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Abstract

This thesis consists of three empirical studies on the effects of exposure to heavy metal pollution in early childhood on a broad set of individual outcomes. The first study analyses how accumulated exposure to metal pollution during childhood affects long-run outcomes. Exploiting policy-driven reductions in metal pollution in Sweden, it shows that accumulated exposure to metals (including cadmium, chromium, copper, lead, nickel, vanadium, and zinc) leads to lower GPA scores, fewer years of education, and reduced adult wages. It also shows that these effects may contribute to intergenerational persistence of socioeconomic status due to inequalities in pollution exposure driven by parental sorting.

The second study estimates the effect of lead pollution on infant mortality in five Sub-Saharan African countries. A sharp phase-out of leaded gasoline provides exogenous variation in changes in lead pollution between those living close to major roads and those living further away to identify a causal effect. The results show that the phase-out led to a large reduction in infant mortality, particularly among girls. This effect was driven by infants born to mothers with low socioeconomic status.

The third study investigates how exposure to lead pollution in early life affects cognitive skills among school age children in Uganda. Again, it relies on the phase-out of leaded gasoline as an exogenous shock to lead pollution levels. The findings suggest a strong negative effect of lead pollution on math and English test scores, which is stronger for children exposed to lead pollution at an earlier age.

Taken together, these studies contribute to our understanding of the benefits of more stringent environmental regulations regarding heavy metal pollution.

Key words

Metal pollution, accumulated exposure, lead pollution, GPA, education, earnings, infant mortality, cognitive skills

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To my mom

Contents

Acknowledgements

"I'm going on an adventure!"

-Bilbo Baggins, *The Hobbit*

I was just as excited to start my PhD program as Bilbo Baggins was to go on an adventurous trip to the Lonely Mountain. I expected the experience to be challenging, but rewarding and fun. What I did not know at that time is how lucky I would be to be accompanied by an amazing group of people who made my journey much less difficult and much more rewarding and enjoyable than it could have been. It is now time to express my sincere gratitude to all those people who helped me achieve my final destination.

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Chapter 1
Introduction

1.1 Background

This thesis consists of three independent studies exploring the negative effects of exposure to heavy metal pollution in early childhood. The subject is a part of a broader, rapidly growing economic literature on the detriments of environmental pollution to human well-being, which aids policy makers in assessing the benefits of pollution abatement measures. As such measures usually come with a cost (for producers, consumers, or governments), a careful evaluation of the pros and cons is needed.

One of the major objectives of pollution abatement policies has been improvement of human health. Although health is valuable in itself (as an important component of quality of life as well as one of the major recipients of public spending), the benefits of health promotion extend far beyond that. As health affects one's productivity, both directly and through its effects on other components of human capital (e.g. education and skills), it is an important determinant of economic growth (Bhargava et al., 2001; Schultz, 1996; Suhrcke and de Paz Nieves, 2011). This thesis recognizes the importance of accounting for all these factors during policy evaluation and therefore analyses the effect of metal pollution on a broad set of outcomes, including health, cognitive skills, education, and wages.

The focus of the thesis is on exposure in early life (which includes prenatal period and childhood). This is motivated by several factors. First, children are more vulnerable to pollution. Since they drink more water, eat more food, and breathe more air per kilogram of body weight, they are exposed to much higher pollution levels than adults. Their pollution exposure is also increased due to their hands-to-mouth behaviour and playing close to the ground. The same level of pollution can have larger impact on foetuses and young children due to their rapid growth and development that can be easily disturbed, and because their

immune and detoxification systems are not fully developed. In addition, children simply have more future years of life to develop a chronic decease triggered by earlier pollution exposure, as many such diseases can take several decades to develop (Landrigan et al., 2003).

Second, early life conditions have been shown to have a persistent impact on adult outcomes (see Currie and Almond (2011) for an extensive review of this literature). One of the explanations behind this persistence is that disruptions in developmental processes caused by health shocks in early life may cause permanent damage to body systems and functions. This results in chronic health conditions and, consequently, worse labour market outcomes. Another mechanism, suggested and formalized by James Heckman (see e.g. Heckman (2007) and Cunha and Heckman (2007)), works through the formation of "capacities" (such as health and cognitive and non-cognitive skills) over the individual's life-cycle. According to the model, a higher stock of capacities in one period rises the stock of capacities in future periods (the "self-productivity" effect); in addition, greater capacities produced in one period make investments in capacities in future periods more productive ("dynamic complementarity"). Therefore, even if the damage to one's capacities is temporary, it can have long-lasting effects. It is notable that the self-productivity and dynamic complementarity effects do not only occur within a specific capacity, but also between capacities. For example, poor health will negatively affect acquisition of skills and vice versa.

Finally, studying pollution exposure in early life overcomes several methodological challenges. First, it is much easier to estimate the lifetime pollution exposure for children than for adults due to limitations in historical pollution data (i.e., during the childhood of today's adults) as well as lack of data on individuals' place of residence throughout life. And second, health responses to pollution exposure are often more immediate in children than in adults, making it easier to identify any effects and lowering the risk of confounding.

1.2 Previous research

The link between pollution and human health has been long recognized. As far back as Ancient Greece, scientists noticed that miners and metallurgists often developed particular diseases, which were attributed to acid mists and toxic metals they were exposed to (EPA, 1993). Since then, a vast number of toxicological and epidemiological studies have documented negative health

effects of environmental toxicants. However, these studies provide only a partial picture on the extent of harm caused by pollution to human well-being.

While toxicological studies provide useful information on the mechanisms of action of various toxicants, they are not generally suited for quantifying the effects on human health, as they are predominantly based on animal studies. Epidemiological studies do attempt to quantify the relationship between actual exposure to toxins and human health, but they do not usually account for the endogeneity of pollution exposure. A non-random assignment of pollution can occur, for example, when health-cautious individuals sort into areas with cleaner environment and at the same time make other investments in their health, which leads to overestimation of the negative effect of pollution on health. An opposite direction of the bias may occur, for example, when highly educated people with more resources for health investments agglomerate in large cities, which tend to be more polluted than other areas.

Economic studies addressing these issues started appearing rather recently, following the landmark study by Chay and Greenstone (2003), who exploited geographic variation in air quality in the US caused by the 1981-1982 recession to estimate the relationship between pollution (measured by total suspended particles) and infant mortality rate. Subsequent economic studies, employing a variety of quasi-experimental techniques, established causal links between exposure to various pollutants and birth outcomes (Currie and Walker, 2011; Currie et al., 2015), infant mortality (Currie et al., 2009b; Agarwal et al., 2010; Knittel et al., 2016), and child respiratory health (Neidell, 2004; Coneus and Spiess, 2012; Jans, 2014). Economists have also extended the focus of analysis to labour market related outcomes, showing negative effects of pollution on school attendance (Currie et al. 2009a), academic performance (Lavy et al., 2014; Bharadwaj et al., 2014), worker productivity (Graff Zivin and Neidell, 2012), labour supply (Hanna and Olivia, 2015), years of education and earnings (Nilsson, 2009).[1]

There is still much to learn, however, and the next section explains how the studies in this thesis contribute to the existing knowledge.

[1] For comprehensive reviews of the literature, see Graff Zivin and Neidell (2013), and Currie et al. (2014).

1.3 Contributions of the thesis

This thesis expands on our knowledge on the negative effects of environmental pollution on human well-being by (1) focusing on pollutants that have been examined very little in economic studies: heavy metals, (2) examining effects of cumulative exposure to pollution rather than exposure during a short period of time (Chapter 2), and (3) studying the effects of pollution in developing countries (Chapters 3 and 4).

1.3.1 Focus on metal pollution

Most previous studies focused on a small number of pollutants such as carbon monoxide, nitrogen dioxide, ozone, particulate matter, and sulphur dioxide. These pollutants come mainly from cars and are routinely monitored in many countries. Our knowledge on other toxicants, however, is very limited. Several studies evaluated health effects of exposure to industrial pollutants by using data on toxic releases (Currie and Schmieder, 2009; Agarwal et al., 2010) or exploiting openings and closures of toxic plants (Currie et al., 2015). However, these studies are unable to disentangle effects of specific pollutants.

This thesis sheds light on the effects of heavy metal pollution. There are at least two important characteristics that distinguish heavy metals from previously studied pollutants. First, they have a very long biological half-life in the human body. For example, lead and cadmium may require several decades to be excreted (Nordberg et al., 2007). Therefore, unlike the previously studied pollutants, metals can intoxicate the body for many years after initial exposure. Second, some metals can affect the nervous system. Therefore, their negative effects may include not only deterioration of physical health, but also behavioural changes and impairment of cognitive and non-cognitive skills. While several previous studies evaluated the negative effects of lead pollution (Clay et al., 2010; Nilsson, 2009; Grönqvist et al., 2014), very little attention has been paid to other heavy metals in economic research.

1.3.2 Evaluating effects of cumulative exposure

To my knowledge, only one economic study (Beatty and Shimshack, 2014) has evaluated the negative effects of cumulative exposure to pollution (and only during one year). Such a gap in the literature is driven by the lack of data, since

the researcher must be able to track individuals across time and space and measure their pollution exposure at each point in time. In addition, estimating the causal effect of accumulated pollution exposure is challenging due to difficulties in finding sources of exogenous variation in exposure to pollution over a prolonged period of time.

Such analysis is, nevertheless, very important since the effects may be different compared to contemporaneous exposure. Evaluating the effects of cumulative exposure is especially important with metal pollution since many metals accumulate in the human body and therefore their level of toxicity may increase with time. Using Swedish register data that contains individuals' municipality of residence throughout their lifetime, I am able to estimate the effect of cumulative exposure to metal pollution on long-run outcomes in Chapter 2.

1.3.3 Providing evidence for developing countries

Most of the existing evidence on the effects of pollution on individual outcomes comes from developed countries. However, there are several reasons why these results may not be directly extrapolated to the developing country context. First, pollution levels in the developing world are usually much higher than in developed countries. If the negative effects of pollution are non-linear (that is, if the marginal effect is higher/lower at higher pollution levels), then relying on the estimates for developed counties to predict the effect in a developing country would be misleading.

Second, there may be other shocks to child health in developing countries that can amplify the negative effects of pollution. For example, malnutrition (especially deficiency in essential minerals such as calcium, zinc and iron) may lead to higher lead absorption and thus higher risk of lead toxicity (Skerfving and Bergdahl, 2007).

Finally, people in developing countries may have limited possibilities to avoid pollution (for example, if they spend a large amount of time outside due to the nature of their work) or do not have the knowledge of how to avoid pollution. Additionally, individuals in developing countries may have limited possibilities to mitigate and compensate for the negative effects of pollution (for example, due to lower quality and accessibility of medical care or lack of resources to make other investments in human capital). All these factors would aggravate the negative effects of pollution at a given concentration level.

Evaluating the effects of pollution in developing countries is difficult due to limitations in available data as well as the lack of exogenous variation in pollution in these countries. Chapters 3 and 4 of this thesis circumvent these

issues by exploiting the rapid phase-out of leaded gasoline in Sub-Saharan African countries to estimate the causal effect of lead pollution on infant mortality and cognitive skills.

1.4 Overview of the thesis

1.4.1 Chapter 2: Metal Pollution, Mobility, and Long-run Outcomes: Evidence from Sweden

This study estimates the effect of accumulated exposure to heavy metal air pollution during childhood on long-run outcomes, as well as analyses migratory responses to pollution. The accumulated exposure to pollution is calculated using data on individuals' municipality of residence throughout childhood combined with geospatial data on atmospheric heavy metal deposition. To identify causal effects, I exploit the fact that emissions of heavy metals decreased significantly in Swedish industries due to an active environmental policy starting in the early 1970s. The decline varied across municipalities and across time, providing spatial and temporal variation in metal pollution.

The results indicate that accumulated exposure to metals negatively affects long-run outcomes. For example, a one standard deviation increase in accumulated exposure to cadmium leads to a 0.04 standard deviations drop in GPA and a decline in education by around 0.13 years and in earnings by 2.5 percent. The effect is larger for individuals with low educated parents. I also find evidence of selective migration. Highly educated parents are more likely to out-migrate following an increase in pollution levels, while being an immigrant or a teenage mother lowers the likelihood of out-migration. The above findings that accumulated exposure to metal pollution negatively effects long-run outcomes and that parents with higher socioeconomic status are more likely to out-migrate from polluted areas have an important implication that pollution exposure may serve as a channel for intergenerational transmission of socioeconomic status.

1.4.2 Chapter 3: Lead Pollution and Infant Mortality in Sub-Saharan Africa: Evidence from the Phase-out of Leaded Gasoline

This study estimates the benefits of reducing lead pollution in developing countries by analyzing the effect of the phase-out of leaded gasoline in five Sub-

Saharan African countries (Ethiopia, Kenya, Uganda, Zimbabwe, and Burkina Faso) on infant mortality. I link data on road networks with geo-coded household survey data to identify those living close to major roads, and employ a difference-in-difference method comparing changes in infant mortality rates in areas close to major roads (affected by the phase-out) to those further away from major roads (not affected).

I find that the phase-out of leaded gasoline caused a substantial decline in infant mortality rates. Specifically, infant mortality in areas within 5 km from major roads fell by at least 7.4 percent during two years after the phase-out. The effect is driven by infants born to mothers with low socioeconomic status and is only present for girls. The results are robust to exclusion of control variables, inclusion of group specific trends, changing the time window around the policy change, and excluding children that could potentially be assigned treatment status incorrectly.

These findings provide a good example of how a relatively simple and inexpensive environmental protection measure can lead to tremendous improvements in population health, as well as reduction in the socioeconomic gradient in health, in developing countries.

1.4.3 Chapter 4: Early-life Lead Exposure and Cognitive Skills of School-age Children in Uganda: Evidence from the Phase-out of Leaded Gasoline

This study estimates the causal effect of early-life lead exposure on the cognitive skills of school-age children in Uganda. Again, the sharp phase-out of leaded gasoline in Uganda in late 2005 serves as a source of exogenous variation in lead pollution. The empirical strategy compares the changes in outcomes of same-aged children born (on average) two years apart and thus having a 2-year difference in the length of exposure to lead pollution in early childhood. The level of lead exposure is approximated by the traffic density in the child's district of residence, which is calculated based on the density of road networks and the annual average daily traffic of roads in the districts. The control group consists of children living in districts with very low traffic density, while there are three treatment groups comprised of children who live in districts with low, medium, and high traffic densities.

I find a strong and statistically significant positive effect of the phase-out of leaded gasoline on both English and math test scores of children aged 6 to 9 in districts with high average traffic density. The effect declines with child's age,

implying that lead pollution is more harmful during earlier years of life. As predicted, there is no effect on older children (aged 10 to 16), who experienced the decline of lead pollution at later ages, suggesting that the results for younger children are not driven by unobserved changes in the control or treatment groups.

The effect found in this study is much higher than the effect of the leaded-gasoline phase-out on 9th Grade GPA in Sweden (Nilsson, 2009), indicating that people in developing countries are more vulnerable to lead exposure than in developed countries. This aligns with prior studies showing that this is also the case for other pollutants (Arceo et al., 2016; Tanaka, 2015).

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Chapter 2
Metal Pollution, Mobility, and Long-run Outcomes: Evidence from Sweden

2.1 Introduction

The detrimental effects of air pollution on humans have received much attention in the recent economics literature. As pollution abatement policies around the world have become more stringent, an increasing number of papers have demonstrated beneficial effects of these measures for the society. For example, reductions of so-called "criteria" pollutants[2] in the ambient air have been shown to improve birth outcomes (Currie et al., 2009; Currie and Walker, 2011), infant mortality (Chay and Greenstone, 2003; Knittel et al., 2016; Arceo et al., 2016), child respiratory health (Neidell, 2004; Coneus and Spiess, 2012; Jans, 2014), and labour market related outcomes (Currie et al., 2009; Nilsson, 2009; Isen et al., 2014).

There is still much to learn, however. For example, few economic studies have analysed the effects of other, "non-criteria", air pollutants, as the data on exposure to them is scarce. In addition, we know very little about the effects of cumulative exposure to pollution during childhood on long-run outcomes. There are at least two reasons why studying cumulative exposure is important. First, many toxicants have a very long biological half-life and thus accumulate in the human body.[3] For example, lead and cadmium may require several decades

[2] The "criteria" pollutants include ozone, particulate matter, carbon monoxide, nitrogen oxides, sulfur dioxide, and lead. They are routinely monitored and heavily regulated in many countries.

