The Impact of Lead Pollution on Human Capital Formation: Size of Dose Matters

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Abstract

Does lead pollution harm educational achievement? And are the marginal effects greater at low or high levels of lead? We use exogenous variation in lead pollution from water treatment in Glasgow, Scotland, combined with within-household sibling differences, to estimate the effect of lead on education. We compare pre and post-treatment sibling differences between treated and control areas with difference-in-differences estimation. We find a clear dose-response relationship. Treated areas with low prevalence of lead piping show no change compared to a control group. In contrast, high lead pipe prevalence areas show improvement in educational outcomes. Our findings indicate that countries and areas with very high levels of lead can expect large educational gains from even small amounts of lead abatement, while those with already low levels of lead can expect much lower marginal improvements.

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1. Introduction

Recent estimates indicate 1 in 3 children suffer from high blood lead levels (GBD, 2019). The short-term health consequences of lead are well known, but in recent decades studies have shown it may also have a variety of long-term, higher order outcomes, including lowering educational attainment (see section 2),. Given there are potentially 800m children worldwide with high lead levels, this implies huge future costs we are imposing now, and consequently large returns on investment in abatement.

But lead is not the only pollutant, nor the only long-term cause of harm to children. Given production and political-economy constraints, lead abatement must compete against other policy needs, such as poverty reducing transfers, or greenhouse gas abatement. Balancing the long-term gains from lead abatement with other concerns requires knowledge of what long-term harm it does and, crucially, the relationship between that harm and the level of lead. This is called the *dose-response* relationship.

Previous studies have found infant lead exposure to be associated with long-term harmful outcomes, but there is disagreement in the literature on the dose-response relationship. Grönqvist, Nilsson and Robling (2019), find a threshold effect, where the later effects of lead on probability of graduation, or grade point average, are low, or non-existent, until around 5μg/dl in blood lead levels is reached. Reyes (2007) finds that for some outcomes the effects of lead are 20 times as large for the 4th quartile of lead exposure than they are for the 1st quartile. Sampson and Winter (2018) show no effect of lead on anti-social behaviour when blood lead is below 5μg/dl and increasing marginal effects at higher levels. Gazze, Persico, and Spirovska (2021) find pre-school blood lead is linearly associated with worse education outcomes at the individual level, but also find large spill over effects from having peers with high lead levels. This potentially means spiralling adverse consequences once individual and networks effects are combined.

In contrast, Evens *et al*., (2015) find higher marginal effects of lead on reading ability at lower levels of lead than at higher levels. Mielke and Zahran (2012) find the relationship between lagged air lead levels and assault rates to be linear. Miranda *et al*., (2007) find the relationship between blood lead and reading and mathematics ability to be linear, while Canfield *et al*., (2003) find that marginal effects on lead on IQ are greater below

5μg/dl blood lead levels. With the exception of Reyes (2007) and Grönqvist, Nilsson and Robling (2019), these studies are correlational estimates, that do not use plausible exogenous variation to identify a causal dose-response relationship.

We use the exogenous variation resulting from a treatment of the water supply in Glasgow, Scotland in November 1989 to estimate the effects of infant lead water ingestion at different doses on long-term education outcomes. Before treatment, Glasgow had water lead levels far in excess of those of Flint, Michigan in 2015, and the highest average blood lead levels of any city surveyed in the UK. After treatment, the percentage of households with lead-water levels greater than 50μg/l fell from 13% to 2% (Watt et al., 1996a) and the blood lead level of mothers from Glasgow decreased from 11.9μg/dl to 3.7μg/dl (Watt et al., 1996a).

We split our sample into high, low, or control group doses based on the prevalence of lead piping in the local area. Our main identification strategy uses the difference between siblings within the same household born either side of treatment, with difference-in-differences estimated between the dosage groups; the second strategy uses a difference-in-differences between dosage groups with outcomes averaged at the school level. We find little evidence of an effect for the low dosage group, but we do find evidence of socially significant effects for the high dosage group.

Our findings are in contrast to the literature stating lead pollution has the highest marginal effects on human capital formation when it is at low levels. Our contribution is to show that at low levels lead has little effect on education outcomes, instead it is at the high levels where the greatest marginal effects can be found. The implications are that countries with low average lead levels cannot expect large gains in educational attainment from lead abatement, except in targeted programmes aimed at the highest lead polluted areas. However, countries and areas with high infant lead ingestion such as India, where as many as 60% of its 470m children have lead levels greater than 5μg/dL, can expect huge future educational gains from lead abatement policies.

2. Background

2.1 Lead Pollution and Human Capital

Lead has been recognised as harmful for thousands of years (See Needleman, 1992), but the long-term effects of infant lead ingestion on educational outcomes have only been investigated in recent decades. Lead water pollution when young is thought to be especially harmful for three reasons: firstly, children absorb up to 50% of ingested lead compared to 10% in adults (WHO, 2010); secondly, the blood-brain barrier is the main defence against large, water-soluble molecules, and this is not fully developed until after the first year of life, with in utero absorption being the most dangerous period (Goldstein, 1990); thirdly, a much higher share of infant diet tends to come from water, either through breast milk and their mother's water lead ingestion (Ettinger *et al*., 2004), or, more directly, from bottles of milk formula mixed with water (Baum and Shannon, 1997). In a 1993 survey, 84% of infants in Glasgow were bottle-fed (Watt *et al*., 1996b).

Lead impairs nerve conduction (Sindhu and Sutherling, 2015), damages myelination in the nerve system (Brubaker et al., 2009), and can impede brain development (Lanphear, 2015). This may affect educational outcomes directly, through nerve and brain injury. Lead has been associated with impaired cognitive functioning (Vlasak et al., 2019) and lower IQ scores (Schwartz, 1994). A second possible mechanism is through behavioural changes. Blood lead levels are associated with aggressiveness, anti-social behaviour, and delinquency (e.g.: Thomson *et al*., 1989, Needleman, 1996, and Reyes, 2015). These behaviours may have spill over effects on peers, so that even children with low-lead levels may experience worse educational outcomes due to peer behaviour (Gazze, Persico, and Spirovska, 2021).

Given the likely strong relationship between infant lead levels and water lead, due to bottle feeding, water lead may be particularly harmful during early development, yet few studies look primarily at water lead levels and human capital. Zheng (2021) uses an instrumental variable estimation and finds increases in water lead levels reduce both mathematics and reading scores. Ferrie, Rolf, and Troesken (2012) find childhood water lead exposure to lower intelligence scores in US army enlistees.

2.2 The Glasgow Water Treatment

Glasgow's population grew from around 90,000 citizens in 1801, to 300,000 in 1841 (University of Portsmouth, 2022). Even in 1801, the water supply of 30 wells was inadequate being "impregnated with sewage and other deleterious matter" (Burnet,

1869). The Council and several private companies in turn attempted to improve matters, by taking water from the Clyde River and water to the south.

The Clyde water was pumped without being filtered and, due to the industrial use of the water, it was considered of poor quality, while the water supplied to the south of the city by the Gorbals Gravitation Water Company was of better quality. Even combined, however, these waterworks were not sufficient to keep the city supplied, especially not with water of good quality, so the Council eventually decided upon a new water supply: Loch Katrine. Katrine, a large and picturesque mountain lake, was considered more than adequate to supply Glasgow's growing population with good quality water. Indeed, the quality of the water was much remarked upon. A report to the council from a chemist, one Dr Smith, reported that the water "was almost absolutely pure, clear to the utmost and without colour…[and] needs no purification". He then recommended this water to the council over any other option saying, "no town will have an equal abundance of such remarkably pure water" (Burnet, 1869).

