The Impact of Lead Water Pollution on Birth Outcomes: A Natural Experiment in Scotland[1]

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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. We suggest our findings indicate future research should further explore the mediating pathways between lead and health outcomes.

JEL codes: I18; Q53

Keywords: Under-5 mortality; pollution; lead; Difference-in-Differences

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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 2022a). 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 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, and it is said to be highly *plumbosolvent.*

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 50μg/l, compared to only 10% in England. Glasgow was badly affected, with 50% of households surveyed having water lead levels above 100μg/l (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 2006). Much previous evidence shows mixed findings on birthweights and on under-5 mortality (for example: Xie et al. 2013; Bornschein et al. 1989; Taylor et al. 2014; Azayo et al. 2009; Golmohammadi et al. 2007; McMichael et al. 1986, and 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 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 casual 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.

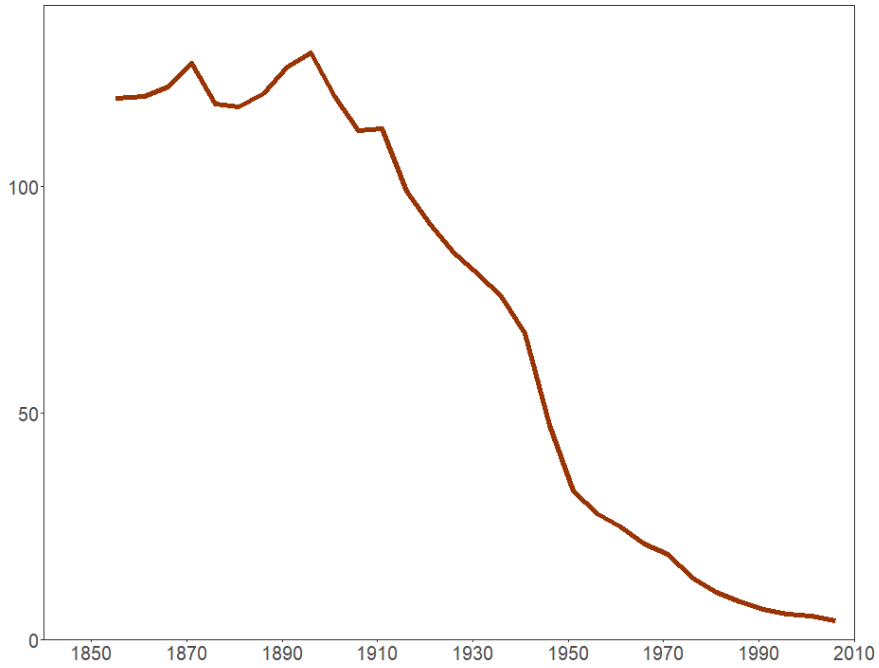
High levels of lead exposure in the womb are harmful. So much so, that lead oxide was described as being used as an abortifacient by women in the 1800s (Hall and Ransom, 1906). In some cases, the amounts of lead ingested were strong enough to cause lead poisoning in the mother (Ransom, 1900). However, 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 (figure 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.

Figure 1 – Deaths Within First Year of Life, per 1000 Births in Scotland



Source: National Records of Scotland (2022)

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, 2014, and Krebs, 2019). Lead piping began to be phased out from the 1970s in Scotland, but by 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 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). The acidic soft water in Scotland's two largest cities made piping there extremely plumbosolvent. In 1975 surveys found 33% of households in Scotland had water lead levels above 50µg/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 100µg/l (Richards and Moore, 1984). Average blood lead levels were higher in both cities than in any country today (figure 5 and Ericson et al., 2021).

This paper exploits two interventions to reduce lead in the water supply. We treat these as natural experiments, given the somewhat arbitrary assignment of water treatment to