[3] Biological half-life of a toxicant is the time required to reduce its amount in the body by half.

to be excreted (Nordberg et al., 2007). Therefore, the levels of toxicity of these pollutants may increase with repeated exposure. Second, some of the long-run outcomes are of cumulative nature. For example, test scores reflect knowledge accumulated during a certain period of time. In this case it is important to account for all the exposure to a pollutant preceding the outcome measurement.

Estimating the causal effect of cumulative exposure during childhood on long-run outcomes is challenging, however. The researcher must be able to track individuals across time and space and measure their pollution exposure at each point in time. Second, if individuals' migration responses to pollution differ depending on their own or parental background characteristics, which may affect children's long-run outcomes on their own, the estimated effect of pollution may reflect both a biological effect of pollution and sorting. The issue of selective migration is of interest in itself as it may be an important mechanism behind the intergenerational persistence of socioeconomic status. For example, if parents with higher income or education are more likely to move to areas with cleaner air, the harm of pollution to their children's health may be smaller. Until now, very few studies have been able to document such parental sorting (see Section 2.2.3 for a review of economic studies in this area).

This paper attempts to fill the existing gaps in the literature by estimating the effect of accumulated exposure to heavy metal air pollution during childhood on long-run outcomes, as well as analysing migratory responses to pollution.[4] Data on metal pollution comes from the Swedish longitudinal moss surveys on atmospheric heavy metal deposition. The metals under study include cadmium (Cd), chromium (Cr), copper (Cu), lead (Pb), nickel (Ni), vanadium (V), and zinc (Zn). These data are combined with Swedish register data, which contains information about individuals' municipalities of residence throughout their lifetime. This allows me to calculate accumulated exposures to metals for almost each individual in nine cohorts born between 1973 and 1985. I estimate how these exposures affect the individual's Grade Point Average (GPA) in 9th grade, years of schooling, and adult earnings. I also analyse how the likelihood of out-migration from a more polluted municipality is affected by various parental characteristics (education, income, being an immigrant or a teenage mother).

To estimate the effects, I exploit the fact that emissions of heavy metals decreased significantly in Swedish industries due to an active environmental policy starting in the early 1970s. The decline varied across municipalities and across time, providing spatial and temporal variation in metal pollution. Some

[4] There is no clear definition of the term "heavy metal" in the literature. Various definitions have been proposed based on elemental density, gravity, atomic mass, or other properties of metals. In this study I will refer to heavy metals as metals with potential human toxicity.

additional (and arguably exogenous) factors also contributed to decline in metal pollution in Sweden during the same period. These are the decline in transboundary metal pollution from neighbouring countries and the phase-out of leaded gasoline. I estimate the relationship between metal pollution and long-run outcomes using a panel data model with municipality and year of birth fixed effects and a large set of individual and municipality level controls to account for potential confounding factors.

The results show that accumulated exposure to metals negatively affects long-run outcomes. For example, a one standard deviation increase in accumulated exposure to Cd leads to a 0.04 standard deviations drop in GPA, a decline in education by around 0.13 years and a reduction in earnings by 2.5 percent. The effect is larger for individuals with low educated parents. I also find evidence of selective migration. Highly educated parents are more likely to out-migrate following an increase in pollution levels, while being an immigrant or a teenage mother lowers the likelihood of out-migration. Income, on the other hand, plays a minor role in parental responses to metal pollution.

The rest of the paper is organized as follows. Section 2.2 briefly describes existing research on the effects of exposure to metals on health and labour market outcomes and reviews what is known on selective migration. Section 2.3 discusses the data used in this study. Section 2.4 explains the estimation strategy. Section 2.5 presents the results. Finally, Section 2.6 concludes.

2.2 Background

2.2.1 The epidemiology of metals

Metals are naturally occurring elements that are found throughout the Earth's crust. Pollution of the environment with metallic elements may occur naturally, for example through metal corrosion, atmospheric deposition, weathering, and volcanic eruptions (Tchounwou et al., 2012). Yet, most environmental contamination with metals results from human activities such as mining, smelting, fossil fuel combustion, and industrial application of metals. Once released into the air, metal particles may be transported long distances (sometimes more than 1000 km) until they are deposited. Humans may be exposed to metals via inhalation, ingestion, or skin penetration (Nordberg et al., 2007).

An increasing number of epidemiological studies attest the adverse health effects of metals.[5] Prolonged exposure to metals has been shown to have reproductive, developmental, and neurological effects on humans (e.g., lead is a known neurotoxin; cadmium has also been linked to adverse neurological effects). Several metals have been classified as carcinogens by the International Agency for Research on Cancer (IARC).[6] Various acute effects on the respiratory and gastrointestinal systems have also been documented (e.g. for cadmium, chromium, copper, nickel, and zinc) (EPA, 2014; Nordberg et al., 2007).

Most of this knowledge was gained by analysing occupational exposures or severe episodes of water, food, or air contamination. It is, however, less clear how the levels of metal pollution to which the general population is exposed today affect their health. Moreover, most epidemiological studies fail to account for endogeneity of pollution exposure stemming from, for example, residential sorting, avoidance behaviour, or confounding with other environmental pollutants.[7]

2.2.2 Economic studies on the effects of metal pollution

A number of economic studies have tried to estimate the causal effects of ambient metal pollution. Apart from addressing the issue of endogeneity, economists have also recognized that the negative effect of metal pollution extends beyond the mere health effects. As health is an important part of human capital, an increase in metal pollution may have negative consequences for economic growth. Consequently, the main focus of economic studies has been evaluation of the negative effects of metal pollution on labour market related outcomes, directly or indirectly through the effects on birth outcomes and child health.

For example, Currie and Schmieder (2009) analyse the effects of industrial toxicants on health at birth and infant mortality in the US using data from the Toxic Release Inventory (TRI).[8] To identify a causal link, the authors compare

[5] See Nordberg et al. (2007) for a comprehensive examination of the literature.

[6] These are arsenic, beryllium, cadmium, chromium, and nickel.

[7] Graff Zivin and Neidell (2013) describe these sources of endogeneity and their implications at length.

[8] The TRI tracks industrial releases of 300 to 600 toxic substances starting from 1988. Manufacturing plants in the US are required to annually report their releases of these substances to the Environmental Protection Agency (EPA).

the estimated effects of developmental toxicants with the effects of other chemicals. As expected, the developmental toxicants have much larger negative effects on birth outcomes and infant health than the non-developmental toxicants (implying that the results are not driven by other factors affecting toxic releases in general, such as e.g. economic cycles). The authors also look at the effects of a number of specific substances, including cadmium and lead. They find that a two-standard-deviation increase in lead emissions decreases gestation by 0.008 weeks and decreases birth weight by 1.8 grams. A similar increase in cadmium emissions reduces gestation by 0.012 weeks and reduces birth weight by 2.4 grams; it also increases the probability of low birth weight (< 2500 grams) by 1.2 percent, the probability of very low birth weight (< 1500 grams) by 1.4 percent, and the infant mortality rate by 5 percent.

Nilsson (2009) analyses the effect of the sharp phase-out of leaded gasoline in Sweden on long-term outcomes. He exploits the fact that the decline of lead exposure varied between the municipalities and time periods due to differences in initial concentrations. The results suggest that exposure to lead pollution in early childhood has a negative effect on cognitive ability, scholastic performance, and labour market outcomes (such as earnings and welfare dependency). These effects were observed at relatively low levels of exposure. The author also finds that the effects are stronger for children with low socioeconomic status.

Carson et al. (2011) examine the effect of water-borne arsenic exposure on labour supply in Bangladesh. They exploit high variation of arsenic content in tube wells. Since households were generally unaware of the arsenic contamination, the endogeneity of exposure is reduced. The authors find that arsenic pollution leads to an 8% reduction in labour supply.

This paper contributes to the existing literature by evaluating the long-run effects of metals that have not been investigated previously. Moreover, unlike other studies, it looks at cumulative exposure to metal pollution.

2.2.3 Economic studies on selective migration

Since selective migration may introduce endogeneity bias to the estimates of the effect of pollution on long-run outcomes, knowing its sign and magnitude is of critical importance. A handful of previous studies have directly tested for selective migration of parents. The existing evidence is mixed. For example, Currie (2011) shows that mothers living close to a Superfund site in the US are

more likely to be white and college educated following a clean-up.[9] However, Currie et al. (2015) find that closure of toxic plants leads to lower likelihood of white college educated mothers living in close proximity.

A larger literature looks at the mobility of the general population in response to changes in pollution. These studies typically look at changes in the composition of the population in localities following changes in environmental quality.[10] Again, the evidence is mixed. For example, Greenstone and Gallagher (2008) compare the demographic characteristics of those living around 400 Superfund sites in Census years before and after clean-ups. They conclude that Superfund clean-ups are associated with economically small and statistically insignificant changes in the total population and the demographic composition of areas surrounding the sites. Banzhaf and Walsh (2008), on the other hand, find that areas situated within 0.5 miles of a TRI facility become less populated and poorer as releases increase.

In this paper I analyse parental selective migration by looking at whether mother's likelihood of emigrating from a more polluted municipality is affected by her (and the father's) characteristics.

2.2.4 Research design

Taking a cue from Nilsson (2009), I exploit reductions in metal pollution that varied across municipalities and time periods as a result of vigorous environmental policy. The main driving force behind these reductions was the Swedish industrial pollution control program. The Swedish Environmental Protection Act, adopted in 1969, set out a strategy that was based on heavy use of performance standards, environmental innovation, and close cooperation between controller and polluter (including jointly funded R&D programs) (Bergquist et al., 2013). As described by Lundqvist (1980), the Swedish regulatory approach employed means that are "technologically practicable and economically feasible". This implies that the pollution abatement should not have had a tangible effect on companies' economic performance, which excludes

[9] Superfund sites are pieces of land that have been contaminated with hazardous waste and identified by the US EPA as candidates for a clean-up.

[10] These studies relate to the so-called "environmental justice" literature which is concerned with analysing the differences in exposure to pollution between different demographic groups.

an important source of potential confounding from this analysis.[11] As the pollution abatement strategies were negotiated with each plant owner (sometimes over prolonged periods of time) and due to differences in initial concentrations, the resulting reductions of pollution levels differed across municipalities and time periods. In addition to reductions stemming from industry, the levels of lead pollution dropped significantly due to the phase out of leaded gasoline carried out at the same time. As with industrial pollution, the traffic lead pollution declined unevenly between municipalities (Nilsson, 2009).

The scope of the resulting reductions in emissions was dramatic. During 1970-1985 alone the air emissions from point sources and traffic in Sweden decreased by around 90% for chromium, 80% for cadmium, nickel, copper, and zinc and 57% for lead (Naturvårdsverket, 2000).

An additional source of metal pollution in Sweden (especially the southern regions) has been long-range transport from Europe (Kindbom et al., 2001). Declines in emissions across Europe during the period under study contributed significantly to the reduction in Swedish metal pollution (Pacyna et al., 2009; Harmens et al., 2010).

I estimate the relationship between metal pollution and long-run outcomes using a panel data model with municipality and year of birth fixed effects. Under the assumption that the above mentioned pollution control measures do not affect other unobserved individual and municipality characteristics that determine long-run outcomes, the results reflect the causal effect.[12]

[11] If plants had to reduce production or shut down as a result of these measures, the economic conditions in municipalities could worsen and thus negatively affect child outcomes. However, this would lead to a *downward* bias in the estimates of the impact of metal pollution.

[12] I examine the reasonableness of this assumption with robustness checks throughout the paper.

2.3 Data

The main source of data in this study is Swedish administrative data from Statistics Sweden covering the entire Swedish population born between 1973 and 1985. It includes the Income Tax Register (which provides data on the annual income of both the child and his/her parents); Educational Register (data on years of schooling for the child and parents); Grade 9 Register (data on GPA in grade 9);[13] Medical Birth Register (birth-related data and information on mother's civil status); and Total Population Register (data on mother's immigration status and child's municipality of residence on December 31 of each year).[14] The Multigenerational Register is used to link children with their fathers. I also use data on population size in municipalities as one of the municipality level controls.

The data on metal pollution come from Swedish nationwide surveys on atmospheric heavy metal deposition, which use carpet-forming mosses as bio-monitors.[15] The surveys have been undertaken by the Swedish Environmental Protection Agency every five years since 1970, and contain data on air concentrations of arsenic, cadmium, chromium, copper, lead, nickel, zinc, vanadium, iron, and mercury. Every observation in the dataset gives a concentration of a certain metal in μg per kg of moss at a specific sampling site, and represents an average concentration over the three years prior to the sampling (which is performed during the summer months). In each survey there are about 700-1000 sampling sites that are distributed across the country, with somewhat higher concentrations in areas where high levels of metal pollution were expected (See Figure A.1).

[13] The 9th grade is the last year of compulsory schooling in Sweden and is typically completed at the age of 16. The GPA data is available only for children born up until 1980.

[14] The borders of Swedish municipalities changed somewhat throughout the period under study. Most of the changes involved merging of municipalities into larger ones. I assign municipalities to individuals according to the current division. However, since several pairs of municipalities merged together and then split back into two, I join these municipalities in my dataset. This is because I cannot distinguish between the two municipalities during the years when they were merged. The resulting number of municipalities in the dataset is 277.

[15] These mosses are used to measure deposition rates of metals with accuracy similar to (or even higher than) that obtained from direct deposition measurements at a much lower cost. This provides a larger number of sampling sites and greater spatial detail. See e.g. Rühling and Tyler (2001) for more information on the method. The data is available at: <http://www3.ivl.se/db/plsql/dvsmossa$.startup>.

In this study I focus on seven of the metals covered by the surveys, for the years 1975 through 2000. I exclude arsenic since data for this metal is absent in the 1990 survey; I also exclude iron and mercury for which the data is available only from 1985. Moreover, some studies indicate that the moss surveys data for Cr and Ni may not adequately reflect actual pollution levels. For example, Ross (1990) compared moss concentrations of several metals (including all the metals in this study) with pollution data obtained from the traditional (wet deposition) method in Sweden. While he found strong correlations between pollution levels measured by the two methods for most of the metals, correlations for Cr and Ni were weak and statistically insignificant. In a similar study for Norway, Berg et al. (1995) observed similar results for Cr, but not for Ni (which was found to be measured adequately well by the moss method compared to the wet deposition method). Given this evidence, the results of Cr and (perhaps) Ni should be treated with caution.

To assign pollution levels to municipalities, I first calculate pollution levels in 1 km^2 cells using inverse distance weighted interpolation. That is, each cell's value is determined as an average of the values of several closest sampling points,[16] weighted by the inverse distance between the cell and the sampling point. A municipality pollution level is then calculated as an average of the cell values situated within its borders.

To assess the effectiveness of this interpolation method in predicting cells' values, I performed a cross-validation exercise. It involves subsequent removal of one data point and then predicting its value using the above-stated interpolation method. Then the correlations between actual and predicted values are calculated. The results indicate very high predictive power for lead and cadmium (correlations are 0.88 and 0.86 respectively). For the rest of the metals the correlations are lower (between 0.62 and 0.71). This is expected since air concentrations of these metals are more localized (see Figures A.2-A.8). In the present analysis, however, capturing high-frequency spatial variation is not needed. This method should thus adequately predict pollution concentrations of these metals at the municipality level.

As was mentioned above, the pollution data are collected every 5 years and represent a 3-year average. In this analysis I treat this average as a measure of the annual level of pollution for the sampling year as well as for each of the two preceding years; the two years following the sampling year are missing. The

[16] The optimal number of closest sample points is chosen by the software (ArcGIS) automatically, with the range set manually to 5-10 points.

accumulated exposure for each individual is calculated according to the following equation:

$$M_i^m = \sum_{t=b_i}^{b_i+14} P_{ijt}, \quad (1)$$

where M_i^m represents accumulated exposure to metal m for individual i. P_{ijt} is the pollution level in municipality j where individual i resided during the year t; such municipality pollution levels are summed over the period from the year of birth b_i to the year when the individual turns 15 years old. I focus on children for whom pollution data during their birth year is available (i.e. those born in 1973-1975, 1978-1980, and 1983-1985), since exposure during gestation period and the first year of life may be of critical importance.[17] I exclude individuals for whom municipality information is missing for any of the years involved in the calculations (4.2% of the original sample).