The water is very soft and pure. Soft water lacks the mineral content found in harder waters and this means it has a low pH. Low pH water reacts with lead pipes (high plumbosolvency), dissolving the metal into the water supply (Kim *et al*., 2011). This was known at the time, due to the experiments of Robert Christison (1844). In 1854 one chemist, a Professor Penny, found that Loch Katrine water, after travelling through lead pipes, was "highly charged with lead", and believed it would be hazardous to supply such water to Glasgow (Burnet, 1869). The city council collected statement from various professors, engineers, and inhabitants of cities with soft water, before deciding there was no health risk. Glasgow has been supplied with Loch Katrine water ever since.

Professor Penny's worries over the Glasgow water lead levels would not be returned to until the mid-20th century. UK blood lead monitoring surveys in the 1970s found that Glasgow had the highest geometric mean blood lead level in any city surveyed (Quinn, 1985) at 18*f*g/dl. Six separate lead working sites were monitored in the same survey, and the Glasgow mean blood-lead level was higher than 5/6 of the lead-working sites. It was higher than the mean level of the lead workers themselves in 4/6 sites (Quinn, 1985). Using these monitoring results, Quinn (1985) found that local plumbosolvency was much more closely related to local blood lead levels than distance to a road, and that lead-water intake was likely the biggest factor in the UK.

By this time, the health impacts of "moderate" levels of lead were being taken seriously, and an EU directive set the maximum water supply concentration of lead to 50μg/l from the previous 100μg/l (Watt *et al*., 1996a). The Glasgow water supply was treated in 1978 with lime (Calcium hydroxide) to raise the pH and reduce plumbosolvency, with an increase in this treatment in 1981. This raised the pH from 6.3 to 9, and reduced the water lead levels (Moore *et al*., 1981).

However, in the late 1980s, the remaining levels of lead were deemed to still be too high. Surveys of water lead levels in residences found that in 1981, after the initial treatments, 13% of Glasgow households had water lead levels greater than 50μg/l (Moore *et* al, 1998), and 5% of homes had lead-water levels greater than 100μg/l (Moore *et al*., 1982). For comparison, the 90th percentile of lead-water samples in Flint, Michigan in 2015 was 31μg/l (Pieper *et al*., 2018).

Therefore, a second treatment of adding orthophosphate to the water was begun in November 1989 (Watt *et al*., 1996a). Correspondence with the engineering team involved with the project indicate the treatment was successful within a few weeks (author correspondence, 2020), and lead-water levels fell. The percentage of households with lead-water levels greater than 50μg/l fell from 13% to 2% (Watt *et al*., 1996a). A long-term survey of mothers giving birth from Glasgow shows a decline in geometric mean blood lead levels from 11.9μg/dl in 1981 to 3.7μg/dl in 1993 (Watt *et al*., 1996a).

We use the plausibly exogenous reduction in lead intake resulting from the 1989 water treatment to identify the effect of lead on education outcomes. However, we also distinguish between areas with a high lead pipe prevalence and areas with a low lead pipe prevalence withing Glasgow.

In Glasgow at this time there were estimated to be 160,000 housing units with some lead piping out of the 300,000 in the city (Watt *et al*., 1996a), but this was not equally concentrated. Far more of the older housing units had lead piping, either as service pipes under the ground, or internal piping. Surveys of the population in Glasgow showed that 19% in the high lead areas said they had lead piping compared to 9% in the other areas of Glasgow (Watt *et al*., 1996a). They also had far higher concentrations of lead in their water supply even after the 1989 treatment (table 1). McDonell, Campbell,

and Stone (2000) found that the reduction in neural tube defects in the years after treatment was much greater in the high lead areas than in the low lead areas (table 2). These facts indicate a dose response relationship, where the effect of treatment will be higher in areas which have a higher prevalence of lead piping. We therefore divide our sample into "High Lead" and "Low Lead" for our main estimates.

Table 1 – Water Lead Concentrations in High and Low Lead Pipe Prevalence Areas, 1993

	Percent of Households	
µg/l	High Lead Areas	Low Lead Areas
<2	37.4	53.8
2-9	35.5	31.8
10-24	17.7	8.3
25-49	5.6	3.9
≥50	3.7	1.5
Observations	*785*	*941*

Notes: Data from table 5 in Watt *et al.* (1996a).

Table 2 - Pregnancy prevalence of neural tube defects for each 1000 live births

	1983-95	1990-95
High Lead Area	2.1	0.69
Low Lead Area	2.4	1.8

Notes: Data from table 2 in McDonell, Campbell, and Stone (2000). Neural tube defects are early stage in utero damage to the brain, spine, or spinal cord.

3. Data

Our education data for each child comes from the Scottish Qualifications Authority (SQA). They provide the education outcomes for each child, the year of examination, and the centre they attended (usually a school or college) for all of Scotland. We only have data for state schools, not for private schools or academies. However, 96% of pupils in

Scotland use state schools[4]. We exclude schools in Edinburgh from our sample, as they underwent a similar treatment in 1991/92 partly due to the findings of the Glasgow lead monitoring, but all our results are robust to inclusion of the Edinburgh data (see online appendix). SQA also provide matching indicators for siblings, where children are matched to the same family by surname, postcode and first line of address. Finally, they provide the Scottish Index of Multiple Deprivation (SIMD) 2009 quintile for each child's postcode. The SIMD is a ranked index of deprivation on multiple dimensions (Income, Health, Education, Housing etc). The index is recalculated every 3 years. Although the ranking of each postcode moves around somewhat, the quintiles are relatively stable. We also use youth unemployment data for each year at the local authority level.

All children in Scotland during this period sit exams in their fourth year at Standard Grade. They sit exams in several subjects. The passing grades for these exams go from 1, the highest, to 7, a fail. A grade of 1 or 2 is called a "Credit" grade and allows one to go on to study the next level in the following year (called a "Higher"). Points are also awarded for each grade in each subject, and these are used as a marker for progressing to tertiary level education. The better the marks received, the higher the number of points. We only include the first examination year where a child sits Standard Grades in our sample (i.e., we do not include resits or repeated years).

We consider three outcome variables. The first is the total Standard Grade points achieved in that examination year. More points are better, but some subjects, such as Physics or Chemistry, are considered harder, but nevertheless taken as they are a prerequisite for some university courses (e.g., medicine often requires at least two science subjects). Some other subjects may be chosen instead if they are believed to be easier to get a Credit grade in, and the child does not wish to study medicine or engineering for example. Therefore, we consider two other outcomes: whether a child achieves a Credit grade in Mathematics, or in English. These are two subjects every child must sit, and therefore may give a better indication of change in ability rather than tastes in subjects. We use a Credit grade because this is the level needed to progress to "Highers" (a more difficult level of study, and a prerequisite for university) in the following year.

[4] Scottish Council of Independent Schools census: https://www.scis.org.uk/facts-and-figures/#:~:text=SCIS%20uses%20the%20information%20collected,4%25%20of%20pupils%20in%20Scotland

We sort each child into the "High Lead", "Low Lead" or "Control" based on their 1993 school postcode, using the plan of Loch Katrine supplied households, and high and low leaded pipe prevalence used in the map of Watt *et al*. (1996a). See figures 1 and 2 for a map of the schools and the high and low lead areas. Postcodes are UK government administrative boundaries used for a variety of purposes including the sorting of mail. In urban areas they tend to be smaller than in rural areas. We do not have access to the postcode of the child, so the matching of child to area is not exact. However, state school catchment areas are based on the postcodes surrounding the school so we expect the child postcodes to be nearby, especially in urban areas, but there will be some classical measurement error which may attenuate estimates. Summary statistics for our data are included in table 3.