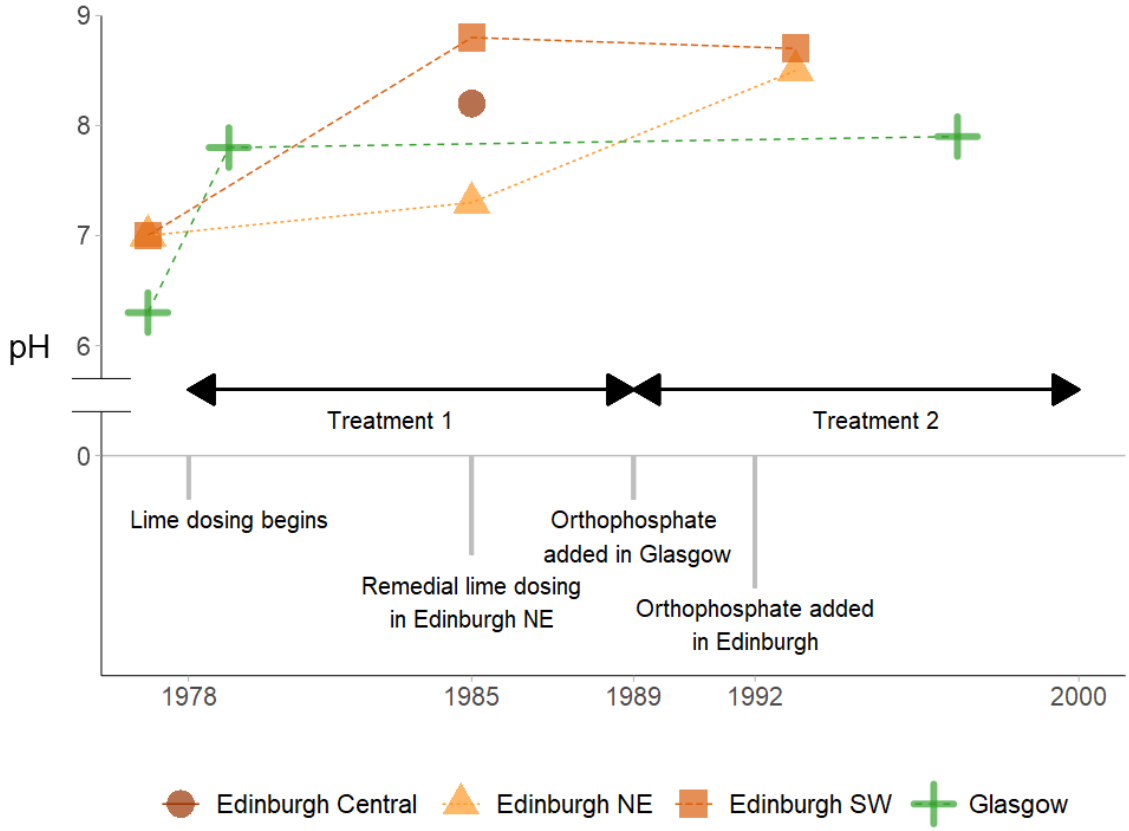
Glasgow and Edinburgh (treatment groups), while leaving many adjacent areas untouched until years later (control groups). These control areas were not only treated later, but also had higher pH levels for their untreated water than the treatment areas. In particular, we rely on these plausibly exogenous 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. 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 bioavailabiity of lead . 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 figure 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 figure 6). This timeline, along with different treatment groups, provides the scope for the implementation of staggered difference-in-differences methods, described in Section 4.

Figure 2 - pH Levels in Each Water Supply Area



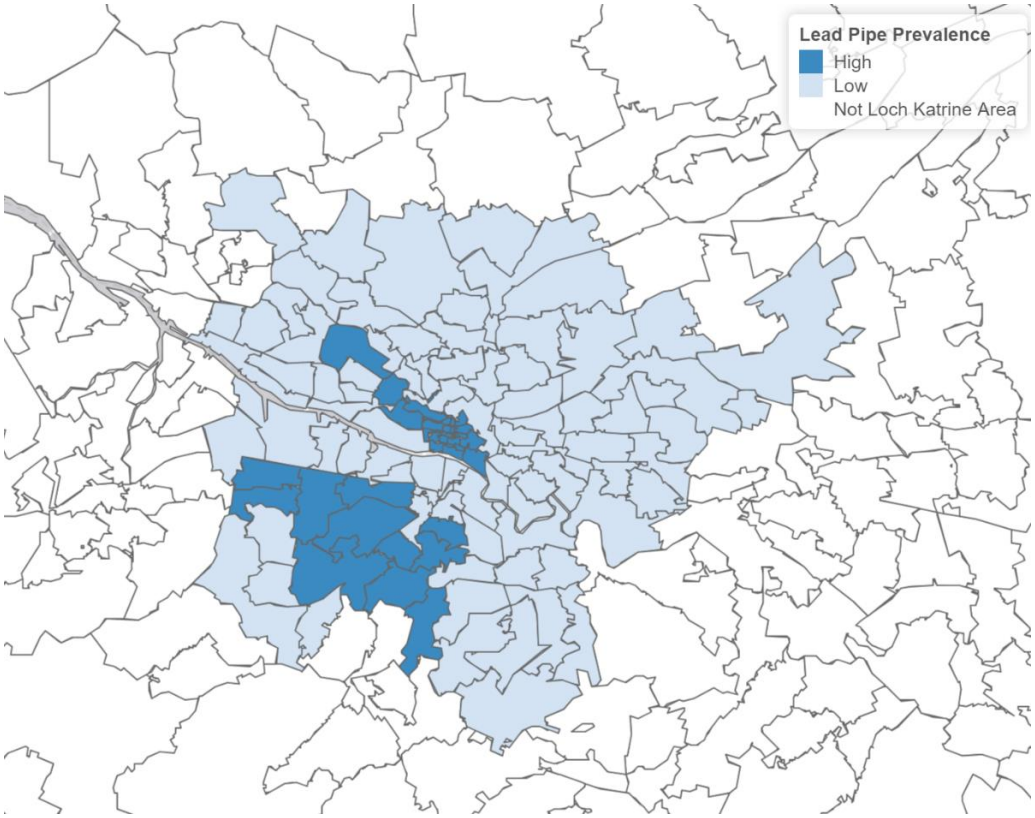
Notes: This chart show pH levels for the various water supply areas in different years. It also shows when the lime dosing (treatment 1) and orthophosphate dosing (treatment 2) began in each area. Water engineers targeted pH levels to be well above 7 to reduce plumbosolvency. Sources: Macintyre et al., (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 (figure 2). The Glasgow water supply area at this time is mapped in figure 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.