Since the pollution data are missing for two years out of each 5-year period, the measure of accumulated exposure is a sum of exposures during several periods in childhood rather than during the whole childhood. To be more specific, for individuals born in 1973, 1978, and 1983 the accumulated exposure consists of exposures at ages 0-2, 5-7, 10-12, and 15; for individuals born in 1974, 1979, and 1984, it is the sum of exposures at ages 0-1, 4-6, 9-11, and 14-15; and for individuals born in 1975, 1980, and 1985, it is the sum of pollution levels measured at ages 0, 3-5, 8-10, and 13-15.

Finally, in order to control for precipitation, I obtained data on daily average precipitation levels per weather station from the Swedish Meteorological and Hydrological Institute. The municipality level yearly averages are then constructed using the same method as for the pollution data.

Table 1 reports summary statistics of the variables used in the analysis. It is clear from the table that the accumulated exposure to metals is significantly lower for individuals born in 1985 compared to those born in 1975 on average. Moreover, the variation of the within municipality difference between the 1975 and 1985 cohorts is quite high, which confirms the rationale behind the identification strategy used in this study.

[17] Sensitive periods are examined in more detail in Section 2.5.3.

Table 1: Summary statistics

	All cohorts			Within-municipality difference between the 1975 and 1985 cohorts			
Variables	Obs.	Mean	Std. Dev.	Mean	Std. Dev.	Min	Max
Panel A. Outcomes							
GPA in grade 9	569,544	3.21	0.73	-	-	-	-
Years of education	828,498	12.77	2.02	0.08	0.26	-0.78	0.87
Earnings (SEK 100)	843,944	1733.5	1093.3	344.8	146.7	-117.1	908.4
Panel B. Accumulated exposure to metals until age 15 (in µg per kg of moss)							
Cadmium	882,495	3.56	1.37	-2.19	0.44	-3.30	-0.55
Chromium	882,495	21.20	19.61	-14.5	15.44	-119.2	-2.42
Copper	882,495	66.61	19.02	-30.57	5.99	-58.55	-8.06
Nickel	882,495	22.11	9.39	-12.71	5.05	-45.55	-3.03
Lead	882,495	200.4	97.23	-152.5	38.49	-247.3	-35.4
Vanadium	882,495	39.04	14.19	-17.21	6.72	-32.3	5.84
Zinc	882,495	494.7	133.4	-214.5	32.93	-299.8	-58.89
Panel C. Controls variables (measured at birth)							
Child is male	882,496	0.49	0.50	-0.01	0.06	-0.15	0.21
Birth parity	882,496	1.88	0.98	0.14	0.14	-0.41	0.56
Multiple birth	882,016	0.02	0.13	0.00	0.02	-0.05	0.12
Mother is married	882,496	0.70	0.46	0.32	0.20	0.00	0.95
Mother is born abroad	882,496	0.11	0.32	0.00	0.05	-0.16	0.23
Mother's age (years)	882,012	27.00	5.02	1.65	0.71	-2.04	4.54
Mother's years of education	702,862	10.79	2.18	0.49	0.17	-0.18	0.87
Father's years of education	661,787	10.62	2.65	0.55	0.20	-0.20	1.10
Mother's income (SEK 100)	875,894	369.7	296.6	406.9	39.64	303.33	642.7
Father's income (SEK 100)	863,910	657.6	471.5	552.4	74.35	402.98	1040.0
Population (1000 people)	874,828	113.7	173.1	0.544	2.864	-19.16	16.74
Average income	874,828	433.0	197.7	459.9	43.72	391.4	760.6
% mothers with tertiary education	882,495	0.15	0.087	0.09	0.04	-0.07	0.23
% fathers with tertiary education	882,495	0.18	0.098	0.06	0.05	-0.07	0.16
Average yearly precipitation (mm)	874,828	1.78	0.38	0.65	0.24	-0.09	1.16

An important feature of the pollution data is that pollution levels of different metals are highly correlated (see Table A.1). Except for Cr, all the correlation coefficients are above 0.65 and reach as high as 0.96 between Cd and Pb. This is not very surprising since many of the metals come from the same sources. However, it implies that controlling for all the metals simultaneously may be problematic. For that reason, I focus on the one-metal models in the main analysis. While the estimated effects may capture influences of other metals, they will still give an indication of the extent of negative effects of metal pollution.

2.4 Estimation strategy

2.4.1 Accumulated metal exposure and long-run outcomes

To identify the effects of cumulative exposure to metal pollution I estimate the following equation:

$$y_{ijt} = \alpha + \beta_1 M_{ijt}^{m} + Z'_{ijt}\beta_z + \theta_t + \varphi_j + \varepsilon_{ijt}, \quad (2)$$

where y_{ijt} is a long-run outcome (GPA in grade 9, years of schooling at age 26, and (ln) annual earned income at age 26) of individual *i*, born in municipality *j* in year *t*. M_{ijt}^{m} is a measure of accumulated exposure to a certain metal m (Cd, Cu, Cr, Pb, Ni, V, and Zn). Z_{ijt} is a set of variables controlling for individual background characteristics and municipality characteristics measured at birth. It includes dummy variables for month of birth, birth order (2nd, 3rd, 4th or higher), mother's age (21-25, 26-30, 31-35, 36 or higher), mother's and father's years of schooling (12, 13-14, 15 or higher), an indicator for whether it is a multiple birth, whether the child is male, whether the mother is married, whether the mother is an immigrant, mother's annual earnings, father's annual earnings, average yearly precipitation in the municipality, average income in the municipality, population size, and the share of mothers and fathers with tertiary education.[18] The regressions for the GPA also control for municipality of residence in 9th grade. φ_j and θ_t are municipality and year of birth fixed effects respectively, and ε_{ijt} is the error term. In all regressions the standard errors are clustered at the municipality level.

I control for precipitation because it directly affects metal deposition and at the same time may affect child outcomes (e.g. by affecting the amount of time spent outdoors). The municipality characteristics should control for the changes in municipalities' economies that may have happened as a result of the environmental policy. The municipality fixed effects capture differences between municipalities that are constant over time, while the year of birth fixed effects capture national-wide changes.

[18] For the categorical variables with a significant share of missing observations (mother's/father's years of education and mother's civil status), I include a separate category for missing values to preserve sample size.

As was mentioned before, the correlations among metals are very high, which motivates the use of one-metal models. However, I also present the results from multi-metal models for comparison purposes.

2.4.2 Selective migration

The main obstacle to the identification strategy in Equation 2 is a possibility of endogenous migration. Namely, β_1 will be biased if changes in pollution affect migration decisions differently for parents with different characteristics which, in turn, affect child outcomes. For example, highly educated parents may move away from a polluted area due to health concerns, thus lowering their children's accumulated exposure to pollution compared to the exposure of children that stayed. If highly educated parents also invest more in their children's human capital, it may appear as if these children have better outcomes because of lower exposure to pollution. The effect of pollution on long-run outcomes will thus be overestimated.

I analyse selective migration by looking whether mother's likelihood of emigrating from a more polluted municipality is affected by her (and the father's) characteristics. To do this, I estimate an equation of the following form:

$$Prob_{ijt}^{k} = \alpha + \beta_1 M_{jt}^{m} + \beta_2 C_{ijb}^{c} + \beta_3 M_{jt}^{m} * C_{ijb}^{c} + Z'_{ijt}\beta_z + \theta_b + \varphi_j + \varepsilon_{ijt}, \quad (3)$$

where $Prob_{ijt}^{k}$ is an indicator variable for whether the mother i, residing in municipality j during year t, resides in a different municipality in 5 years' time. Such probability is estimated at four different ages of the child, $k = \{-5, 0, 5, 10\}$. If b is the child's year of birth, then $t = b + k$.

M_{jt}^{m} is the level of pollution of metal m in municipality j during year t; its coefficient shows whether mothers in general have higher likelihood of emigrating from municipalities that become more polluted. C_{ijb}^{c} denotes one of the parental characteristics c, measured at the year of child's birth. These include whether the mother/father is highly educated (has more than 12 years of schooling), mother's/father's annual earned income, whether the mother is an immigrant, and whether the mother is under 20 at the time of birth (teenage mother). This variable controls for different mobility of parents with different characteristics in general. Our coefficient of interest is β_3, which shows whether highly educated and/or well-paid parents are more likely to emigrate when pollution levels rise compared to poorer and less educated parents. If there is

indeed positive selective migration, we expect $\beta_3>0$ for parent's education and income, and $\beta_3<0$ for being an immigrant or a teenage mother.

I also control for other parental and municipality characteristics that may affect one's decision to migrate and, at the same time, be correlated with municipality pollution and/or parental characteristics under study. Vector Z_{ijt} includes mother's/father's age (except for regressions for teenage mother), average income in the municipality, and population size. As in Equation 2, I include municipality and year of birth fixed effects, φ_j and θ_b; ε_{ijt} is the error term. Standard errors are clustered at the municipality level.

2.5 Results

2.5.1 Long-run outcomes

2.5.1.1 Main results

Table 2 presents the estimated β_1 coefficients from Equation 2. I start by analysing the single-pollutant models and controlling only for individual level characteristics and fixed effects. The results in columns (1) to (3) show that most of the metals have statistically significant negative effect on the outcomes under study. The largest effects are observed for Cd, Cu, and Zn. For example, a one standard deviation increase in accumulated exposure to each of these metals leads to a reduction in GPA by around 0.04 standard deviations. Similarly, schooling is reduced by 0.13 years following a one standard deviation increase in accumulated exposure to Cd and Zn and by 0.08 following a similar increase in exposure to Pb. A one standard deviation increase in exposure to Cd also reduces income by 2.5 percent.

As the next step, I add municipality of birth characteristics as control variables. As was mentioned earlier, the Swedish industrial pollution control program should not have significantly affected firms' performances and therefore economic conditions in municipalities. If this is indeed the case, then controlling for municipality characteristics will not change the estimates of the effect of metal pollution on long-run outcomes. We see from columns (4) to (6) of the Table 2 that this is indeed the case as the point estimates are virtually unchanged. Therefore, bias stemming from unobserved changes in municipality economic conditions should be minimal.

Table 2: Effect of accumulated metal exposure on long-run outcomes

	Single-pollutant models *(without municipality controls)*			Single-pollutant models *(with municipality controls)*			Multi-pollutant models *(with municipality controls)*		
	(1) GPA	(2) Schooling	(3) Earnings	(4) GPA	(5) Schooling	(6) Earnings	(7) GPA	(8) Schooling	(9) Earnings
Cadmium	-0.0192***	-0.0912***	-0.0186**	-0.0216***	-0.0934***	-0.0198***	-0.0250**	-0.0796***	-0.0113
	(0.00635)	(0.0122)	(0.0074)	(0.0065)	(0.0131)	(0.00689)	(0.00986)	(0.0235)	(0.0111)
Chromium	-0.00016	0.00042	-0.0002	-0.00009	0.00048	-0.00017	0.00004	0.00036	-0.00008
	(0.00012)	(0.0003)	(0.00013)	(0.00011)	(0.0003)	(0.00013)	(0.00024)	(0.00051)	(0.00023)
Copper	-0.00197**	-0.00305**	-0.00130***	-0.00121***	-0.00249*	-0.00119***	-0.00077	0.00132	-0.00059
	(0.00081)	(0.00154)	(0.00043)	(0.00045)	(0.00146)	(0.00044)	(0.0005)	(0.00172)	(0.00054)
Nickel	-0.00057*	-0.00031	-0.00083**	-0.00047	-0.00041	-0.00081**	-0.00072	0.00021	-0.00035
	(0.00031)	(0.00135)	(0.00038)	(0.00029)	(0.00134)	(0.00038)	(0.00066)	(0.00187)	(0.00075)
Lead	-0.00015**	-0.0008***	-0.00023**	-0.00015**	-0.00085***	-0.00025***	0.00014	0.00048	-0.00006
	-0,00007	(0.00015)	(0.00011)	(0.00007)	(0.00016)	(0.00009)	(0.00016)	(0.00044)	(0.00017)
Vanadium	0.00052	-0.00280***	-0.0005	0.00024	-0.00351***	-0.00067	0.0008*	-0.00182*	0.00014
	(0.00039)	(0.00098)	(0.00056)	(0.00036)	(0.00095)	(0.00054)	(0.00041)	(0.00095)	(0.00056)
Zinc	-0.00026**	-0.00096***	-0.00022***	-0.00022**	-0.00094***	-0.00022***	-0.00009	-0.00071*	-0.00001
	(0.00012)	(0.0002)	(0.00006)	(0,00009)	(0.0002)	(0.00006)	(0.00015)	(0.00037)	(0.00015)
Observations	552,772	808,273	777,315	552,772	808,273	777,315	552,772	808,273	777,315

Notes: Columns (1) – (6) present coefficients from 42 separate regressions, while columns (7) – (9) show coefficients from 3 regressions. The significant estimates with unexpected sign are highlighted in grey. All regressions control for child and parental characteristics (as in Eq. (2)), as well as municipality and year of birth fixed effects. Standard errors (in parentheses) are clustered at the municipality level. * indicates statistical significance at the 10% level, ** at the 5%, *** at the 1%.

Columns (7) to (9) of Table 2 show results from estimating the models in which all the metals are included simultaneously. Only Cd retained its statistically significant negative effect on GPA, while only Cd, V, and Zn have statistically significant negative effects on years of education (Zn and V at 10% significance level only). In addition, several estimates turned positive, with V being statistically significant. The results from the multi-metal models may be biased due to possible multicollinearity. However, robustness of the estimates for Cd across the specifications gives some confidence in the results for this metal. From this point on all the regressions are based on single-pollutant models.

The fact that the effects for most of the metals disappear when they are included simultaneously may be a sign that the effects found for these metals in the single-pollutant models reflect the negative impact of one (or few) of the metals. This is important to keep in mind when interpreting the results for the single-pollutant models. Another important note is that the effects of Zn and Cu may to some extent reflect the impact of traffic exposure since motor vehicle emissions constitute a large source of Zn and Cu air pollution (Hjortenkrans et al., 2007; Johansson et al., 2009).

2.5.1.2 Effects by gender and socioeconomic status

This sub-section examines how the long-run effects of accumulated metal pollution vary across population sub-groups. Table 3 presents the results of estimating Equation 2 separately for males and females. In general, the coefficients for males are more precisely estimated. The effects on earnings are also generally larger in size for males than for females. The effects on GPA and schooling, however, are larger for males only for Cu and Ni.

To analyse the differences in the effects between socioeconomic groups, I estimate Equation 2 separately for children from higher- and lower-income families, and separately for children with high- and low-educated parents.[19] A child belongs to a higher-/lower-income family if his/her parents' combined earnings are above/below the median at the year of birth. A child has highly-educated parents if at least one parent has more than 12 years of schooling;

[19] This type of heterogeneity could arise for several reasons: 1) if children with lower socioeconomic status are exposed to higher levels of pollution (for example, due to parental sorting, see Section 2.5.2); 2) if the same level of pollution has a stronger negative effect on children with lower socioeconomic status (for example, because these children have poorer health in general and are thus more sensitive to pollution); 3) if high-income or/and highly educated parents are able to avoid pollution to a higher degree (see Neidell, 2004); 4) if high-income or/and highly educated parents are more successful in compensating for negative effects of pollution.