Table 3 – Descriptive Statistics

Panel A - Full Sample

Variable	N	Mean	Std Dev	Min	Max
Outcomes					
Standard Grade Points	522661	163.106	80.269	0	422
Mathematics Credit Pass (=1 if passed with credit score)	468490	0.307	0.461	0	1
English Credit Pass (=1 if passed with credit score)	490212	0.425	0.494	0	1
Covariates					
Child SIMD quintile 1	544041	0.213	0.41	0	1
Child SIMD quintile 2	544041	0.204	0.403	0	1
Child SIMD quintile 3	544041	0.201	0.401	0	1
Child SIMD quintile 4	544041	0.198	0.399	0	1
Sex (1 = Male)	558379	0.505	0.5	0	1
Year of Birth	558379	1988.526	2.839	1984	1993
Area Youth Unemployment (%)	558379	62.625	6.33	45.2	87.5

Panel B - High Lead Areas Sample

Variable	N	Mean	Std Dev	Min	Max
Outcomes					
Standard Grade Points	18248	155.499	74.628	0	339
Mathematics Credit Pass (=1 if passed with credit score)	17004	0.259	0.438	0	1
English Credit Pass (=1 if passed with credit score)	17650	0.337	0.437	0	1
Covariates					
Child SIMD quintile 1	17763	0.41	0.492	0	1

Child SIMD quintile 2	17763	0.234	0.423	0	1
Child SIMD quintile 3	17763	0.155	0.362	0	1
Child SIMD quintile 4	17763	0.117	0.322	0	1
Sex (1 = Male)	18616	0.518	0.5	0	1
Year of Birth	18616	1988.436	2.852	1984	1993
Area Youth Unemployment (%)	18616	52.079	3.034	45.2	87.5

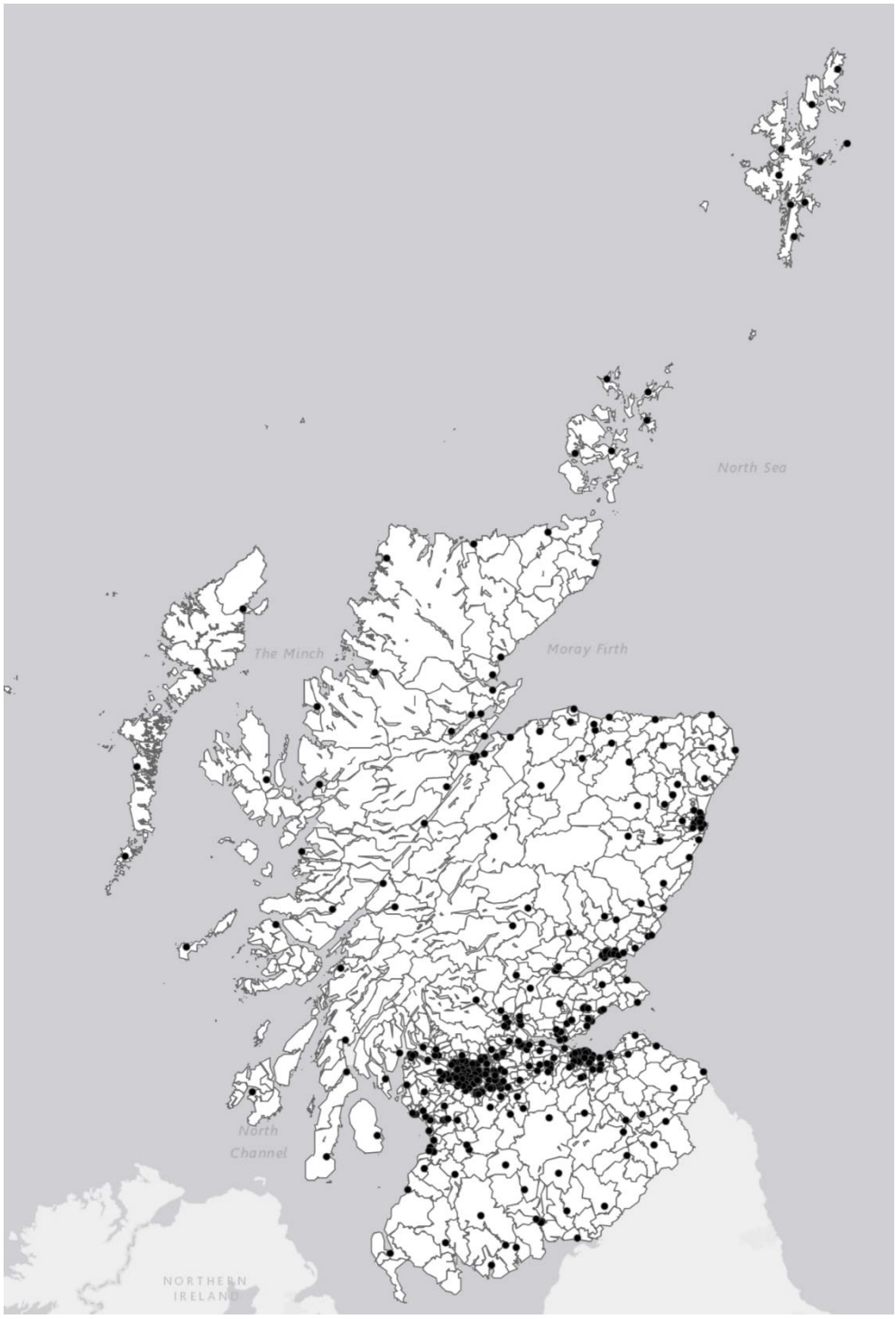
Panel C - Low Lead Areas Sample

Variable	N	Mean	Std Dev	Min	Max
Outcomes					
Standard Grade Points	35012	144.936	75.292	0	350
Mathematics Credit Pass (=1 if passed with credit score)	31039	0.233	0.423	0	1
English Credit Pass (=1 if passed with credit score)	33628	0.335	0.472	0	1
Covariates					
Child SIMD quintile 1	35153	0.564	0.496	0	1
Child SIMD quintile 2	35153	0.145	0.352	0	1
Child SIMD quintile 3	35153	0.1	0.301	0	1
Child SIMD quintile 4	35153	0.093	0.29	0	1
Sex (1 = Male)	37144	0.48	0.5	0	1
Year of Birth	37144	1988.535	2.841	1984	1993
Area Youth Unemployment (%)	37144	54.563	6.016	47.3	72.9