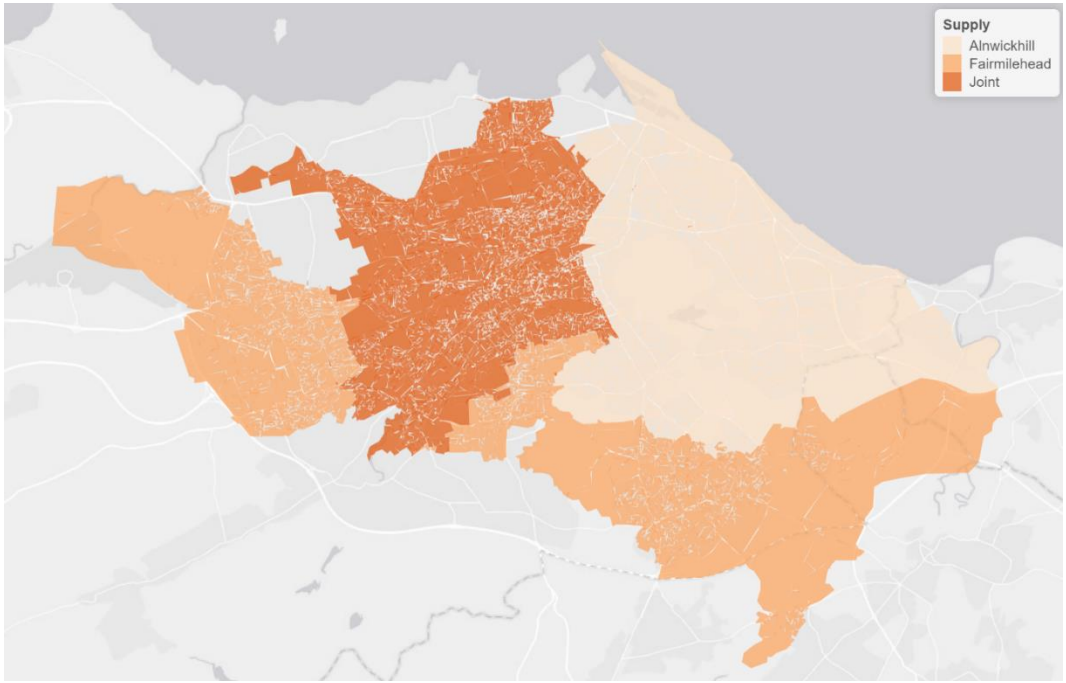
In Edinburgh, the city was supplied with water from two main sources: Alnwickhill, which served the north-eastern part of the city (referred to as Edinburgh NE for simplicity), and Fairmilehead, which catered to the south-western region (Edinburgh SW). The central area of the city (Edinburgh Central) received a combined supply from both these sources. See figure 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. 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 figure 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.

Figure 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).