Table 3: Effects of accumulated metal exposures on long-run outcomes by gender

	GPA		Schooling		Earnings	
	(1)	(2)	(3)	(4)	(5)	(6)
	Males	Females	Males	Females	Males	Females
Cadmium	-0.0211***	-0.0209**	-0.0925***	-0.0932***	-0.0236***	-0.0167**
	(0.00764)	(0.00972)	(0.0166)	(0.0146)	(0.00801)	(0.00815)
Chromium	-0.00008	-0.00009	0.00056	0.00043	-0.00029**	-0.00005
	(0.00017)	(0.00016)	(0.00037)	(0.00034)	(0.00013)	(0.00019)
Copper	-0.00175***	-0.00068	-0.00342**	-0.00157	-0.00098*	-0.00140***
	(0.00064)	(0.00049)	(0.00144)	(0.00163)	(0.00053)	(0.00045)
Nickel	-0.00079*	-0.00016	-0.00124	0.00042	-0.00127**	-0.0004
	(0.00042)	(0.00043)	(0.00140)	(0.00147)	(0.00049)	(0.00047)
Lead	-0.00019**	-0.00011	-0.00087***	-0.00083***	-0.0003***	-0.00016
	(0.00008)	(0.00009)	(0.00018)	(0.00019)	(0.00009)	(0.00011)
Vanadium	0.00011	0.00037	-0.00365***	-0.00337***	-0.00134**	-0.00006
	(0.00043)	(0.00050)	(0.00115)	(0.00106)	(0.00064)	(0.00058)
Zinc	-0.00025**	-0.00019*	-0.00096***	-0.00092***	-0.0003***	-0.00016**
	(0.00011)	(0.00009)	(0.00022)	(0.00022)	(0.00007)	(0.00007)
Observations	270,192	282,580	392,262	416,011	378,313	399,002

Notes: Each entry corresponds to a separate regression. All regressions control for child, parental, and municipality characteristics, as well as municipality and year of birth fixed effects. Standard errors (in parentheses) are clustered at the municipality level. * indicates statistical significance at the 10% level, ** at the 5%, *** at the 1%.

otherwise the child is assigned to a group with low-educated parents. Table 4 shows the results. As seen from the table, the effect of metal pollution on long-run outcomes does not differ significantly between children with higher- and lower-income parents. However, for some metals (particularly Cd, Pb, and Zn), the effect is much higher for children with low-educated parents than for children with highly-educated parents. The estimates are also more statistically significant for low-educated parents, but this could be due to a much larger sample size.

2.5.1.3 Critical periods

In this sub-section I analyse whether there are certain ages at which a child is most vulnerable to the negative effects of metal pollution. Economic literature points to particular susceptibility to environmental shocks during early childhood when growth is most rapid and the body's ability to fight

Table 4: Effects by parental socioeconomic status

	GPA				Schooling				Earnings			
	(1) High income	(2) Low income	(3) High education	(4) High education	(5) High income	(6) Low income	(7) High education	(8) High education	(9) High income	(10) Low income	(11) High education	(12) High education
Cadmium	-0.0244***	-0.0262***	0.00487	-0.0208**	-0.0939***	-0.111***	-0.0243	-0.111***	-0.0208***	-0.0227***	-0.00879	-0.0205***
	(0.00807)	(0.0076)	(0.0116)	(0.00878)	(0.0147)	(0.0152)	(0.0191)	(0.0168)	(0.00779)	(0.00738)	(0.0135)	(0.00603)
Chromium	-0.00007	-0.00007	-0.00012	-0.00009	0.00044	0.00065**	0.0003	0.00065*	0.000003	-0.00026*	-0.00015	-0.00011
	(0.00014)	(0.00014)	(0.00027)	(0.00013)	(0.00045)	(0.00028)	(0.0006)	(0.00035)	(0.00018)	(0.00014)	(0.00028)	(0.00015)
Copper	-0.0013***	-0.00108*	-0.00122	-0.00104*	-0.00221	-0.00299*	-0.00052	-0.00249	-0.00113**	-0.00113**	0.00023	-0.00101**
	(0.00049)	(0.00063)	(0.00082)	(0.00055)	(0.00144)	(0.00166)	(0.00135)	(0.00168)	(0.00044)	(0.00051)	(0.00077)	(0.00046)
Nickel	-0.00041	-0.00053	-0.00083	-0.00046	-0.00084	-0.00008	-0.00077	0.00083	-0.00017	-0.00134***	0.00044	-0.00092**
	(0.00037)	(0.00035)	(0.00079)	(0.00032)	(0.00144)	(0.00143)	(0.00201)	(0.00155)	(0.00064)	(0.00035)	(0.00075)	(0.00037)
Lead	-0.0002**	-0.0002**	0.000003	-0.00011	-0.00089***	-0.00101***	-0.00024	-0.00097***	-0.00034***	-0.00019*	-0.00012	-0.0002***
	(0.00008)	(0.00009)	(0.00014)	(0.00009)	(0.00017)	(0.00018)	(0.00022)	(0.0002)	(0.0001)	(0.0001)	(0.00021)	(0.00007)
Vanadium	-0.00026	0.00049	-0.00016	0.00051	-0.00302***	-0.00427***	-0.00133	-0.00310**	-0.00067	-0.00077	-0.00112	-0.00027
	(0.00044)	(0.00042)	(0.0008)	(0.00048)	(0.001)	(0.00117)	(0.00094)	(0.00124)	(0.00058)	(0.00059)	(0.00114)	(0.00046)
Zinc	-0.0003***	-0.00022**	-0.00008	-0.0002**	-0.00103***	-0.00098***	-0.00055**	-0.00108***	-0.0002***	-0.00024***	-0.00004	-0.00024***
	(0.00009)	(0.00011)	(0.00017)	(0.0001)	(0.00021)	(0.00022)	(0.00023)	(0.00023)	(0.00007)	(0.00007)	(0.00013)	(0.00006)
Observations	277,902	274,870	92,697	282,680	404,948	403,325	163,284	426,358	388,950	388,365	154,334	413,479

Notes: Each entry corresponds to a separate regression. All regressions control for child, parental, and municipality characteristics (as in Eq. (2)), as well as municipality and year of birth fixed effects. Standard errors (in parentheses) are clustered at the municipality level. * indicates statistical significance at the 10% level, ** at the 5%, *** at the 1%.

toxins is not fully developed.[20] However, young children potentially have capacity to recover from health shocks (Currie et al., 2009). Thus, for some toxicants more recent exposure may be more important. To explore which period of exposure (if any) has the greatest effect on long-run outcomes, I estimate Equation 2 replacing the measures of accumulated exposure with measures of pollution levels in the municipality of residence at ages 0, 5, 10, and 15 (in the same regression).

The results are presented in Table 5. The estimates for the GPA suggest that exposure to Cd, Pb, and V is most harmful in early childhood, while exposure to Cr, Cu, and Zn has negative effect only at the age of 15. Considering that Cd and Pb are neurotoxins, and some evidence suggest that IQ scores become stable after age 10 (see Hopkins and Brecht, 1975), it is possible that the negative effect of these metals on the GPA are manifested through their effect on cognitive skill formation. The fact that Cr, Cu, and Zn affect 9th grade GPA only at age 15 suggests a link of a more contemporaneous nature, for example short-run effects on health and/or cognitive performance.

The results for schooling are more uniform across ages. Exposure to Cd and V has a negative effect on schooling from birth and until the age of 15. However, exposure to Pb and Zn has an effect only until the child is five. The results for earnings suggest that exposure to Cd and Pb is harmful during both early and late childhood, while Ni, V, and Zn have a negative effect on earnings only when exposed at birth. The analysis of critical periods has to be treated with caution, however, since exposure to metals is highly correlated across ages (the correlations are especially high for Cd and Pb).

2.5.2 Selective migration

Table 6 presents the $\beta_3$coefficients from Equation 3. The results in Panel A (for k= –5) and Panel B (for k = 0) show strong evidence of positive selective migration during the five years before and after the child's birth. The vast majority of the point estimates are statistically significant and have the "right" sign (except for Cu and V in Panel A, and father's income in Panel B). The size of the coefficients is similar between the two panels, with the strongest effect observed for Pb, Cd, and Zn (the metals which also have the strongest effect on long-run outcomes). The probability of mother's living in a different

[20] See Currie and Almond (2011) for a comprehensive review of economic literature on the effects of early life shocks on long-run outcomes. Landrigan et al. (2004) explain in more detail why small children are especially vulnerable to environmental toxicants.

Table 5: Critical periods of the effect of metal pollution on long-run outcomes

	GPA				Schooling				Earnings			
Age (years):	0	5	10	15	0	5	10	15	0	5	10	15
Cadmium	-0.0818**	-0.0763**	0.0241	-0.00395	-0.207*	-0.380***	-0.139	-0.191**	-0.126***	-0.00813	0.0246	-0.0935*
	(0.032)	(0.0329)	(0.05)	(0.0394)	(0.107)	(0.0820)	(0.0953)	(0.0884)	(0.0344)	(0.0319)	(0.0422)	(0.0504)
Chromium	-0.00002	0.00111	0.00169*	-0.00475**	0.00136	0.00221	0.00384	-0.00590	-0.00070	0.00049	0.00051	-0.0031
	(0.00042)	(0.0007)	(0.001)	(0.00187)	(0.00136)	(0.00167)	(0.00254)	(0.00462)	(0.00052)	(0.00089)	(0.0009)	(0.00236)
Copper	0.0033	-0.00033	-0.00343	-0.00701***	-0.00999	-0.00373	0.0124**	-0.0244***	-0.00218	-0.00024	-0.00215	0.0001
	(0.0032)	(0.00182)	(0.00224)	(0.00225)	(0.0101)	(0.0036)	(0.00618)	(0.00614)	(0.00335)	(0.00249)	(0.00206)	(0.00234)
Nickel	-0.00055	0.00078	-0.00082	-0.00175	0.00374	-0.0277***	0.0101	-0.00698	-0.00367***	0.00785*	0.00385	0.00078
	(0.00138	(0.00405)	(0.00464)	(0.00494)	(0.00398)	(0.0106)	(0.0110)	(0.00943)	(0.00137)	(0.00399)	(0.00434)	(0.00387)
Lead	0.00024	-0.00092**	-0.00017	-0.00069	-0.00296***	-0.00307***	-0.00112	-0.00042	0.00007	-0.0008*	0.00034	-0.0028***
	(0.00035)	(0.00043)	(0.00061)	(0.00078)	(0.00108)	(0.00118)	(0.00164)	(0.00141)	(0.00035)	(0.00045)	(0.00064)	(0.00074)
Vanadium	-0.00031	-0.00513**	0.0044	0.00306*	-0.0136**	-0.0248***	0.00001	-0.0120***	-0.00649***	0.0005	0.00029	-0.00305
	(0.00177)	(0.00231)	(0.00276)	(0.00178)	(0.00611)	(0.00777)	(0.00368)	(0.00426)	(0.00196)	(0.00281)	(0.00232)	(0.00226)
Zinc	-0.00023	-0.00026	-0.00038	-0.00139***	-0.00302***	-0.00333***	-0.00114	-0.00145	-0.00087***	-0.00056	0.00031	-0.00044
	(0.00026)	(0.00047)	(0.00040)	(0.00051)	(0.00088)	(0.0011)	(0.00078)	(0.00095)	(0.00028)	(0.00038)	(0.00042)	(0.00041)

Notes: Each row represents an individual regression. The significant estimates with unexpected sign are highlighted in grey. All regressions control for child, parental, and municipality characteristics (as in Eq. (2)), as well as municipality and year of birth fixed effects. Standard errors (in parentheses) are clustered at the municipality level. * indicates statistical significance at the 10% level, ** at the 5%, *** at the 1%.

municipality following a one standard deviation increase in Pb pollution in the municipality of origin is about 1.8 percentage points higher if the mother is highly educated and 2.4 percentage points higher if the father is highly educated.[21] The corresponding estimates for Cd are 1.5 and 2.4 percentage points respectively, and for Zn they are 1.5 and 2.2 percentage points respectively. Being an immigrant or a teenage mother has an effect of a similar magnitude (around 2 percentage points decline in probability of migration) for the above metals. Income, on the other hand, plays a relatively small role in the environmental sorting of parents. If mother's income increases by 10%, her probability of emigration following a one standard deviation increase in metal pollution will increase at most by 0.06 percentage points. Father's income, while having a somewhat stronger effect on probability to emigrate before birth (up to 0.16 percentage points increase), has a negative effect after birth (up to 0.06 percentage points decline in probability).

As is apparent from Panels C and D, the magnitude of selective migration declines as the child gets older. The sizes of the coefficients decline between Panels B through D. A notable exception is father's income, which, for the most part, exhibits small in size but statistically significant *negative* selective migration after the child is born. This could imply that fathers with higher income are more driven by possible monetary gains from living (and working) in more polluted municipalities.

The analysis above has several important implications. First, the finding of positive selective migration implies that children born to parents with lower socioeconomic status are exposed to higher levels of pollution. If metal pollution indeed leads to worse long-run outcomes, such "environmental injustice" would reinforce the socioeconomic gradient in outcomes across generations. Second, the main driving force of the selective migration seems to be the ability of parents to process information about environmental quality, while the role of income is negligible.[22] This implies that raising awareness about the dangers of pollution may be the key strategy to reducing inequality in pollution exposures between socioeconomic classes. Finally, the results suggest that the socioeconomic gradient in environmental mobility declines with child's age. This could stem from the fact that young children are more susceptible to

[21] The mean probability of mother out-migrating within five years from k is 0.33 for k = -5 and 0.18 for k = 0.

[22] Income could be an important determinant of selective migration, for example, if increased pollution depresses housing prices (see e.g. Greenstone and Gallagher, 2008).

Table 6: Effects of metal pollution on the probability to migrate based on parental characteristics

	(1) Mother highly educated	(2) Father highly educated	(3) Mother's (ln) income	(4) Father's (ln) income	(5) Immigrant mother	(6) Teenage mother
Panel A. Models for migration during 5 years before the child's birth (k = -5)						
Cadmium	0.0781**	0.100***	0.0199**	0.0786***	-0.157***	-0.105**
	(0.03740)	(0.03240)	(0.00974)	(0.02170)	(0.04390)	(0.04440)
Chromium	0.00391***	0.00321***	0.00046**	-0.0001*	-0.00022	-0.00093
	(0.00095)	(0.00087)	(0.00021)	(0.00052)	(0.00063)	(0.00071)
Copper	-0.00250	-0.00211	-0.00229**	0.00656*	-0.0247***	-0.0115*
	(0.00351)	(0.00351)	(0.00092)	(0.00351)	(0.00886)	(0.00691)
Nickel	0.00876***	0.00794***	0.00088*	0.00128	-0.00334*	-0.00449*
	(0.00218)	(0.00182)	(0.00050)	(0.00161)	(0.00184)	(0.00232)
Lead	0.00127***	0.00133***	0.00025**	0.00079***	-0.00146**	-0.00149**
	(0.00030)	(0.00034)	(0.00010)	(0.00030)	(0.00074)	(0.00057)
Vanadium	0.00223	0.00285	-0.00047	0.00723***	-0.0169***	-0.0119***
	(0.00325)	(0.003000)	(0.00075)	(0.00205)	(0.00480)	(0.00435)
Zinc	0.00123***	0.00141***	0.00019*	0.00081***	-0.00153***	-0.00127***
	(0.00035)	(0.00031)	(0.00010)	(0.00023)	(0.00044)	(0.00044)
Observations	445,897	426,011	533,908	526,221	535,510	535,510
Panel B. Models for migration during 5 years after the child's birth (k = 0)						
Cadmium	0.0732***	0.134***	0.0320***	-0.0117	-0.0412	-0.0601**
	(0.01570)	(0.01540)	(0.00477)	(0.01170)	(0.03170)	(0.02460)
Chromium	0.00192***	0.00259***	0.00015	0.00021	0.00072	-0.00016
	(0.00060)	(0.00070)	(0.00013)	(0.00037)	(0.00067)	(0.00033)
Copper	0.00245	0.0029	0.00041	-0.00248	-0.00922	-0.00606*
	(0.00177)	(0.00211)	(0.00068)	(0.00175)	(0.00840)	(0.00354)
Nickel	0.00758***	0.0107***	0.00107**	-0.00053	-0.00249	-0.00346**
	(0.00189)	(0.00267)	(0.00051)	(0.00110)	(0.00172)	(0.00142)
Lead	0.00095***	0.00166***	0.00036***	-0.00021*	-0.00049	-0.0006*
	(0.00010)	(0.00019)	(0.00005)	(0.00013)	(0.00058)	(0.00033)
Vanadium	0.00502**	0.00608***	0.00135**	-0.00323**	-0.0119**	-0.00596**
	(0.00215)	(0.00231)	(0.00055)	(0.00126)	(0.00469)	(0.00293)
Zinc	0.00089***	0.0016***	0.0003***	-0.00012	-0.00071**	-0.00075***
	(0.00022)	(0.00022)	(0.00006)	(0.00015)	(0.00030)	(0.00029)
Observations	700,226	657,516	816,378	840,375	866,761	866,761

Table 6 (Cont.)