Panel D - Control Sample

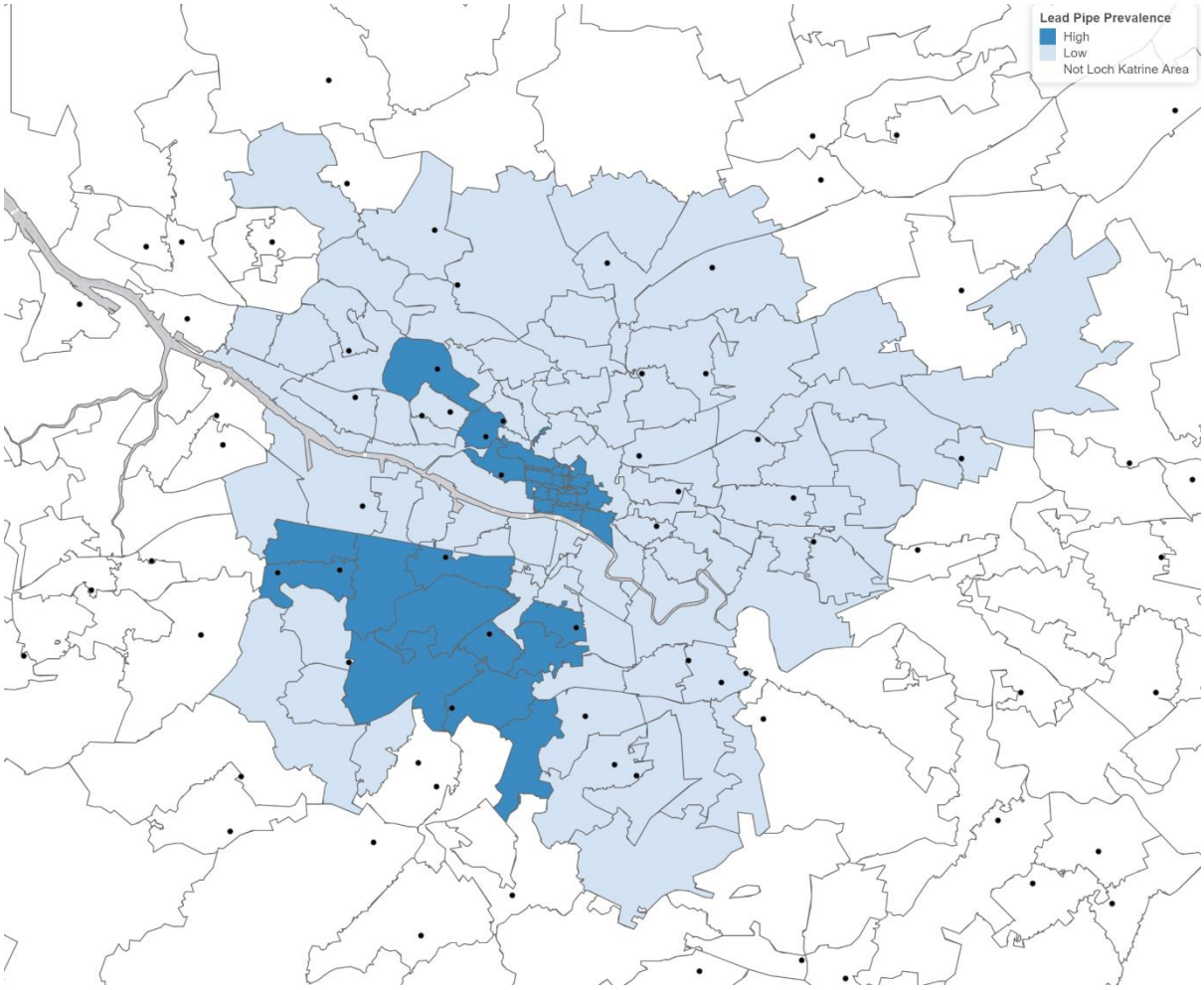
Variable	N	Mean	Std Dev	Min	Max
Outcomes					
Standard Grade Points	469401	164.757	80.655	0	422
Mathematics Credit Pass (=1 if passed with credit score)	420447	0.315	0.464	0	1
English Credit Pass (=1 if passed with credit score)	438934	0.435	0.496	0	1
Covariates					
Child SIMD quintile 1	491125	0.181	0.385	0	1
Child SIMD quintile 2	491125	0.207	0.405	0	1
Child SIMD quintile 3	491125	0.21	0.407	0	1
Child SIMD quintile 4	491125	0.209	0.407	0	1
Sex (1 = Male)	502619	0.506	0.5	0	1
Year of Birth	502619	1988.529	2.838	1984	1993
Area Youth Unemployment (%)	502619	63.646	5.616	45.2	87.5

Figure 1 – Distribution of Schools in Scotland



Notes: Dots represent school locations. Due to travel distances and sparse population several markings for "Schools" on the islands may represent the same nominal school but are given different IDs in the data.

Figure 2 – Distribution of Schools, High and Low Lead Areas in Loch Katrine Water Supply Area



Notes: shaded area is the Loch Katrine water supply area. Darker shading indicates a high prevalence of lead piping. Lines represent different postcode sectors. Dots represent school locations.

4. Empirical Strategy

All of our analysis relies on the plausibly exogenous variation in lead ingestion, in the womb and in childhood, resulting from the orthophosphate treatment of the Loch Katrine water supply to Glasgow in November 1989. As lead ingestion has been shown to be particularly harmful in the womb, our treatment start date is for children conceived after treatment. Of course, we do not have data on when our sample was conceived, only the date of birth. We take as our start date 1st of September 1990. That is, given the treatment would be effective by end of November (author correspondence,

2020), we take as the treatment group children born 9 months after this. The distribution of birth time from conception for term births is unimodal and symmetrical within the 10th-90th percentiles (Jukic *et al*., 2013). We believe there will only be minor classical measurement error resulting from this, but it may attenuate our estimates. Therefore, our estimates, if unbiased, may be a lower bound of the effect.

Our main identifying assumption is that the water treatment in November 1989 is exogenous variation in the lead intake of children conceived within the Loch Katrine supply area in Glasgow. Therefore, we assume that this treatment has an effect on education outcomes and is not associated with any confounding variables. We estimate our main results with a variety of difference-in-differences specifications. Our estimand is the Average Effect of Treatment on the Treated (ATT). This requires an assumption of parallel trends, the change in outcomes would be the same for treated and untreated without treatment. Given Glasgow is an urban area, with much higher concentrations of poverty than the Scottish average, we therefore also condition on a variety of covariates so that we assume parallel trends conditional on these covariates in some specifications.

As we have only one treatment period, that is common to all treated units, and a control group that is always untreated, we do not have to consider potential negative weighting arising from comparing earlier treated to later treated groups. Therefore, certain elements of the modern difference-in-difference literature, such as Goodman-Bacon decomposition (Goodman-Bacon, 2021) or reweighting of estimates (Calloway and Sant'Anna, 2021), do not apply. However, we do need to factor in another facet of the recent difference-in-difference literature: continuous treatment.

Different treatment doses combined into one treatment group can mean biased estimates of the ATT. Callaway, Goodman-Bacon, and Sant'Anna (2021) show that when you combine different doses you need stronger assumptions than with standard two-way fixed effects. The identifying assumption with standard two-way fixed effects difference-in-difference is parallel trends (or conditional parallel trends). With continuous treatment (i.e. different dosages) combined, this assumption will be violated if there is selection effects into different dosage units. For example, if people within the treatment area begin to move to housing that has more lead piping, due to the water now being safer. With combined dosage difference-in-differences, we require stronger

assumptions of either no selection into different dosage areas on average, or homogenous treatment effects.

These stronger assumptions can be relaxed back to standard parallel trends if, following the advice in Callaway, Goodman-Bacon, and Sant'Anna (2021), we separate the dosage units and compare them individually with the never-treated groups. Now all that is required is parallel trends between each dosage level separately with the control group. This is analogous to the traditional parallel trends assumption and can be made conditional on covariates. Callaway, Goodman-Bacon, and Sant'Anna (2021) show that this approach recovers an unbiased estimate of the ATT for that group and dose, but we sacrifice some efficiency by excluding some of the sample.

Our best proxies for treatment dosage are the high and low lead areas of Glasgow as described in section 2. Following the advice in Callaway, Goodman-Bacon, and Sant'Anna (2021), we compare each separate dose group to the never treated group in separate regressions.

In our first approach, we use the matched sibling-household data and carry out a simple difference-in-differences estimation. Given matched siblings live in the same household, with the same lead piping exposure before and after treatment, household and area characteristics will be the same between siblings, and we should be able to recover the ATT with this approach, given our assumptions.

First we exclude households without siblings either side of the treatment divide. That is, we only consider households which have at least one older sibling born before 1st of September 1990, and at least one younger sibling born after this date. We take the difference between the siblings' outcomes within the household. If there are more than one sibling on one side of the treatment divide we average their outcomes as shown in below:

$$(1) \qquad Household\_Difference_h = \frac{\sum_{j=1}^{J} Y_{hj}}{n_{h1}} - \frac{\sum_{i=1}^{I} Y_{hi}}{n_{h0}}$$

Where the *Y* is one of three outcomes outlined in the data section, *h* is the household identifier, and *j* is the individual identifier of a sibling born before treatment, and *i* for an individual born after treatment, n_{h0} is the number of siblings in household *h* born before treatment and n_{h0} the number born after. We expect this to be negative on

average, as older siblings tend to outperform younger ones (see Keller, Troesch, and Grob, 2015; Lehmann, Nuevo-Chiquero, and Vidal-Fernandez, 2016; or Havari and Savegnago, 2022).