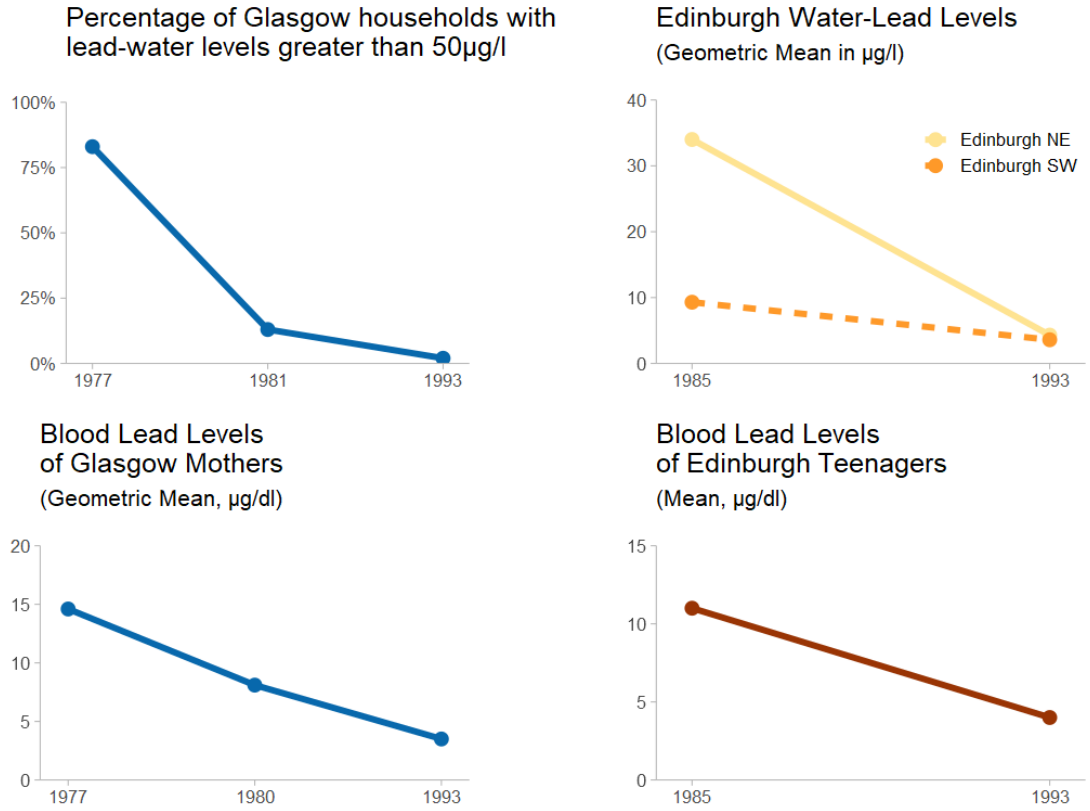
Figure 4 - Historical Water Supply Areas in Edinburgh



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 Farmilehead water supply. The central area (Edinburgh Central) is jointly served by both water supplies.

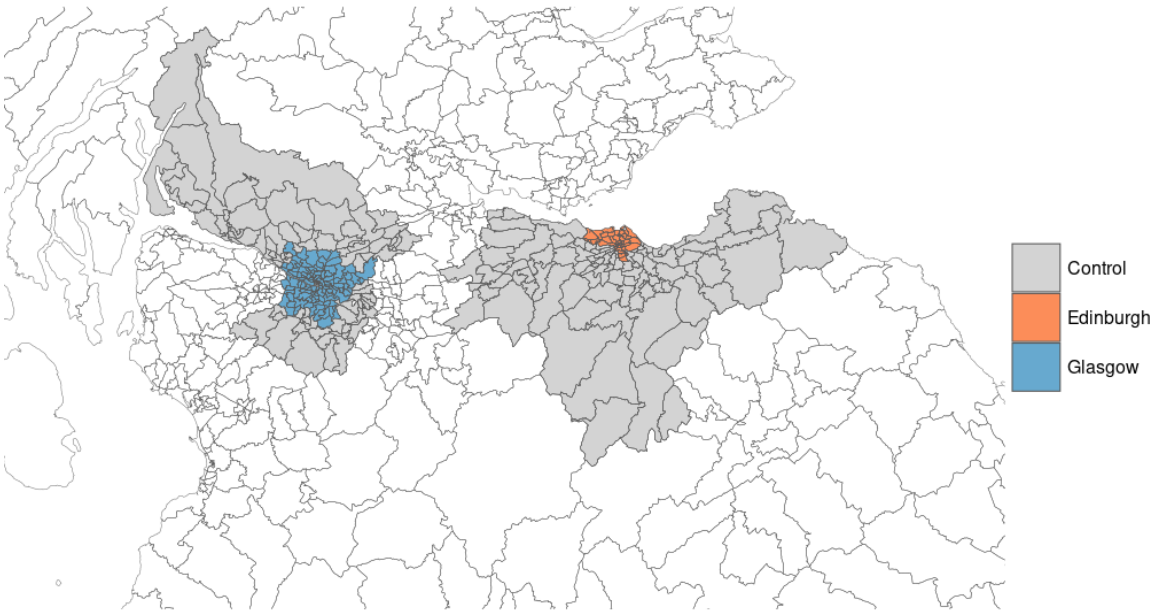
These treatments lowered both water and blood lead levels (figure 5), but not by enough from the perspective of the government. Treatment 2, orthophosphate dosing, was therefore carried out to further decrease lead in the water supply. Orthophosphate dosing reduces the solubility, and therefore bioavailability, 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 (figure 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).

