance-and the unimportance- of birthweight. Int J Epidemiol 30:1233–1241. https://doi.org/10.1093/IJE/30.6.1233

Wooldridge JM (2010) Econometric analysis of cross section and panel data, 2nd edn. MIT Press

Wooldridge JM (2021) Two-way fixed effects, the two-way Mundlak regression, and difference-in-differences estimators. SSRN Electron. J. https://doi.org/10.2139/SSRN.3906345

Xie X, Ding G, Cui C, Chen L, Gao Y, Zhou Y, Shi R, Tian Y (2013) The effects of low-level prenatal lead exposure on birth outcomes. Environ Pollut 175:30–34. https://doi.org/10.1016/J.ENVPOL.2012.12.013

Yurdakök K (2012) Environmental pollution and the fetus. J. Pediatr. Neonatal Individ. Med 1:33–42. https://doi.org/10.7363/010116

Zheng J (2021) Lead exposure, human capital formation, and inequality: the impacts of drinking water lead on short-run and long-run educational outcomes. Working paper. https://www.jiamengzheng.com/assets/files/lead_JMP_zheng.pdf

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.