We then average these household differences for the Control sample, the Low Lead sample, and the High Lead sample for all three outcomes. Finally, we take the difference-in-differences using these means.

(2) $$\hat{\theta} = \frac{\sum_{h \in G_1} Household\_Difference_h}{N_{G_1}} - \frac{\sum_{h \in G_2} Household\_Difference_h}{N_{G_2}}$$

Where $\hat{\theta}$ is our difference-in-differences estimate of the ATT, G is the sample group (Control, Low Lead, or High Lead), and N_G the number of households in that group.

Our second approach uses the whole sample, but as we only observe each child once we must average outcomes at the school level. The baseline two-way fixed effects difference-in-difference specification is in (3).

(3) $$Y_{st} = \alpha + \theta\, Treat_s \times Post + \boldsymbol{X}_{st}\boldsymbol{\beta} + \gamma_s + \lambda Post + \epsilon_{st}$$

Where α is an intercept term, $Treat$ is an indicator variable for if a school lies within the Loch Katrine water supply area, $Post$ is an indicator for the time periods after 1st of September 1990, $\boldsymbol{X}_{st}$ is a vector of school level characteristics, γ_s are school fixed effects, and ϵ_{st} is the error term. We cluster our estimated errors by school. The variable of interest is θ , the coefficient on the interaction $Treat_s \times Post$. This is also an estimate of the ATT, but at the school level.

Given the different dosage groups, we also split the school treated areas into High Lead and Low Lead. For example, the estimate of the causal effect on the High Lead group can be recovered from (4).

(4) $$Y_{st} = \alpha + \hat{\theta}\, High_s \times Post_{st} + \boldsymbol{X}_{st}\boldsymbol{\beta} + \gamma_s + \lambda Post + \epsilon_{st}$$

Where the $Treat$ variable has been replaced with an indicator for if a school is in the High Lead area. (4) is estimated by excluding the Low Lead sample. Similarly, we can estimate the casual effect of Low Lead dosage by excluding the High Lead sample and estimating (4) but using an indicator for if a school is in a Low Lead area.

We also use an event study specification, to see the placebo effects of $Treat$ interacted with years before the treatment, and to see if the effect is monotonic after treatment. This specification is outlined in below.

(5) $$Y_{st} = \alpha + \lambda_t + \sum_{\tau=-q}^{-1} \delta_t \, Treat_{st} \; + \; \sum_{\tau=0}^{m} \theta_t \; Treat_{st} + \boldsymbol{X}_{st}\boldsymbol{\beta} \; + \; \gamma_s + \epsilon_{st}$$

Where *m* and *q* are the leads and lags. To check the effects of different dosages we exclude either High or Low Lead groups as before and check the event studies individually compared to the control group.

5. Results

5.1 Matched Sibling Difference-in-Differences

Table 4 panel A shows the average difference between siblings within a household, pre and post treatment for each dose group, as calculated in (1). As expected, older siblings tend to perform better than their younger siblings, within the same household across all outcomes, as can be seen by the negative signs. However, younger siblings in the High Lead area appear to perform better than their peers in the other dosage groups across the three outcomes. In contrast, the Low lead dosage group does not appear to perform better than the control group in Standard Grade points, and performs worse for the English credit outcome, but is better in the Mathematics Credit outcome.

In panel B of table 4 we calculate the difference-in-differences as in (2). In the first column we compare the Low Lead sibling differences to the control group sibling differences. Wide standard errors mean that Bonferroni corrected 95% confidence intervals cover zero for all outcomes except the Mathematics Credit outcome, where the effect of treatment on the Low Lead group is estimated to have increased the probability of achieving a Mathematics credit pass by 3.6 percentage points.

Table 4 – Differences Between Siblings in Same Housing, Pre and Post-Treatment

Panel A – Mean Sibling Differences

	Control	Low Lead	High Lead
Standard Grade Points			
Mean Sibling Difference	-31	-27	-15
Standard Deviation	(83)	(76)	(70)
Observations	37302	2228	1232
Mathematics Credit			
Mean Sibling Difference	-0.083	-0.047	-0.040
Standard Deviation	(0.518)	(0.516)	(0.510)
Observations	35361	2123	1222
English Credit			
Mean Sibling Difference	-0.069	-0.074	-0.030
Standard Deviation	(0.546)	(0.560)	(0.550)
Observations	36314	2202	1229

Panel B – Difference in Differences

	Low - Control	High - Control	High - Low
Standard Grade Points			
Difference-in-Differences	4	17	12
Standard Error	(2)	(2)	(3)
Mathematics Credit			
Difference-in-Differences	0.036	0.043	0.007
Standard Error	(0.012)	(0.015)	(0.018)
English Credit			
Difference-in-Differences	-0.005	0.038	0.043
Standard Error	(0.012)	(0.016)	(0.02)

Notes: Panel A shows difference between siblings born before treatment and siblings born after, averaged by dosage group. Panel B shows the difference-in-differences estimate between the averaged differences in panel A.

In the second column we take estimate difference-in-differences for the High Lead minus the control group sibling difference. Here the results are clearer. The treatment is estimated to increase the Standard Grade points achieved by 17, and the probability of achieve a mathematics credit pass by 4.3 percentage points. The point estimate for the

increase in probability of an English credit pass is 3.8 percentage points, but Bonferroni corrected 95% confidence intervals cover zero for this outcome.

In the third column we compare High and Low lead treatment areas. Here, the point estimates are all positive, suggesting dosage does make a difference and younger siblings in High Lead areas perform relatively better after the treatment than those in Low Lead areas. However, Bonferroni corrected 95% confidence intervals cover zero for all but the Standard Grade points outcome.

The sibling difference results in table 4 suggest it is only in High Lead areas that there is a socially significant difference in education outcomes after treatment. We next move on to the school level difference-in-differences.

5.2 School Level Difference-in-Differences

In table 5 we present the two-way fixed effect estimates for the full treatment and control sample. We present estimates for all three outcome variables, with and without school level covariates. All the point estimates are positive, suggesting the lower lead resulting from the water treatment may have had an effect, but Bonferroni corrected 95% confidence intervals cover zero for all estimates.

In table 6 we present the two-way fixed effect estimates for only the High Lead and Control schools, excluding those in the Low Lead zone of the Loch Katrine water supply area. This decreases the potential bias and lowers the misidentification risk resulting from differential treatment dosage as shown in Callaway, Goodman-Bacon, and Sant'Anna (2021). In all cases the point estimates are higher than in table 5, where we use the whole of the Loch Katrine water supply area. The estimates in Panel A suggest that the treatment increased by 18 points the average Standard Grade points achieved in High Lead area schools. Bonferroni corrected 95% confidence intervals do not cover zero in either case. The estimates in panel B suggest the treatment increase the proportion of pupils achieving a credit pass in mathematics by around 5 percentage points. Bonferroni corrected 95% confidence intervals do not cover zero for the estimate without school level covariates but do when these covariates are added. Panel C suggests the treatment increases the proportion of students achieving a credit pass in

English by around 1-2 percentage points, but the Bonferroni corrected 95% confidence intervals cover zero in both cases.

Table 5– Total Loch Katrine Water Supply Area, School Level Difference-in-Differences

	(1)	(2)
Panel A – Standard Grade Points		
Treatment × Post	11.029	10.505
	(5.72)	(5.818)
Observations	727	663
Unit Level Covariates	No	Yes
Panel B – Mathematics Credit Pass Share		
Treatment × Post	0.049	0.048
	(0.022)	(0.023)
Observations	718	654
Unit Level Covariates	No	Yes
Panel C – English Credit Pass Share		
Treatment × Post	0.007	0.008
	(0.017)	(0.019)
Observations	722	658
Unit Level Covariates	No	Yes

Notes: Table shows difference-in-differences estimation of school level average outcomes between treated schools and control schools. Standard errors are clustered by school and presented in brackets. Column (1) is estimate without school level covariates, and column (2) with. Covariates include index of multiple deprivation quintile, share of boys in school, and the local youth unemployment rate.