	(1) Mother highly educated	(2) Father highly educated	(3) Mother's (ln) income	(4) Father's (ln) income	(5) Immigrant mother	(6) Teenage mother
Panel C. Models for migration during 5 years after the child turns 5 (k = 5)						
Cadmium	0.0463***	0.0650***	0.0167***	-0.0195***	-0.00887	-0.0259*
	(0.01110)	(0.01450)	(0.00433)	(0.00728)	(0.02760)	(0.01410)
Chromium	0.0008	0.00152	0.00028*	-0.00071**	-0.00001	0.00096*
	(0.00087)	(0.00112)	(0.00015)	(0.00036)	(0.00075)	(0.00053)
Copper	0.00212*	0.00268	0.00115***	-0.00131	-0.00219	-0.00034
	(0.00119)	(0.00174)	(0.00035)	(0.00080)	(0.00393)	(0.00150)
Nickel	0.00539*	0.00532*	0.00018	-0.00418***	-0.00492	-0.0009
	(0.00261)	(0.00271)	(0.00073)	(0.00112)	(0.00303)	(0.00241)
Lead	0.00075***	0.00088***	0.00031***	-0.00028***	-0.00028	-0.00022
	(0.00022)	(0.00033)	(0.00006)	(0.00010)	(0.00057)	(0.00023)
Vanadium	0.00228*	0.00179	0.0012***	-0.00231***	-0.00358	0.00033
	(0.00133)	(0.00193)	(0.00045)	(0.00065)	(0.00374)	(0.00175)
Zinc	0.00018	0.00031	0.00014*	-0.0003***	-0.0006*	0.00008
	(0.00022)	(0.00023)	(0.00007)	(0.00011)	(0.00031)	(0.00024)
Observations	698,243	653,790	807,570	831,596	857,802	857,802
Panel D. Models for migration during 5 years after the child turns 10 (k = 10)						
Cadmium	0.0160	0.0437**	0.0214***	-0.0250**	-0.0282	-0.00732
	(0.0177)	(0.0215)	(0.00458)	(0.0102)	(0.0268)	(0.0291)
Chromium	0.00048	0.00205**	0.00051***	-0.00013	-0.00019	-0.00084
	(0.00056)	(0.00085)	(0.00016)	(0.00047)	(0.00043)	(0.00056)
Copper	-0.00201	-0.000004	0.00065**	-0.00061	-0.00234*	-0.00023
	(0.00173)	(0.0018)	(0.00028)	(0.00064)	(0.00135)	(0.00151)
Nickel	0.00029	0.00568**	0.00209***	-0.00319***	-0.00482***	-0.00417*
	(0.00186)	(0.00245)	(0.00051)	(0.00109)	(0.00158)	(0.00232)
Lead	0.00031*	0.00075***	0.00035***	-0.00033***	-0.00022	-0.00044
	(0.00016)	(0.00022)	(0.00005)	(0.00011)	(0.00025)	(0.0003)
Vanadium	-0.00124	-0.00093	0.00038	-0.00087*	-0.0016	0.00330**
	(0.00094)	(0.00122)	(0.0003)	(0.00045)	(0.00179)	(0.00164)
Zinc	-0.00029	-0.00001	0.0002***	-0.00034***	-0.00065***	-0.00002
	(0.00024)	(0.00027)	(0.00005)	(0.0001)	(0.00019)	(0.00026)
Observations	690,699	653,496	801,331	830,316	851,216	851,216

Notes: The table presents β_3 coefficients from Equation 3. The significant estimates with unexpected sign are highlighted in grey. All regressions control for age (except for column (6)), municipality characteristics, and municipality and year fixed effects. Regressions in Panel A exclude cohorts born during 1973-1975 due to lack of data on municipality pollution levels for the years 1968-1970. Standard errors (in parentheses) are clustered at the municipality level. * indicates statistical significance at the 10% level, ** at the 5%, *** at the 1%.

pollution. More educated parents may be more aware of this fact, or they are more likely to connect the child's worsened health with changes in environmental quality. This finding contributes to the environmental justice literature by showing that the observed socioeconomic gradient in migratory responses to pollution may be driven by certain sub-populations (such as parents with young children).

As for the implications for the analysis of the long-run effects of accumulated metal exposure in Section 2.5.1, these results cast doubt on the causality of those estimates. Since I control for the most important parental characteristics (education, income, age, immigration status) the bias should be greatly reduced. However, there may be some unobserved characteristics that drive selective migration and at the same time affect long-run outcomes. To indirectly test whether the estimated negative effects of metal pollution are driven by migration, I include an indicator variable for whether the mother migrated at any point from five years before child's birth until the child is fifteen. According to the results (see Table 7), controlling for migration does not reduce the negative effect of metals on long-run outcomes.

Table 7: Effect of accumulated metal exposures on long-run outcomes when controlling for maternal migration

	(1) GPA	(2) Schooling	(3) Income
Cadmium	-0.0241***	-0.0954***	-0.0213***
	0.00651	0.0126	(0.00608)
Chromium	-0.0001	0.00048*	-0.00016
	0.00013	0.00029	(0.00012)
Copper	-0.0013***	-0.00334**	-0.00148***
	0.00046	0.00150	(0.00034)
Nickel	-0.000492*	-0.0007	-0.00102***
	0.00029	0.00129	(0.00037)
Lead	-0.000155**	-0.00087***	-0.00026***
	0.00007	0.00016	(0.00008)
Vanadium	0.0002	-0.00363***	-0.00077
	0.00036	0.00941	(0.00048)
Zinc	-0.000225***	-0.00097***	-0.00024***
	0.00009	0.00021	(0.00006)
Observations	552,772	808,273	777,315

Notes: Columns (1) – (3) present coefficients from 21 separate regressions. The significant estimates with unexpected sign are highlighted in grey. All regressions control for child, parental, and municipality characteristics (as in Eq. (2)), as well as municipality and year of birth fixed effects. Standard errors (in parentheses) are clustered at the municipality level. * indicates statistical significance at the 10% level, ** at the 5%, *** at the 1%.

2.6 Conclusion

This paper is the first to analyse how accumulated exposure to heavy metal pollution during childhood affects labour market related outcomes. I exploit reductions in pollution levels of different metals in Sweden that varied across municipalities and time periods and were driven by active environmental policy. Using pollution data from moss surveys combined with register data on births between 1973 and 1985, I find that accumulated exposure to metals has a negative effect on long-run outcomes. For example, a one standard deviation increase in accumulated exposure to cadmium leads to a 0.04 standard deviations drop in 9th grade GPA, a decline in education (at age 26) by around 0.13 years and a decline in earnings (at age 26) by 2.5 percent. The effect is generally larger for individuals with low educated parents but does not differ by parental income.

Another important finding of this study is that highly educated parents are more likely to out-migrate from their municipality of residence following an increase in pollution levels. Such parental sorting implies that children with highly educated parents are exposed to lower levels of pollution and thus have better long-run outcomes. Therefore, avoidance of pollution by parents can serve as a mechanism of intergenerational transmission of socioeconomic status.

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Appendix

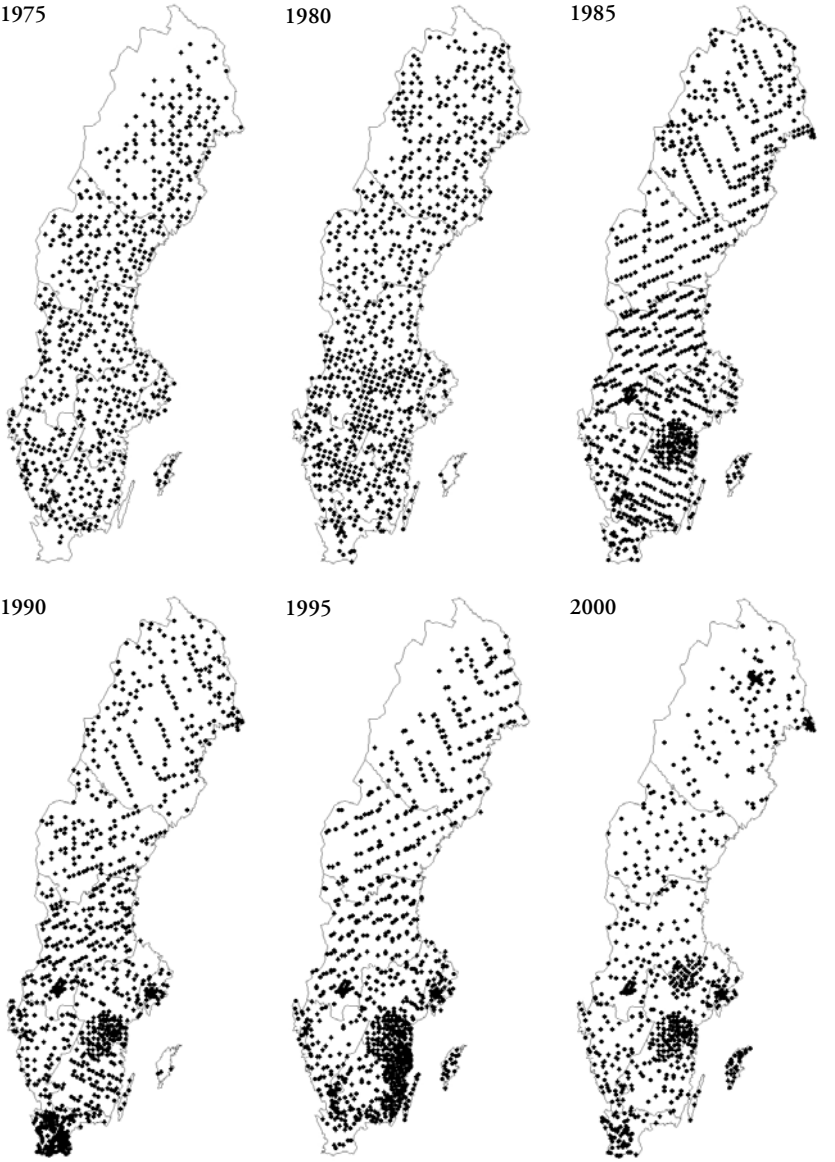

Figure A.1: Moss sampling sites in Sweden during 1975-2000

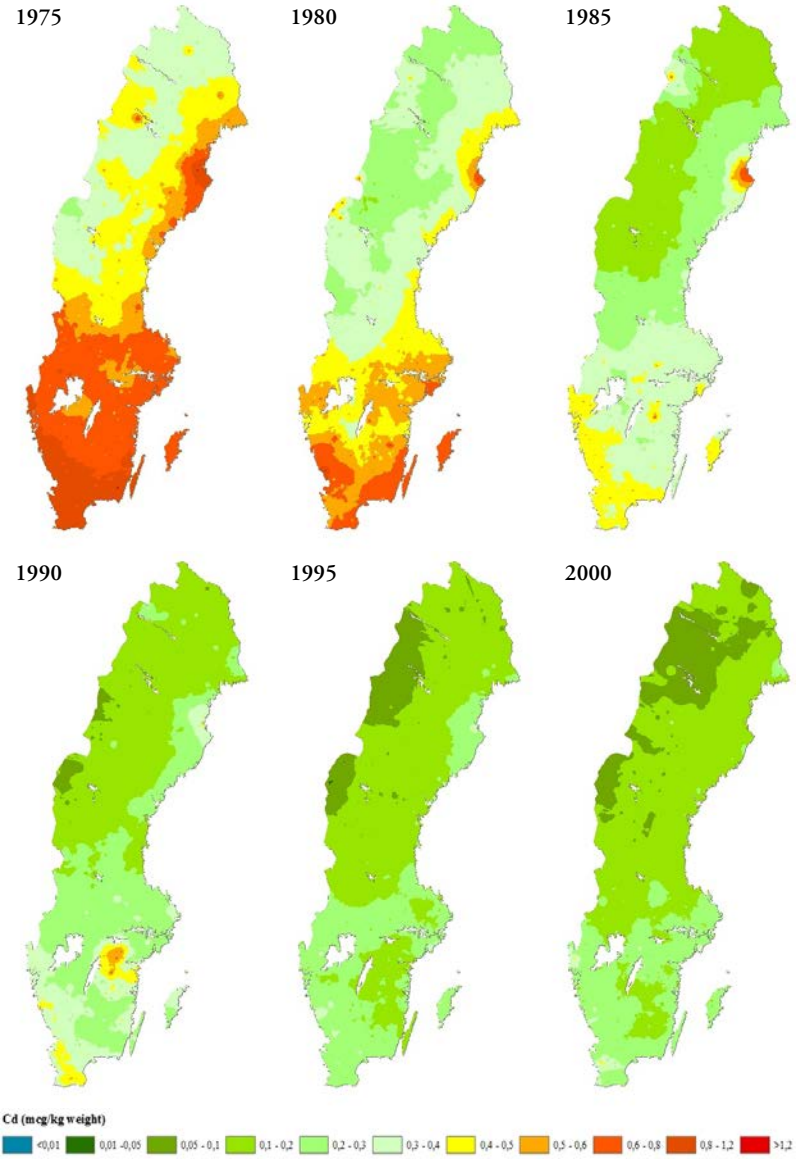

Figure A.2: Atmospheric deposition of cadmium in Sweden during 1975-2000

Source: Interpolated using the moss data (see Section 2.3 for details)

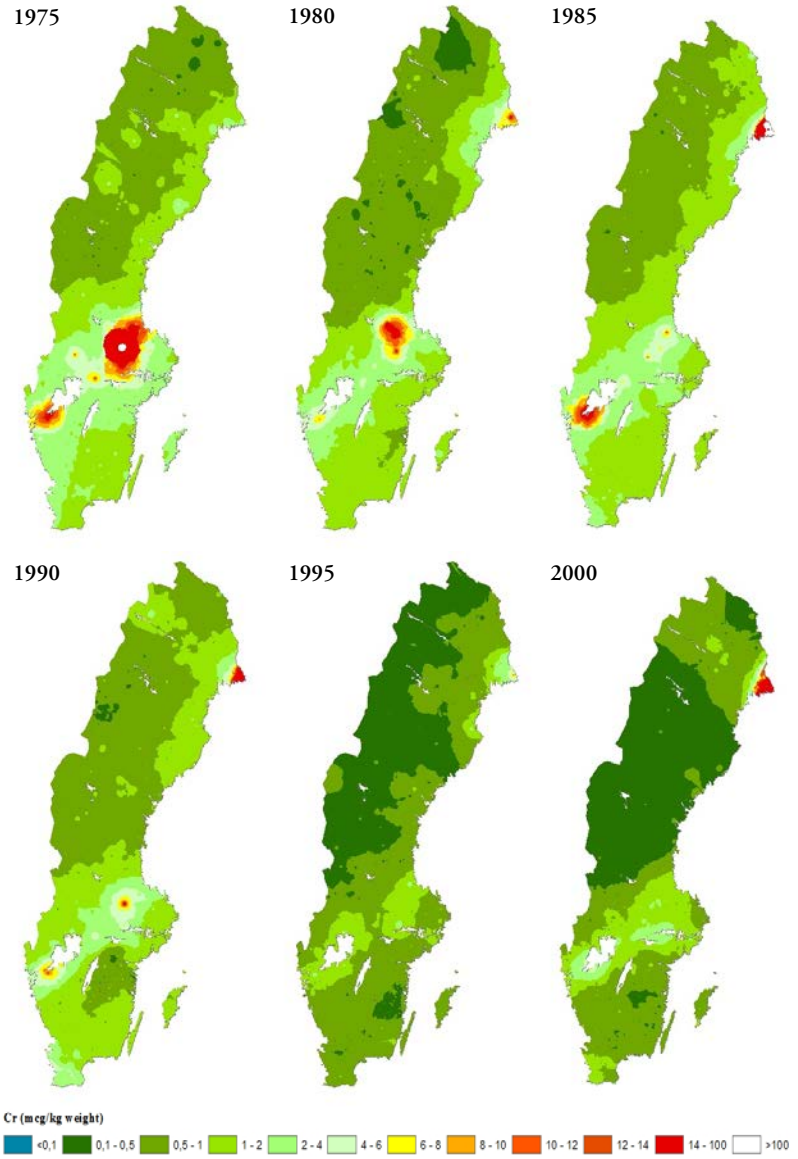

Figure A.3: Atmospheric deposition of chromium in Sweden during 1975-2000

Source: Interpolated using the moss data (see Section 2.3 for details)