Figure 5 – Water and Blood Lead Levels in Glasgow and Edinburgh



Sources: Watt et al., (1996a) and Moore (1998), Macintyre et al., (1998)

Figure 6 - Map of Treatment and Control Areas.



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[5].

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/control group. The map of water supply areas, and the various treatment groups are included in figures 3, 4, and 6. Figure 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%. Figure 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

[5] We would like to thank the Electronic Data Research and Innovation Service (eDRIS) of Scotland for their help with data handling and access.

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 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"[6]. 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[7], 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.

[6] See the SDG targets and indicators here: https://sdgs.un.org/.
[7] 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 Cairstairs 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
	Edinburgh SW	0.52		0.50	8,315
	Joint	0.51		0.50	42,011
	Glasgow	**0.51**		**0.50**	**216,771**

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 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.

$$(1) \qquad \tau_{rt} = E[\, y_{it}(r) - y_{it}(0) \mid d_{ir} = 1\,],\ r = q, \dots, T;\ t = r, \dots, T.$$

Where $y_{it}(r)$ is the outcome for child i at time t given their water supply began treatment at time r, and $r \leq t$, and $y_{it}(0)$ 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.

$$(2) \qquad \bar{\tau}_r = E[\tau_{rt}]\,, r = q, \dots, T; t = r, \dots, T.$$

Which we estimate as:

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

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

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

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 by T .

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

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, and Calloway and Sant'Anna, 2021).

However, given the plausibly exogenous change in lead exposure outlined in section 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.

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:

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

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 section 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.

Conditional Parallel Relative Trends, Staggered Treatment (CPRTS)

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

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).

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

Where η is the intercept and $f s_t$ 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, $\dot{\boldsymbol{x}}_{\boldsymbol{ir}}$ 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.

$$(9) \quad E[\, y_{it} \mid d_{ir}, \boldsymbol{x_i}\,] = \boldsymbol{\Lambda}\,[\, \eta +, \boldsymbol{x_i}\boldsymbol{\kappa} + \sum_{r=q}^{T} \lambda_r d_{ir} + \sum_{r=q}^{T} \zeta_r (d_{ir} \times, \boldsymbol{x_i}) + \sum_{s=2}^{T} \theta_s f s_t + \sum_{s=2}^{T} (f s_t \times \boldsymbol{x_i})\boldsymbol{\pi_t} + \sum_{r=q}^{T} \sum_{s=r}^{T} \tau_{rt} (d_{ir} \times p_{irt} \times f s_t) + \sum_{r=q}^{T} \sum_{s=r}^{T} (d_{ir} \times p_{irt} \times f s_t \times \dot{\boldsymbol{x}}_{\boldsymbol{ir}})\boldsymbol{\rho_{rt}}\,]$$

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

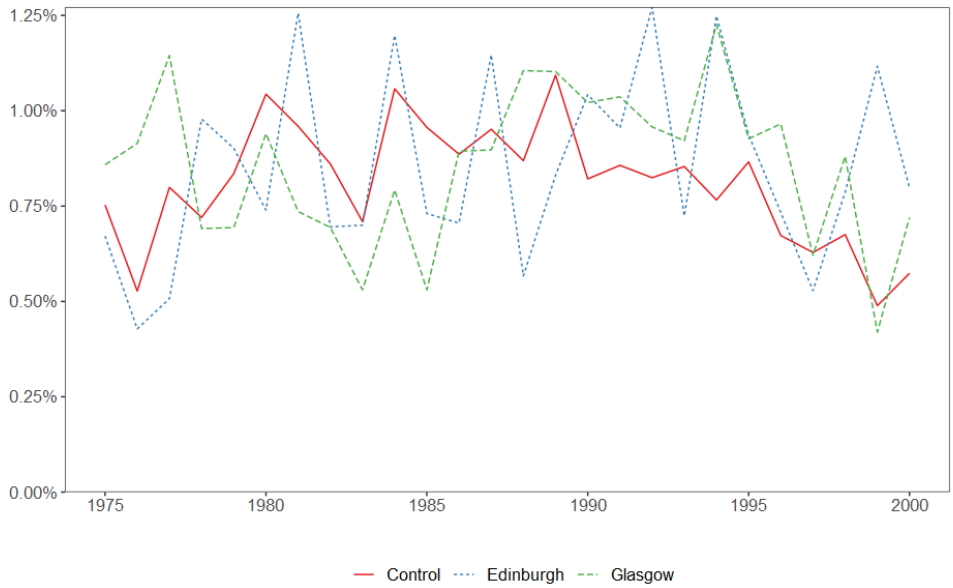
We first plot the mean birthweight for Glasgow, Edinburgh and the control group of surrounding areas in figure 7. In all groups there is a clear upward trend. The Edinburgh average closely tracks the control group, while the Glasgow average remains below both at all times, and there is no convergence 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 section 5.1.