Table 6 – High Lead Areas, School Level Difference-in-Differences

	(1)	(2)
Panel A – Standard Grade Points		
Treatment × Post	18.662	18.204
	(4.931)	(4.936)
Observations	680	616
Unit Level Covariates	No	Yes
Panel B – Mathematics Credit Pass Share		
Treatment × Post	0.056	0.054
	(0.019)	(0.022)
Observations	671	607
Unit Level Covariates	No	Yes
Panel C – English Credit Pass Share		
Treatment × Post	0.016	0.017
	(0.014)	(0.016)
Observations	675	611
Unit Level Covariates	No	Yes

Notes: Table shows difference-in-differences estimation of school level average outcomes between High Lead area treated schools and control schools. Standard errors are clustered by school and presented in brackets. Column (1) is estimate without school level covariates, and column (2) with. Covariates include index of multiple deprivation quintile, share of boys in school, and the local youth unemployment rate.

In table 7, we show the same results but comparing the Low Lead areas to control areas, excluding the High Lead area schools. Here again all points estimates are positive, but Bonferroni corrected 95% confidence intervals cover zero in all cases. The point estimates are lower than for the High Lead sample for Standard Grade points and for Mathematics Credit passes, but higher for English Credit passes.

Table 7 – Low Lead Areas, School Level Difference-in-Differences

	(1)	(2)
Panel A – Standard Grade Points		
Treatment × Post	8.373	7.854
	(7.007)	(7.085)
Observations	711	647
Unit Level Covariates	No	Yes
Panel B – Mathematics Credit Pass Share		
Treatment × Post	0.047	0.045
	(0.027)	(0.028)
Observations	702	638
Unit Level Covariates	No	Yes
Panel C – English Credit Pass Share		
Treatment × Post	0.004	0.005
	(0.022)	(0.023)
Observations	706	642
Unit Level Covariates	No	Yes

Notes: Table shows difference-in-differences estimation of school level average outcomes between Low Lead area treated schools and control schools. Standard errors are clustered by school and presented in brackets. Column (1) is estimate without school level covariates, and column (2) with. Covariates include index of multiple deprivation quintile, share of boys in school, and the local youth unemployment rate.

As explained in section 4, the identifying assumption with difference-in-difference and continuous treatment when comparing each dose level individually with the control is parallel trends between the control group and the group that has a given dose level. This is analogous to the traditional parallel trends assumption and can be made conditional on covariates. When regressing with all dosages, stronger assumptions are required of either no selection into different does areas on average, or homogenous treatment effects.

In this section we show the school-level average means for each dose group, and event studies for each outcome and group, estimates using (5). Figure 3 shows the school-level mean outcomes over time. The Treatment group means are more volatile as they have far lower sample sizes. For the High Lead group, starting from a lower base, there is convergence in outcome post-treatment with the control group. The High Lead group

has a higher mean standard grade points achieved, and mean Mathematics Credit passes achieved by the end of the period but remains lower for English. There is less convergence for the Low Lead group, and this group appears to match the patterns on the control group before and after treatment.

To test whether the effects observed in our two-way fixed effects are spurious, we perform event studies. These show the coefficient on treatment interacted with each year of our sample. Figure 4 shows the results for the full sample. There does appear to be some increase in coefficient size after treatment, but the confidence intervals are wide.

Figure 5 shows the event study for High Lead compared to control. Here we see larger increases in the coefficient after treatment than in figure 4 for Standard grade points and Mathematics credit passes, but not for English Credit passes. The confidence intervals are also tighter, widening at the end as we get further from the reference year (1989), as is standard with event studies. Figure 6 presents the same results for the Low Lead sample. Here there is some upturn in the coefficients after treatment year, but the effects are more muted than for the high lead sample and the confidence intervals are extremely wide throughout.