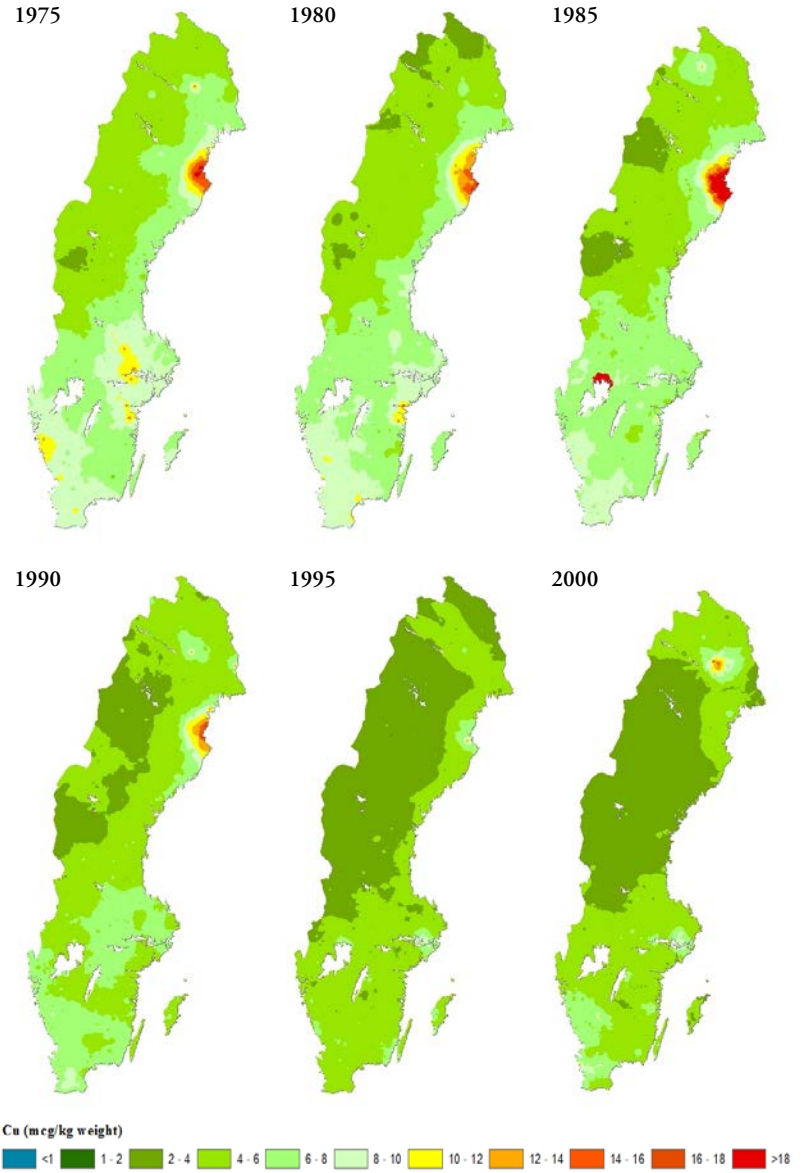

Figure A.4: Atmospheric deposition of copper in Sweden during 1975-2000

Source: Interpolated using the moss data (see Section 2.3 for details)

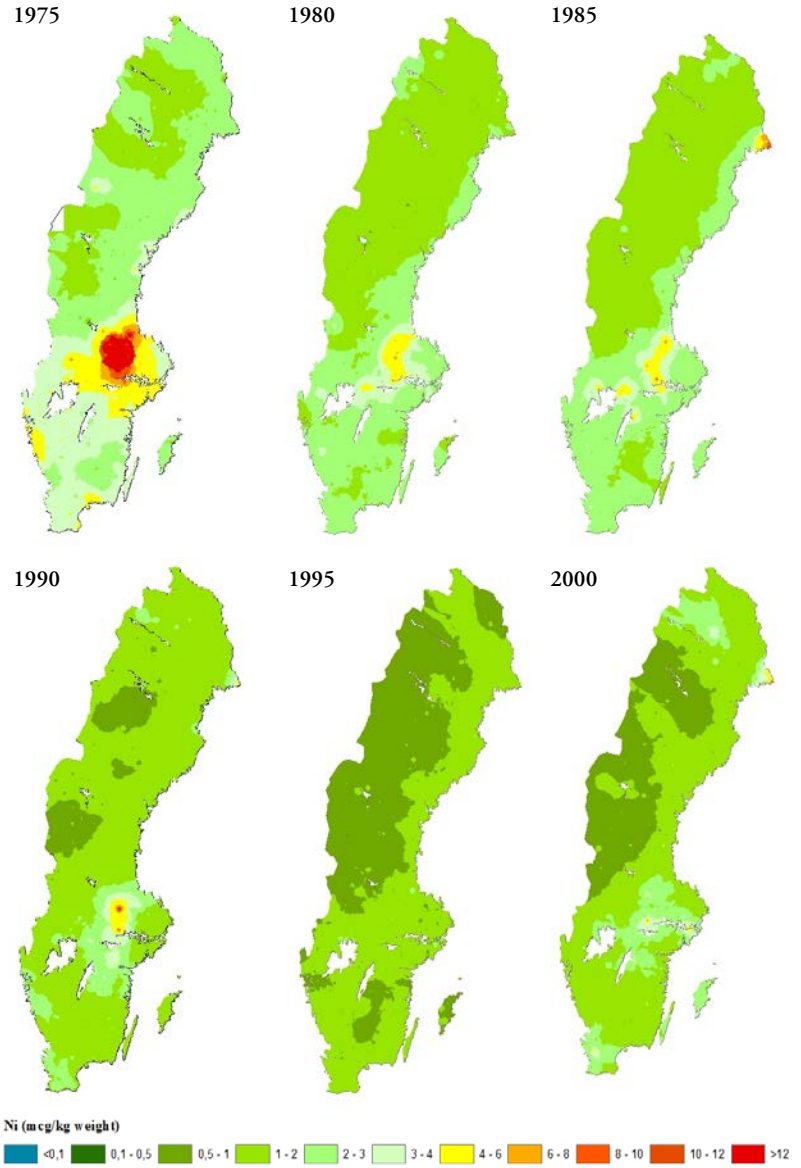

Figure A.5: Atmospheric deposition of nickel in Sweden during 1975-2000

Source: Interpolated using the moss data (see Section 2.3 for details)

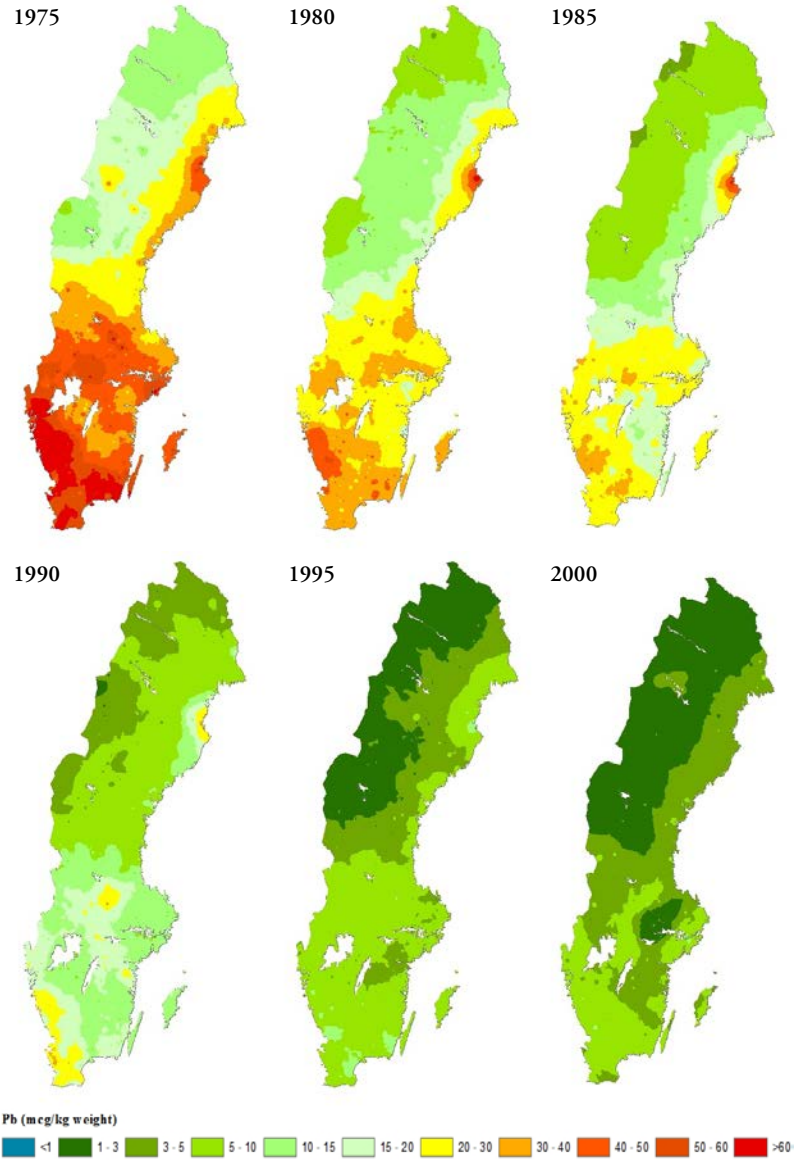

Figure A.6: Atmospheric deposition of lead in Sweden during 1975-2000

Source: Interpolated using the moss data (see Section 2.3 for details)

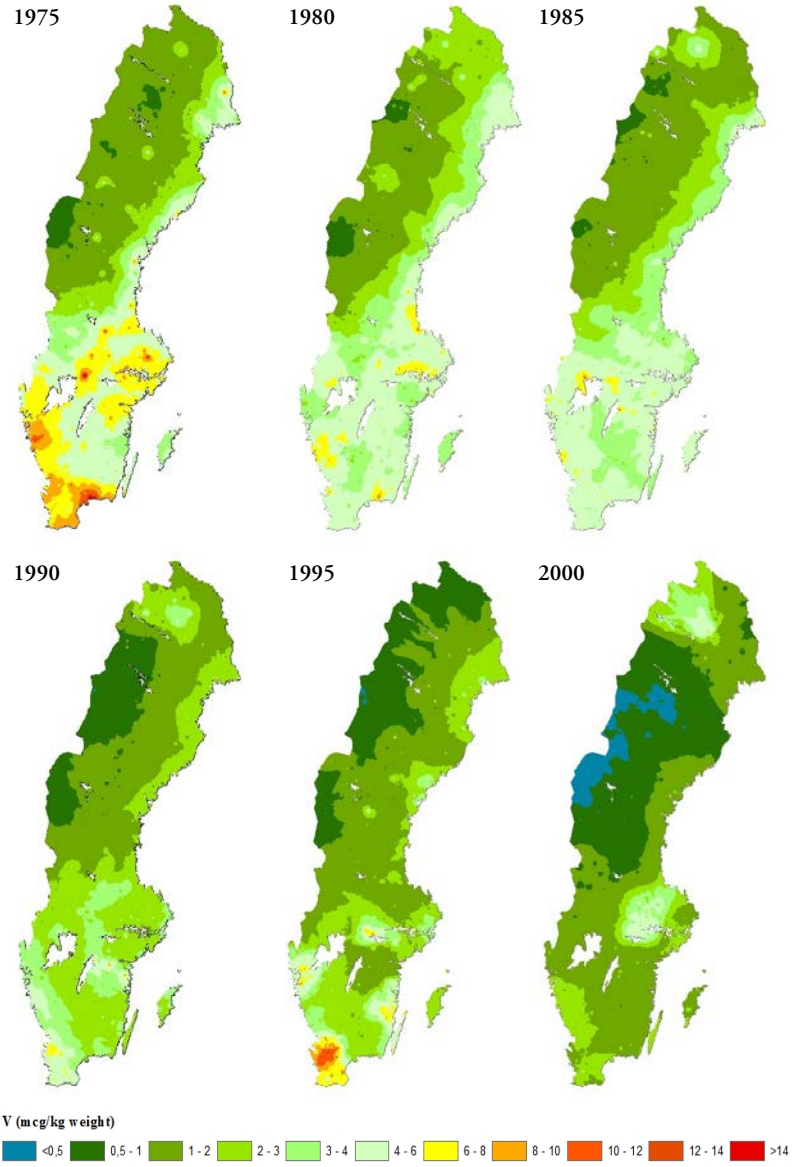

Figure A.7: Atmospheric deposition of vanadium in Sweden during 1975-2000

Source: Interpolated using the moss data (see Section 2.3 for details)

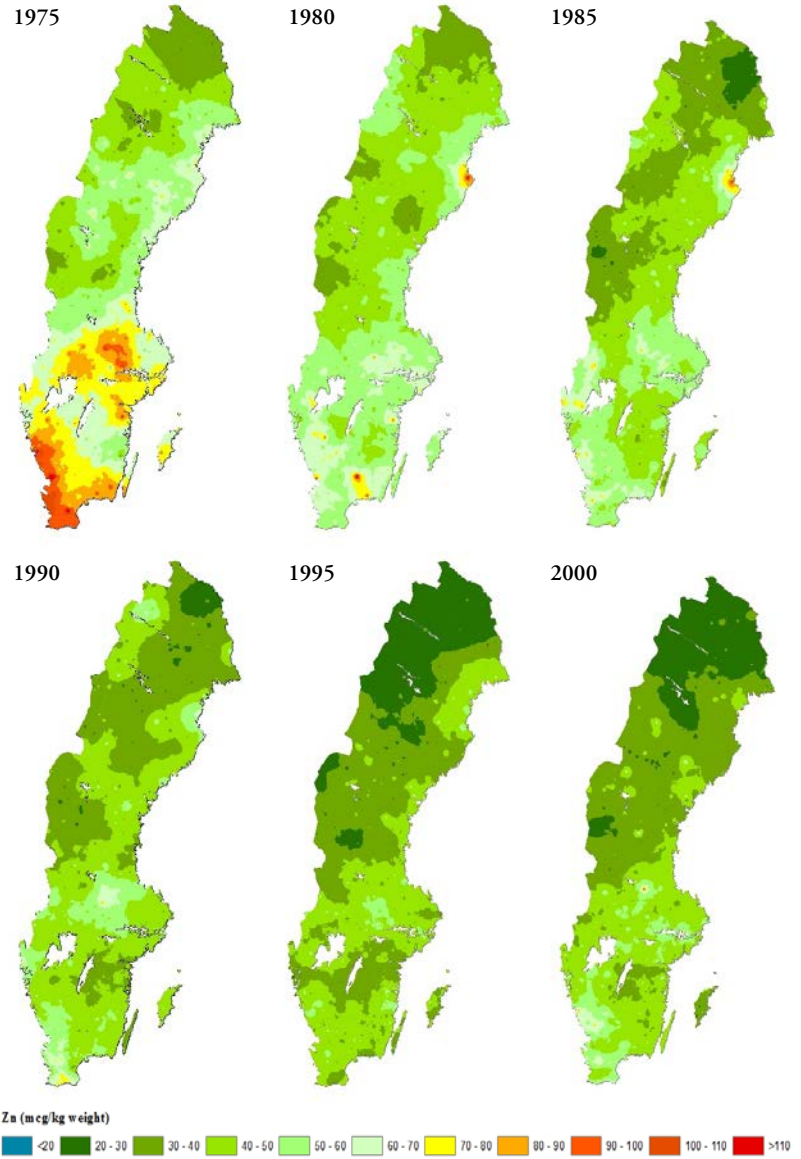

Figure A.8: Atmospheric deposition of zinc in Sweden during 1975-2000

Source: Interpolated using the moss data (see Section 2.3 for details)

Table A.1: Correlation matrix of aggregated exposures to metals

	Cd	Cu	Cr	Pb	Ni	V	Zn
Cd	1						
Cu	0.863	1					
Cr	0.299	0.312	1				
Pb	0.962	0.837	0.357	1			
Ni	0.678	0.674	0.654	0.687	1		
V	0.883	0.790	0.354	0.875	0.652	1	
Zn	0.920	0.902	0.374	0.897	0.757	0.848	1

Chapter 3
Lead Pollution and Infant Mortality in Sub-Saharan Africa: Evidence from the Phase-out of Leaded Gasoline

3.1 Introduction

Lead pollution is considered one of the greatest environmental threats to human health. According to the World Health Organization, as of 2004, lead exposure contributes to about 0.6% of the global burden of disease (World Health Organization [WHO], 2009). Recognition of the dangers of lead exposure led to the worldwide phase-out of leaded petrol as the major contributor to lead pollution.[23] However, lead poisoning still remains a major public health concern, as no level of lead is considered safe. It is still used in various consumer products (e.g. paints, pipes, storage batteries, food cans, etc.) and is also emitted into the atmosphere by industry and by incineration of lead-containing waste.