Similarly, in figure 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.

Figure 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.

Figure 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.

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 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 section 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 24g. 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 18g.

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.

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

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

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 equations (8) and (9), but we include the pre-treatment years as well for each cohort. In figure 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 time-to-treatment, so Edinburgh NE, which was treated later, has more pre-treatment periods than Glasgow and Edinburgh 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 two- 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 a 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).

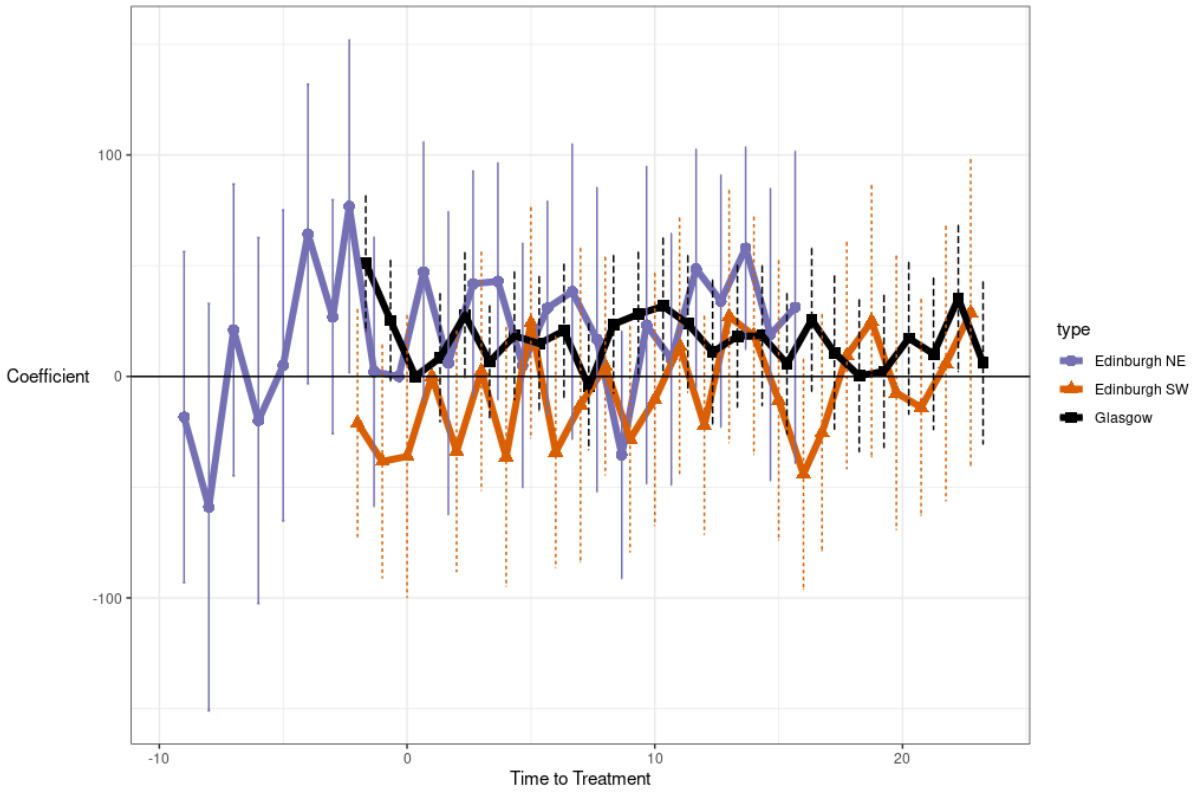
In figure 11 we show event study APE's for each cohort year for the effect on mortality of treatment 1. All pre-treatment intervals cover zero, suggesting no clear evidence of pre-trends. Glasgow has mostly negative point estimates after treatment while

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 figure 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 perfom 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).