Figure 3 – Mean School Outcomes per Dose Group

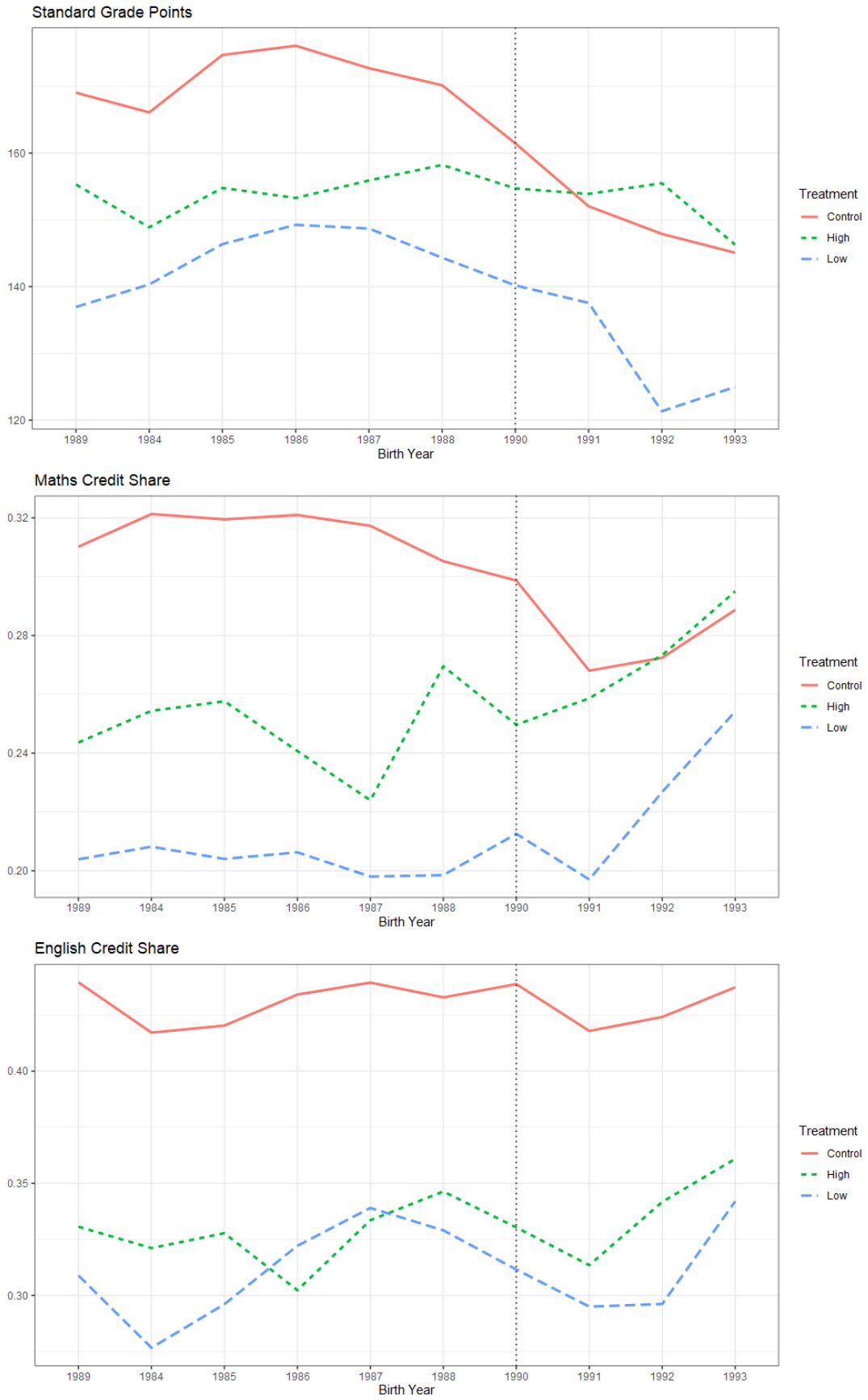
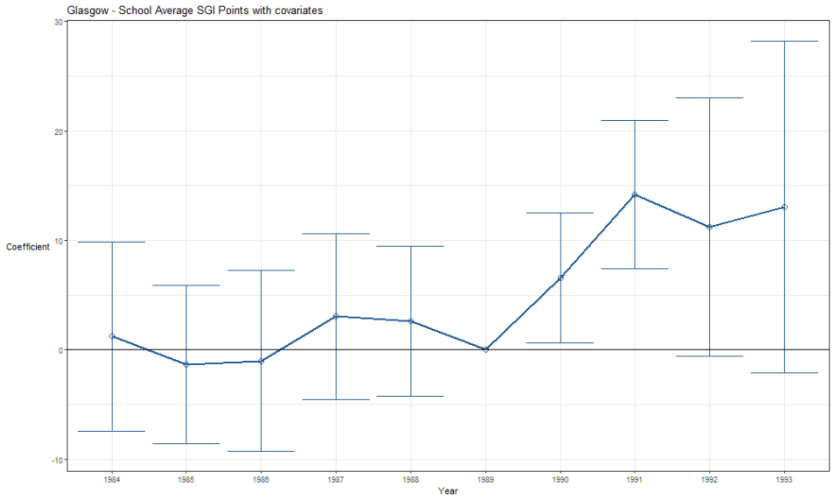
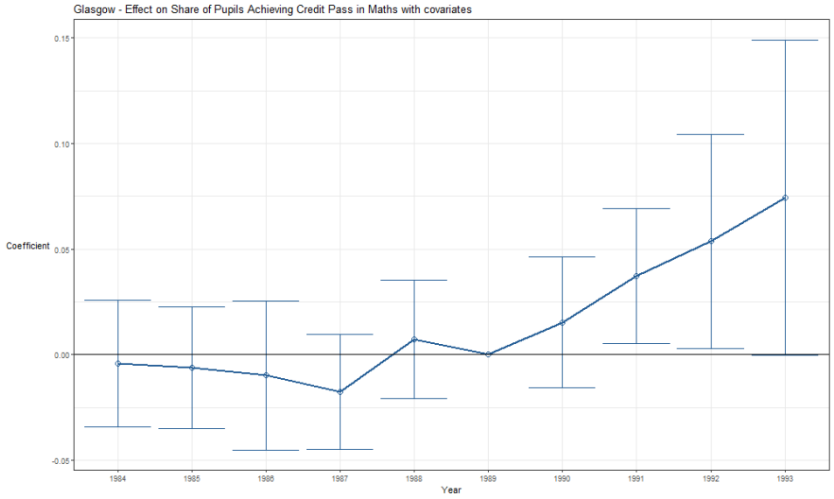
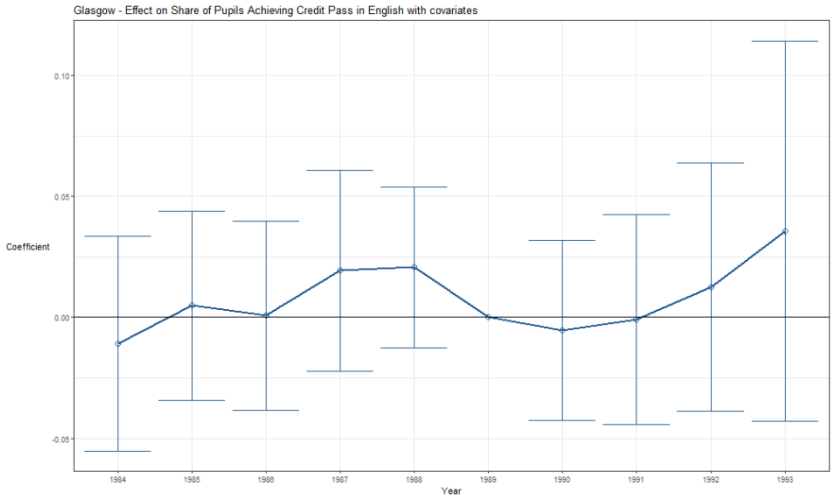