Most of today's lead pollution burden is concentrated in developing countries, where environmental regulation and control policies are weak (WHO, 2010).[24] People in developing countries may also be more vulnerable to lead toxicity than those in developed countries. Lead absorption (and therefore toxicity) increases with present calcium, iron, and zinc deficiencies, which are

[23] As of January 2015, only three countries still use leaded petrol (United Nations Environmental Programme [UNEP], 2015).

[24] The World Health Organization estimates that about 98% of adults and 99% of children affected by lead exposure live in low- and middle-income countries (WHO, 2009).

much more common in developing world (Skerfving and Bergdahl, 2007). Moreover, people in developing countries may be more susceptible to the negative effects of lead exposure due to lower initial health status, limited knowledge and possibilities to avoid pollution, and smaller opportunities to mitigate or compensate for the negative health shocks.

This paper estimates the benefits of reducing lead pollution in developing countries by analysing the effect of the phase-out of leaded gasoline on infant mortality in five Sub-Saharan African (SSA) countries: Ethiopia, Kenya, Uganda, Zimbabwe, and Burkina Faso. These countries went from using leaded to unleaded gasoline within less than four years, which provides a quasi-natural environment to help identify the causal effect of lead exposure on infant mortality. The estimation strategy relies on a difference-in-difference method comparing the changes in infant mortality rates in areas close to major roads (affected by the phase-out) to those further away from major roads (not affected).

The analysis is based on data from the Demographic Health Surveys (DHS). An important feature of these surveys is that they record geographic coordinates of the surveyed households, allowing me to calculate distances to major roads. The road network data come from the Global Roads Open Access Data Set, version 1 (gROADSv1), which combines the best available public-domain road data.

Overall, I find that the phase-out of leaded gasoline caused a large decline in infant mortality rates. Specifically, infant mortality fell by at least 7.4% in areas within 5 km of major roads during two years after the phase-out. The effect is driven by infants born to mothers with low socioeconomic status. The latter finding could be viewed as an indication that individuals in more developed countries, where education and income levels are relatively high, are less susceptible to the detrimental effects of lead pollution. As there are no studies examining the effect of a phase-out of leaded gasoline on infant mortality in developed countries, a direct comparison is, however, not possible.

One of the main methodological challenges of this research design is the possibility that the phase-out of leaded gasoline changed the composition of mothers living close to major roads, which could affect the mortality rates by itself. I show that this is not a major concern by examining how observable characteristics of mothers in the treatment and control groups change before and after the phase-out. I also conduct a number of robustness checks to further examine the validity of the identification strategy. Specifically, the results are robust to exclusion of control variables, inclusion of group-specific trends, changing the time window around the policy change, and excluding children

that could potentially be assigned treatment status incorrectly. I also perform the analysis with a fake phase-out set two years earlier, which, as expected, yields no effect on infant mortality.

The question under study is of interest for at least three reasons. First, it contributes to the on-going debate on the necessity of more stringent environmental regulations. As such regulations more often than not come with a cost (both for producers and consumers), their implementation must be justified with compelling reasons (such as deteriorating human health). Second, there is increasing evidence that health shocks during early life (in utero and during early childhood) have adverse effects on future outcomes (see Almond and Currie, 2010, for a literature overview, as well as Currie and Vogl, 2013, for a review of research for developing countries). Third, this study sheds light on the health effects of pollution in developing countries. Most of the economic literature on the adverse effects of environmental toxins focuses on developed countries (see Graff Zivin and Neidell, 2013; Currie et al., 2014, for extensive reviews). This is mainly due to data limitations and lack of exogenous variation in pollution. A few studies, however, circumvent these difficulties and establish causal links between various pollutants and child mortality in developing countries (see Section 3.2.3). The present paper is the first study for developing countries that estimates the causal effect of lead pollution on infant mortality.

The rest of the paper is structured as follows. Section 3.2 provides background on the link between lead pollution and human health and describes the process of the leaded-gasoline phase-out in SSA countries. Section 3.3 discusses the data used, describes the construction of the treatment variable, and presents the summary statistics. Section 3.4 explains the empirical approach. Section 3.5 presents the results and robustness checks. Finally, Section 3.6 concludes.

3.2 Background

3.2.1 Physiological effects of lead pollution

Lead is a naturally occurring element found in small amounts throughout the Earth's crust. While environmental contamination with lead may occur naturally, most of it results from human activities (WHO, 2010). Lead is a multimedia pollutant: it can enter the human body through several pathways (Figure A.2 shows the routes of exposure to lead from its main sources). The

main route of exposure to lead from automobile exhaust is through inhalation, but it can also enter the body by ingestion of contaminated dust, soil, water, and plants growing in contaminated soil. Once inside the body, it travels by blood to the soft tissues and organs. Eventually, most of the lead gets deposited in bones and teeth where it can stay for decades. As there is a constant skeleton turnover, some lead is released again into the bloodstream causing repeated exposure.

Toxic effects of lead may occur in any organ system of the body, but the most vulnerable are the central and peripheral nervous, haematological, renal, cardiovascular, endocrine, immune, gastrointestinal, and male reproductive systems (Skerfving and Bergdahl, 2007).[25] One of the main mechanisms for lead toxicity includes its ability to inhibit or mimic the actions of calcium and to interact with proteins (Tchounwou et al., 2012).

Foetuses are not protected from lead pollution since lead readily crosses the placenta. Moreover, during pregnancy, lead stored in the mother's skeleton is mobilized and transferred to the foetus. Exposure to lead in utero has been associated with decreased gestation, miscarriage, reduced birth weight, and impaired mental development. These effects were observed even at very low blood lead levels. After birth, infants are additionally exposed to lead through breast milk, which contains lead from recent exposure as well as lead mobilized from the mother's skeleton during lactation (Skerfving and Bergdahl, 2007). In general, young children are more susceptible to the damaging effects of lead than adults since their absorption of lead is much higher and their brains undergo rapid growth and development, which are easily disturbed. Finally, early-life lead exposure can lead to altered gene expression and, thus, an increased risk of disease later in life (WHO, 2010).

3.2.2 Related literature

Several studies have tried to estimate the causal effect of early-life lead exposure on health, cognitive development, and antisocial behaviour. For example, Clay et al. (2010) examine the effect of water-borne lead exposure on infant mortality in the US during 1900–1920. Exploiting the variation in water acidity and types of water pipes (which together determine the level of lead in tap water) across cities, the authors find that just increasing water pH from 6.675 (25th

[25] For a comprehensive account of the known toxic effects of lead, see, for example, Skerfving and Bergdahl (2007) and Agency for Toxic Substances and Disease Registry (2007).

percentile) to 7.3 (50th percentile) in cities with lead-only pipes would cause a 7% to 33% decrease in infant mortality.

Nilsson (2009) analyses the effects of the sharp phase-out of leaded gasoline in Sweden during the 1970s and 1980s on cognitive ability, scholastic performance, and labour market outcomes. He exploits the fact that the decline of lead exposure varied between the municipalities and time periods due to differences in initial concentrations. The results suggest that the reduction in lead pollution during the period under study lowered the probability of ending up in the lower end of the GPA distribution by 3.3%, increased high school completion by 0.9%, increased years of schooling by 0.05 years, and decreased the probability of welfare dependency by 0.6 percentage points. The author also finds that the effects are larger for children with low socioeconomic status.

Reyes (2007) exploits the state-level variation in lead pollution coming from the phase-out of leaded gasoline in the US in 1970s to estimate the effect of childhood lead exposure on crime rates. By linking childhood lead exposure in a specific state in a given year to crime rates in that state 20 to 30 years later, she constructs a panel data set of state–year observations covering all 50 states and the District of Columbia. The effect of lead on crime is identified from the variation in lead exposure over time within each state. The findings suggest that the drop in lead pollution as a result of the phase-out of leaded gasoline could explain as much as 56% of the drop in violent crime observed between 1992 and 2002. Grönqvist et al., (2014) use an approach similar to Nilsson's (2009) to investigate the effect of early-life lead exposure on crime in Sweden. They show that the effect of childhood lead exposure on crime is substantial even at relatively low levels of exposure.[26] The crime rates are reduced by 7% to 14% on average due to the leaded-gasoline phase-out. Again, they find that the effect is largest among children from poorer families. Further analysis reveals that the effect of lead on crime is no longer observed at the municipality average blood levels below 5 μg/dL.

Due to the absence of economic studies on the effects of lead pollution on child health in developing countries, it is useful to look at the literature on other pollutants in a developing-country context. I focus on studies that attempt to estimate causal relationships. Jayachandran (2009) investigates the effect of particulate-matter pollution on foetal, infant, and child mortality in Indonesia. She exploits the sharp variation in pollution between Indonesian subdistricts

[26] For comparison, blood lead levels of individuals in Reyes' (2007) study were estimated to be between 10 and 20 μg/dL, while Grönqvist et al. (2014) analyse individuals who already had blood lead levels below 10 μg/dL at the beginning of the study period.

resulting from massive land fires taking place in 1997. To get the information on the variation in pollution levels between locations and over time, the author uses daily satellite measurements of airborne smoke. Overall, the results show that pollution from land fires led to a 20% increase in the under-three mortality rate (if there is no effect on foetal mortality, otherwise the increase is smaller). This result is mainly driven by prenatal exposure to pollution.

Similarly, Gutierrez (2015) employs air quality data obtained from satellite images to estimate the effect of pollution on infant mortality in Mexico. To disentangle the causal effect, the author instruments pollution levels in municipalities with the dates of installation of small-scale power plants (significant sources of PM10, SOx, and NOx pollution). According to the results, the elasticity for changes in infant mortality due to respiratory diseases with respect to changes in pollution levels is between 0.58 and 0.84.

Exploiting a policy-driven variation in pollution levels (the Two Control Zone policy in China, which caused a 15% reduction in total SO_2 emissions in the country), Tanaka (2015) investigates the policy's benefits for infant health. He finds that, in the cities affected by the policy, infant mortality fell by 20% within three years, with the greatest reduction occurring for mortality during the neonatal period.

Arceo et al., (2016) use a novel design to estimate the causal effect of PM10 and CO pollution on infant mortality in Mexico City. They use thermal inversion (a meteorological phenomenon that traps pollutants in the ground-level air layer) as an instrument for air pollution. This strategy yields the effect of 0.23 weekly infant deaths per 100,000 births following an increase in 24-hour PM10 by 1 μg/m3, and 0.0046 weekly deaths per 100,000 births following an increase in 8-hour maximum for CO by 1 ppb. Comparing the results with relevant studies for the United States, the authors conclude that while their results for PM10 tend to be similar, the results for CO are larger than those derived in the US setting.

Lastly, several previous (mostly epidemiologic) studies exploited proximity to roads to analyse the relationship between traffic pollution and human health (e.g., Hoek et al., 2002; Morgenstern et al., 2006; Hart et al., 2009). Currie and Walker (2011) is closest to the present analysis in terms of method and research question. The authors estimate the effect on birth outcomes of a reduction in traffic pollution resulting from introduction of electronic toll collection (E-ZPass), using proximity to a toll plaza to define control and treatment groups. They find that infants born to mothers living within 2 kilometres of a toll plaza were 10.8% less likely to be premature and 11.8% less likely to have a low birth

weight after the introduction of E-ZPass compared to mothers living 2–10 kilometres from a toll plaza.

3.2.3 Leaded-gasoline phase-out in Sub-Saharan Africa

The process of phasing out leaded gasoline in SSA started at the Dakar Conference in June 2001. Initiated by the World Bank Clean Air Initiative in Sub-Saharan Africa, it focused on 1) raising awareness about the negative health effects of leaded petrol and 2) discussing technical and financial feasibility of the phase-out process.[27] In the resulting Dakar Declaration, the 25 participating SSA countries agreed to phase-out leaded fuel by the end of 2005 (Todd and Todd, 2010). As of 1 January 2006, SSA was completely lead-free (see Figure A.1).

Figure 1 shows the timelines for phasing out of leaded petrol in the five countries under study. Ethiopia was the first to transition. As all petrol was imported, the conversion was performed by simply switching to unleaded imports as of January 2004. Three other oil importing countries (Burkina Faso, Uganda, and Zimbabwe), being landlocked, could not easily switch suppliers due to high transport costs. They transitioned at the same time with their main suppliers (Ivory Coast, Kenya, and South Africa and Mozambique respectively) (Todd and Todd, 2010; UNEP, 2010). Kenya started importing unleaded petrol in 2002. However, since both grades of gasoline were retailed at the same price, the market share of unleaded gasoline was very low (e.g., in July 2004, it was estimated to be about 5%; Mungatana, 2004).[28] By the end of 2005 Kenya upgraded its own refineries to produce unleaded petrol, and thus the transition was complete.

The effect of the SSA leaded-gasoline phase-out on blood lead levels was studied only in Ghana. According to the study conducted by the Ghana Environmental Protection Agency, the mean blood lead level dropped from 26.4

[27] There was a common misconception regarding the need to add lead to gasoline. Lead additives had been used since 1930s to enhance gasoline's octane rating. However, new technologies were available to easily and cost-effectively enhance octane rating (see Lovei, 1998, for more technical details). Another reason for adding lead was that it provided lubrication for the engine valve seats. However, several tests carried out in the United States and Europe had shown that unleaded gasoline does not cause recession of soft valve seats under normal driving conditions (Weaver, 1986). In addition, alternative gasoline additives existed to lubricate the engine (Lovei, 1998).

[28] The consumers preferred leaded gasoline due to a widespread belief that it is required for older cars.

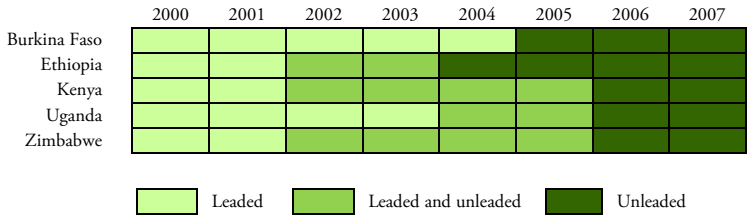

Figure 1: Timelines for eliminating leaded gasoline in five SSA countries

Source: Dumitrescu (2006).

µg/dl in 2003 (before the phase-out) to 5.33 µg/dl in 2006 (Todd and Todd, 2010).

3.3 Data

In this analysis, I consider five SSA countries: Ethiopia, Kenya, Uganda, Zimbabwe, and Burkina Faso. The reason for focusing on these countries is that both the DHS survey data (including GPS coordinates) and good quality data on road networks are available for them. These data sources are described below, followed by an explanation of how the control and treatment groups were constructed and the summary statistics.

3.3.1 Sources

The individual-level data used in this study come from the DHS data sets. DHS are nationally representative household surveys developed by the United States Agency for International Development (USAID). Their main aim is to provide reliable and internationally comparable data on health and fertility in developing countries. In each country, the surveys are conducted at intervals of about five years. As part of the surveys, female respondents are asked questions regarding all their children born alive (their birthdays, whether still alive, and if not, the age of death). I use this information to construct the outcome variable (an indicator for whether the child died within the first year of life) and the birth order of the child. In addition, the surveys inquire about mother's age (from which the age at birth is calculated) and years of education. The DHS data also

contain a variable reflecting the living standard of the households (the so-called *wealth index*), which is used as a control.[29]

Importantly for this analysis, the DHS surveys also contain the GPS coordinates of each household's sample cluster. The GPS location is collected at the centre of clusters (which are usually census enumeration areas). Location information is collected at the cluster rather than household level to protect respondents' confidentiality. For the same reason, the clusters in the data sets are randomly displaced (urban clusters up to 2 km and rural clusters up to 5 km, with 1% of the rural clusters being displaced a distance up to 10 km) (DHS Spatial Interpolation Working Group, 2004).

Figure 2 shows which surveys are used in the main analysis.[30] The red lines on the figure separate the cohorts born before and after the phase-out of leaded gasoline, and the black boxes show the years of birth that I focus on (two years before and two years after the phase-out). It is important to realize that the location of a household collected during the survey does not necessarily coincide with the place of residence during birth as the mother may have moved. If the movement of mothers is random between control and treatment areas, there will be a classical measurement error resulting in downward-biased estimates. In case the migration patterns are not random, the direction of the bias is ambiguous and depends on several factors. I discuss possible biases arising under different scenarios in the Appendix. In Section 3.5.3, I show, however, that the bias associated with mothers' migration is not an issue in this analysis.