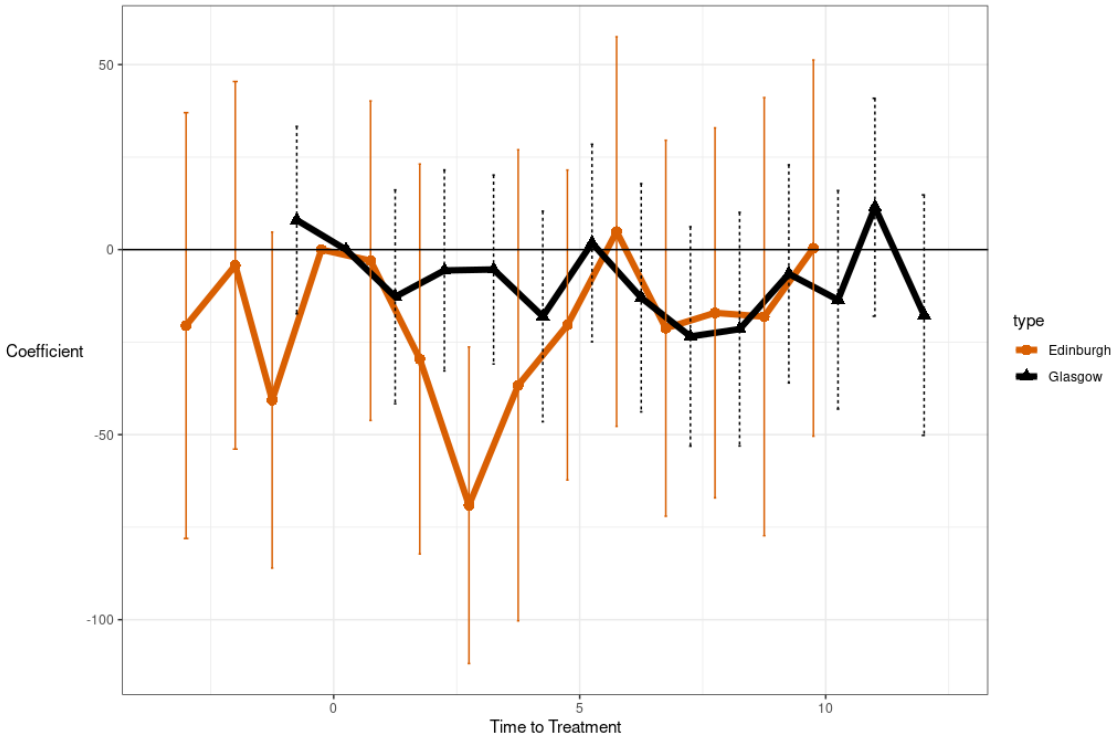
As a robustness check, we include results from a matrix completion difference-in-difference estimation in appendix tables A.10 and A.11. Matrix completion can be thought of as a generalization of synthetic control methods (Athey et al., 2021) and is used to impute the counterfactual trend based on latent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

Figure 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. 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.

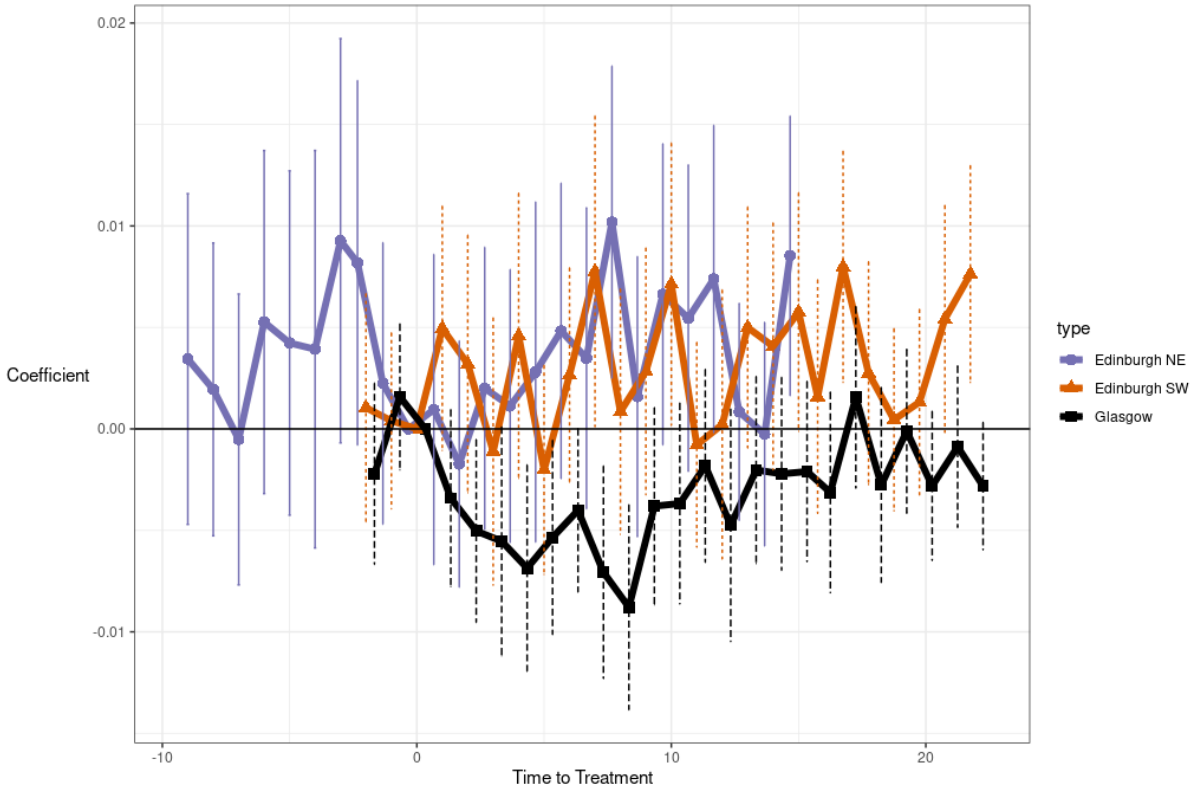
Figure 10 - Effect of Treatment 2 on Birthweights (Event Study with Two-way Mundlak Estimator)