Figure 4 – Event Studies, All Treatment Areas

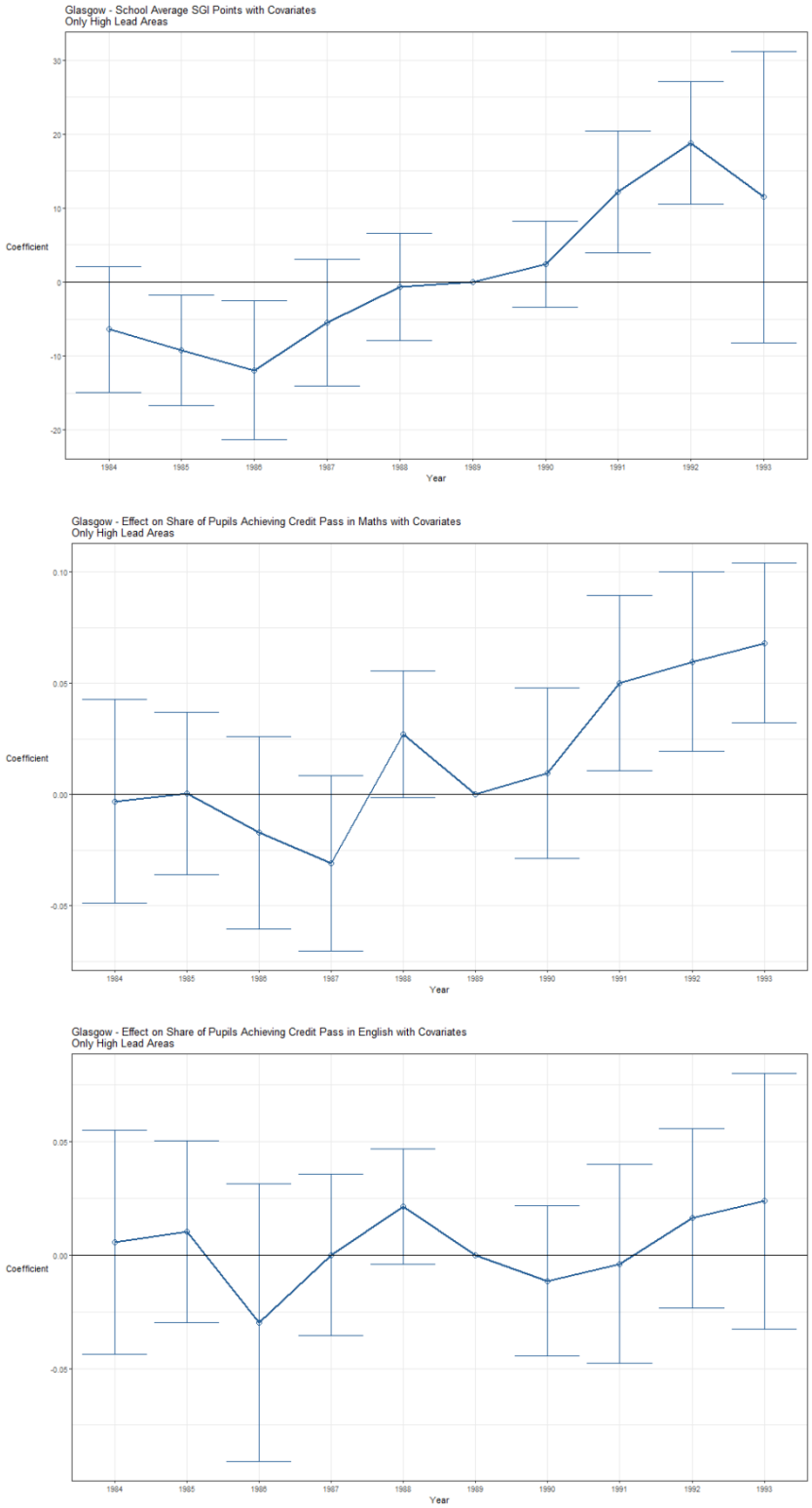






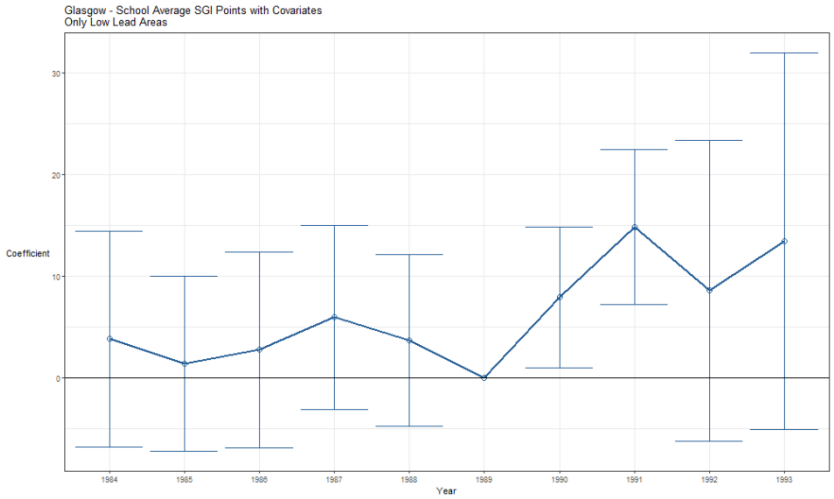
Notes: Charts show event study estimations of an indicator variable for treatment interacted with the year of birth. The outcome variable is the school level-average indicated in each chart heading. Outcomes are averaged by school, and then dosage group. Errors are clustered at school level.

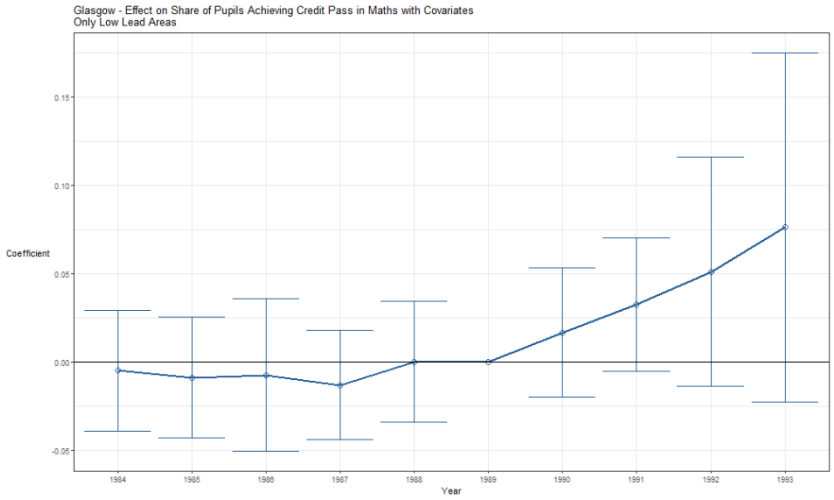
Figure 5 – Event Studies, High Lead Areas

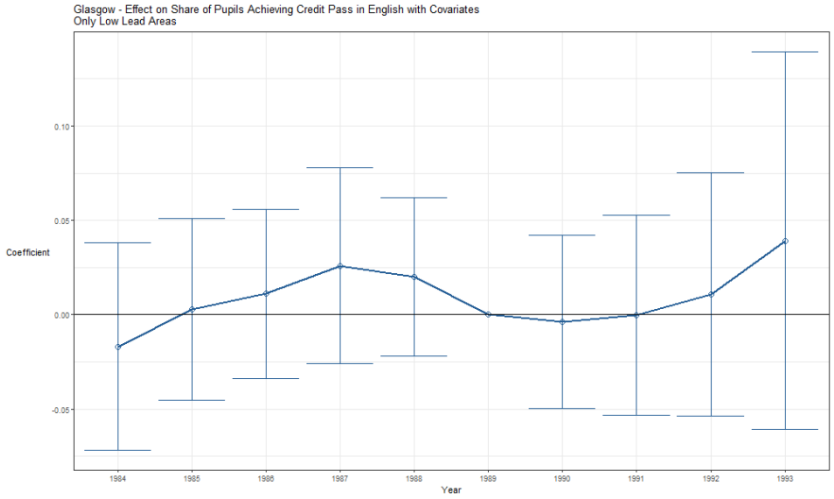


Notes: Charts show event study estimations of an indicator variable for treatment interacted with the year of birth, but Low Lead areas of the treatment group are excluded from the estimation. The outcome variable is the school level-average indicated in each chart heading. Outcomes are averaged by school, and then dosage group. Errors are clustered at school level.

Figure 6 – Event Studies, Low Lead Areas







Notes: Charts show event study estimations of an indicator variable for treatment interacted with the year of birth, but High Lead areas of the treatment group are excluded from the estimation. The outcome variable is the school level-average indicated in each chart heading. Outcomes are averaged by school, and then dosage group. Errors are clustered at school level.

6. Discussion and Conclusion

We estimated the effect of lower infant lead ingestion from drinking water on later educational outcomes using plausibly-exogenous variation from a water treatment programme in Glasgow, Scotland in 1989. Our results suggest that lower lead ingestion from water when an infant, and lower maternal lead ingestion when a child is in the womb, leads to better grades at age 16. However, our results show that the positive effects are concentrated within the areas of high lead pipe prevalence. The levels of lead in the water in Glasgow before the 1989 treatment were generally higher than those seen in Flint, Michigan in 2015. Even after treatment, the distribution of lead-water levels in the High Lead area was similar to that of Flint in 2015 (Table 1). This implies that socially significant improvements in education outcomes will only be seen when the reduction in lead pollution is large.

This is in line with the literature on lead outcomes that shows the does-response effects are non-linear. Grönqvist, Nilsson and Robling (2019), show that the effects of lead are low until a threshold of around 5 μg/dl blood lead levels. Reyes (2007) shows that effects are far stronger for the 4th quartile of lead exposure, in some cases 20 times as large as for the 1st quartile. Sampson and Winter (2018) show a clear non-linear increasing relationship between infant blood lead levels and anti-social behaviour in teenagers, with no effect below 5 μg/dl. Our results are in contrast to those arguing the marginal effects are higher at low levels of lead, or that the effects of lead are linear.

There are a number of limitations to this study. Firstly, the treatment group is concentrated in one urban centre, with treatment at one point in time. This potentially limits the external validity of our results. Glasgow notoriously has a number of unexplained poor health outcomes (known as the "Glasgow Effect") and although we estimate the ATT, this tells us little about the effects of treatment on the control group. Secondly, although there is a reasonably large sample at the individual-sibling level differences in table (3), at the school level averages the sample is very limited. It may be that our failure to find larger effects for the low leads sample is related to the small number of schools within Glasgow, and the measurement error in assigning High or Low lead to children within a school's catchment area. A third limitation is that education is a high-order outcome, with many contributing factors. Our results say

nothing about other, more direct effects, such as those on health, which likely have a different dose-response relationship.

The implications of our findings are that the gains from lead abatement on education are non-linear. Therefore, lead abatement programmes and infrastructure spending should first be targeted at those with the highest levels of lead ingestion when this is possible. This is especially important in low and middle-income countries, where the average blood lead levels are far higher (GBD, 2019). By some estimates 1 in 3 children have blood lead levels above 5 μg/dL, and 280 million in India alone (GBD, 2019). Therefore, while lead abatement in low lead areas may have some benefit, when discretion is possible, resources should be targeted at areas and countries with much higher blood lead levels. We recommend future research on the effect of lead on higher order outcomes, like education, not only test whether an effect is socially significant, but also attempt to map the shape of the dose-response relationship.

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