Notes:

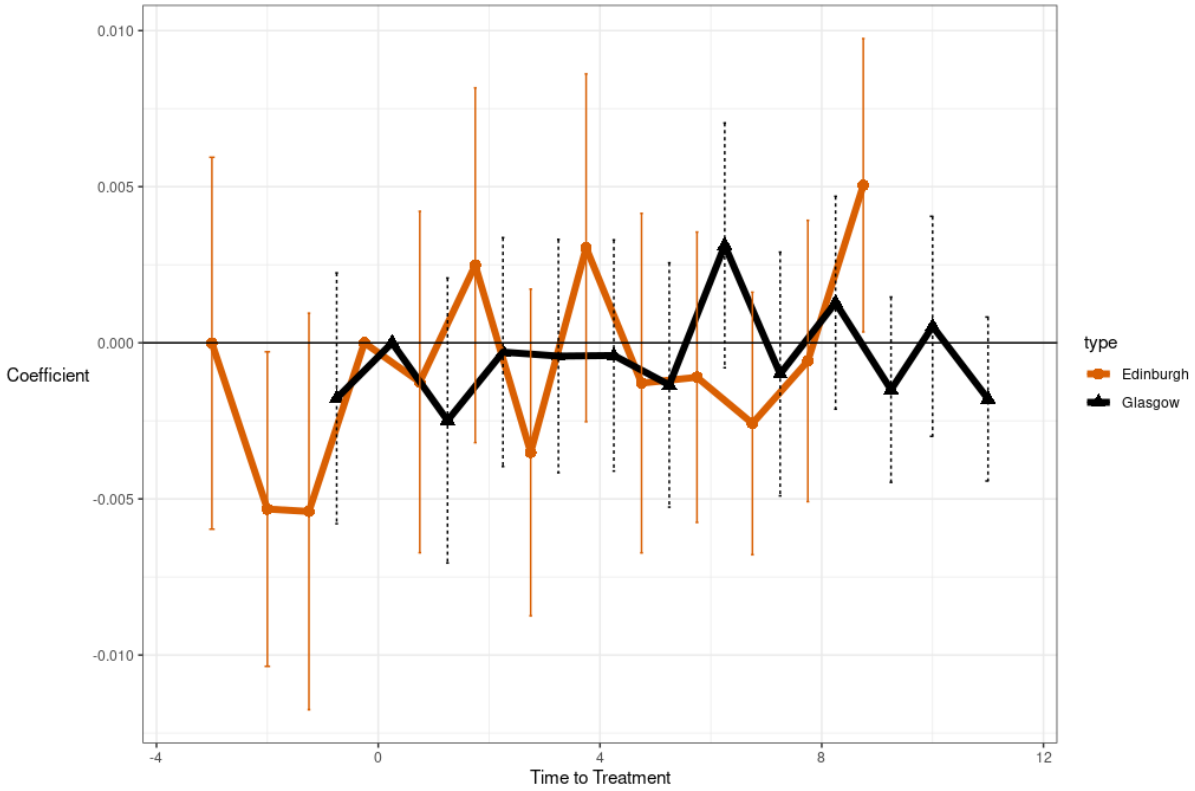
Table shows estimated treatment effects on different groups of lead reduction due to orthophosphate in Glasgow and Edinburgh compared to a never-treated control group. We use standard errors clustered by postcode sector. In all cases the estimates are the average treatment on the treated for that year on that group Birthweights is the birthweight in grams for a child.

Figure 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. 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.

Figure 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. 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.

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 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 several 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.

It is worth thinking about our results in light of the plausible mechanisms laid out in section 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. 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 percent – 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, 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 20th 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. We suggest that future research should investigate the mediating pathways between lead and harmful outcomes, to better understand effective treatments.

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