Finally, the road network data come from gROADSv1 (Center for International Earth Science Information Network [CEISIN] and Information Technology Outreach Services, 2013). The data set was developed by the Global Roads Data Development Task Group of the International Council of Science (ISCU). It integrates best-available public-domain road data (see CEISIN, 2013 for details about construction of the data set). The periods that the data represent range from the 1980s to 2010, depending on the country. This potentially creates a measurement error since the earlier data may exclude some newly build roads while the later data may contain roads that had not yet existed during the period of analysis. Provided the construction of roads is exogenous to

[29] The wealth index is constructed based on the information about the household's possessions (e.g., television, car, and livestock), types of water and fuels used, access to sanitation facilities, etc. (see Rutstein and Johnson, 2004, for a detailed description).

[30] Earlier surveys are also used for plotting group-specific trends in Section 3.3.2 and robustness checks in Section 3.5.3.

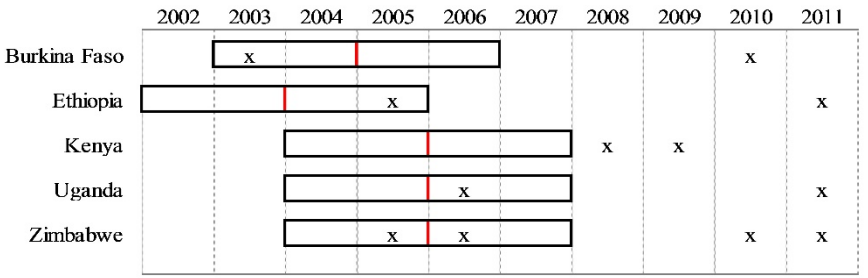

Figure 2: Years of conducting the DHS interviews relative to the timing of the phase-out

Notes: Xs mark the years of the surveys. Red lines separate cohorts born before and after the phase-out of leaded gasoline. Black boxes indicate the years of birth used in the main analysis.

changes in infant mortality, however, the estimates in the main analysis will be biased downwards.

3.3.2 Construction of the treatment variable

Lacking data on actual exposure to lead pollution, I construct the treatment variable based on proximity of mothers' place of residence to major roads. These include highways, primary, and secondary roads.[31] A mother is assigned to the treatment group if she lives within a certain distance of the nearest major road, and to the control group otherwise. The choice of the cut-off distance is, however, not obvious. It has been shown in the literature that concentrations of lead in ambient air halve within about 50 meters from a road, but then decline very slowly and uniformly (Daines et al., 1970). As most of the vehicular lead particles are of very small size (<1 μm aerodynamic diameter), they can travel over thousands of kilometres before being deposited (Harrison and Laxen, 1984). This implies that whatever distance is chosen as a cut-off, the control group will still be affected by the phase-out to some extent.

[31] Tertiary and urban roads, as well as trails are excluded from the analysis. While tertiary roads and trails typically carry comparatively low levels of traffic, this does not necessarily hold for urban roads (roads located within boundaries of cities or towns apart from the major roads). Since the data I use do not contain urban roads, they cannot be incorporated into the analysis. However, since major roads connect in urban areas, most of the individuals residing close to city centres (where pollution on the urban roads is more likely to be high) are assigned to the treatment group anyway.

One natural choice for the cut-off distance would be 50 meters from a major road due to extremely high pollution levels in the immediate proximity from a road. However, this would lead to a large measurement error when assigning control/treatment status due to a random displacement of the GPS coordinates in the data (see Section 3.3.1). Moreover, only a small number of people live that close to major roads (1% in my sample), while the aim of this study is to estimate the effect of lead pollution on the population in general. Therefore, I try several different cut-offs of 1, 2, 3, 4, 5, 10, and 15 km.

The process of creating the treatment and control groups is illustrated in Figure 3. Household clusters falling within the yellow buffers (representing areas within 5, 10, and 15 km from a major road) constitute the treatment groups. Households located outside the respective buffers are assigned to the control groups.

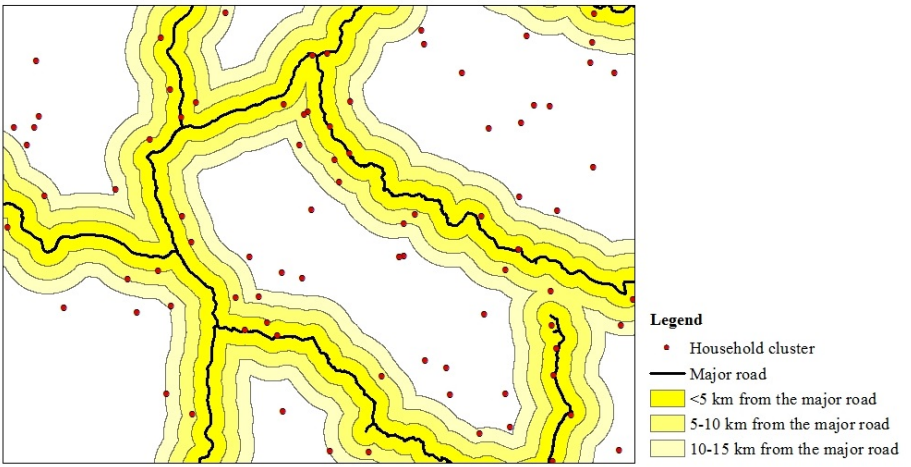

Figure 3: Illustration of the construction of the treatment and control groups

To assess whether such treatment variables are suitable for this analysis, I plot the evolution of infant mortality rates for the control and treatment groups constructed based on the 1 and 5 km cut-offs in Figure 4.[32] The plots show that the evolution of the infant mortality rate is very similar between control and treatment groups prior to the phase-out, suggesting that the control group should provide a valid counterfactual for infant mortality rates in the treatment group.

[32] The plots for the 2, 3, 4, 5, and 15 km cut-offs look very similar to the plot for the 5 km cut-off and are presented in Figure A.4.

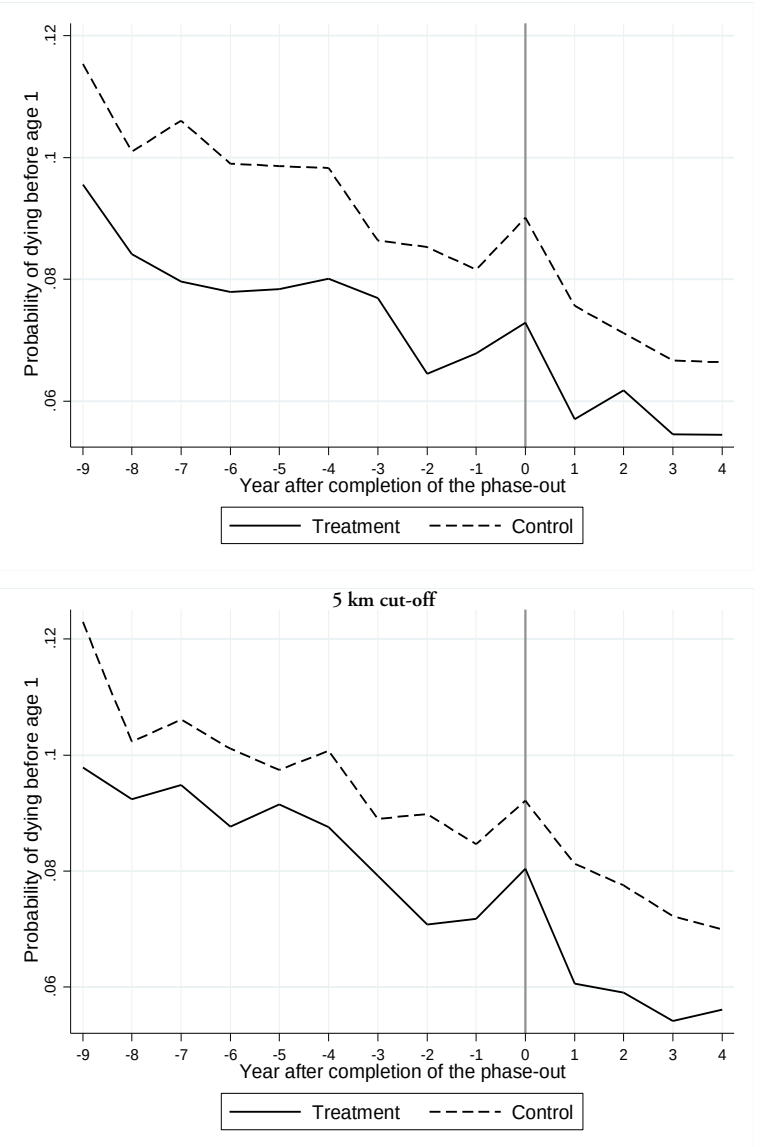

Figure 4: Trends in infant mortality rate

Notes: The figures show the average probability of dying before age 1 in the treatment and control groups before and after the phase-out of leaded gasoline. In the upper panel the treatment and control groups are defined based on a 1 km cut-off, while the lower panel shows the results when using a 5 km cut-off. The vertical grey line marks the last year when leaded gasoline was available in the country.

Figure 4 also shows that control and treatment groups defined based on the 1 km cut-off experience similar declines in infant mortality after the phase-out. However, with the cut-off at 5 km, the treatment group has a much sharper decline in infant mortality following the phase-out compared to the control group. Similar large drops in infant mortality in the treatment group occur using the 2, 3, 4, 10, and 15 km cut-offs (see Figure A.4). One possible explanation for these results is that the measurement error stemming from displacement of mother's locations is too large to identify the effect when choosing a cut-off of 1 km from a road. Another explanation, which is validated in Section 3.5.2, is that the effect of the phase-out on infant mortality does not decline gradually with distance to a road.

3.3.3 Descriptive statistics

Table 1 shows summary statistics for the outcome and explanatory variables. For space considerations, only control and treatment groups defined using the 5 km cut-off are presented.[33] The first and second columns give the means and standard deviations for the treatment group before and after the phase-out, respectively, while the third and fourth columns show corresponding statistics for the control group. The *before* period encompasses two years before the year the country was lead-free, while *after* refers to a two-year period following the year when elimination of leaded petrol was completed (see Figure 1).

As we can see, the average infant mortality rate before the phase-out is lower for the treatment group (7.6%) compared to the control group (8.8%). After the phase-out, the gap increases as the mean infant mortality declines by 1.6 percentage points for the treatment group and only by 0.8 percentage points for the control group. In terms of mothers' characteristics, around half of mothers in the treatment group have at least one year of education, while only a third of mothers in the control group do. Mothers in the treatment group are also wealthier on average than those in the control group. These differences between the control and treatment groups could reflect the fact that areas closer to roads are more densely populated with better economic conditions and access to health care. However, the means of the explanatory variables before and after the phase-out are very similar. This is reassuring as it indicates that the treatment is exogenous to observable characteristics. I explore this issue in more detail in Section 3.5.3.

[33] Summary statistics for the control and treatment groups defined based on 1, 2, 3, 4, 10, and 15 km cut-offs are presented in Tables A.1-A.3.

Table 1: Summary statistics for treatment and control groups (5 km cut-off) before and after the phase-out

	<5 km from major road		>5 km from major road	
	Before	After	Before	After
Infant mortality rate	0.076	0.06	0.088	0.08
	(0.265)	(0.237)	(0.284)	(0.271)
Mother's age (years)	29.65	30.29	30.22	31.11
	(7.61)	(7.7)	(7.74)	(7.81)
Mother has at least 1 year of education	0.54	0.529	0.309	0.283
	(0.498)	(0.499)	(0.462)	(0.451)
Birth order	3.56	3.57	3.99	4.02
	(2.45)	(2.46)	(2.54)	(2.56)
Multiple birth	0.03	0.029	0.029	0.03
	(0.171)	(0.169)	(0.167)	(0.17)
Household wealth (quintiles)	3.415	3.427	2.337	2.33
	(1.45)	(1.455)	(1.255)	(1.275)
Observations	15,773	12,645	20,957	16,293
Burkina Faso observations	2,575	1,830	5,204	3,356
Ethiopia observations	6,938	6,312	9,842	9,334
Kenya observations	1,611	1,699	6,81	6,62
Uganda observations	3,094	2,005	2,954	1,834
Zimbabwe observations	1,555	7,99	2,276	1,107

Notes: The table presents means and standard deviations (in parentheses) of the variables. The *before* period includes 2 years preceding the year when the country was lead-free, while *after* includes 2 years following the completion of the phase-out (see Figure 1). The household *wealth* variable refers to the DHS *wealth index* as described in Section 3.3.1.

3.4 Empirical strategy

To identify the effect of lead pollution on infant mortality, I employ a difference-in-difference method. Specifically, I compare mothers living close to major roads (treatment group) to mothers living further away from major roads (control group) before and after the leaded-gasoline phase-out in their respective countries. The estimated equation takes the following form:

$$Y_{ijtc} = \alpha_1 + \alpha_2 \cdot Treat_j + \alpha_3 \cdot (Treat_j \cdot After_t) + \alpha_4 \cdot X_{ijtc} + \theta_t + \varphi_c + \varepsilon_{ijt}, \quad (1)$$

where i indexes the child, j treatment status, t child's year of birth, and c country. Y is an indicator equal to 1 if the child died before age 1. *Treat* is 1 for mothers living close to major roads and 0 for mothers living further away. I estimate seven separate regressions with alternative definitions of the *Treat* variable – where the cut-off between control and treatment groups is set at 1, 2,

3, 4, 5, 10, and 15 km from a major road. *After* is 1 for the years when the country was completely lead-free, and 0 for the preceding years. I focus on births that occurred during two years before and two years after this threshold.[34] X is a set of child and mother characteristics that includes dummy variables for birth order (2nd, 3rd, 4th, 5th, 6th or higher), mother's age at birth (21–25, 26–30, 31–35, 36 or higher), household wealth (quintiles), an indicator for whether it is a multiple birth, and an indicator for whether the mother has at least one year of education. θ and φ are year and country fixed effects, respectively, and ε is the error term. Later, I also add group-specific linear trends interacted with the *After* variable as a robustness check. All standard errors are clustered at the district–treatment-group level.

The parameter of interest is α_3, which is the reduced-form effect of leaded-gasoline phase-out on infant mortality. It captures the difference in infant mortality before and after the phase-out for the affected individuals less the corresponding difference for the unaffected (or less affected) group. If reductions in lead pollution following the phase-out had a positive effect on child health, we expect α_3 to be negative. It is important to keep in mind that since I only observe children born alive, α_3 potentially underestimates the beneficial effects of the phase-out. If the phase-out increased the probability of foetus survival, and if the marginal surviving foetus is less healthy than others are, this might translate into higher infant mortality rates after the phase-out and thus a downward bias of the estimates. The estimated effect should therefore be treated as a lower bond of the true effect.

The main requirement for the parameter α_3 to have a causal interpretation is that the control group provides a valid counterfactual for changes in infant mortality in the treatment group had the phase-out not occurred. As has been shown in Figure 4, the control and treatment groups exhibit very similar trends in infant mortality rates prior to the phase-out, which is a good indicator that this requirement holds. In Section 3.5.3, I perform an additional check by explicitly controlling for group-specific linear trends in Equation 1.

Another important assumption is that there is no selection of mothers to the treatment group following the phase-out. This assumption would be violated, for example, if improvements in air quality close to major roads attracted mothers with characteristics that positively affect child's health. In such a case,

[34] I chose such a narrow time window around the phase-out to minimize the risk of confounding by other events or trends that could affect infant mortality in the treatment or the control group during a longer time span. I show in Section 3.5.3, however, that the results remain unchanged if the time window is increased to nine years before and four years after the phase-out.

the effect of lead pollution on infant mortality would be overestimated. I show in Section 3.5.3, however, that this is not a concern in this analysis.

3.5 Results

3.5.1 Baseline results

Table 2 shows the effect of leaded-gasoline phase-out on infant mortality as formalized by Equation 1. Each column presents the results from a separate regression where the treatment group is alternatively defined as residing within 1, 2, 3, 4, 5, 10, and 15 km from a major road (and the control group residing further away). We see that at the 1 km cut-off, the effect is positive (although not statistically significant), implying that the phase-out led to an increase in infant mortality. However, the point estimates at all the other cut-offs indicate a negative effect, which increases until the cut-off distance reaches 5 km and then declines. Moreover, the estimates are statistically significant only at the 5 km and 15 km cut-offs.

Table 2: Effect of leaded gasoline phase-out on infant mortality

	1 km cut-off	2 km cut-off	3 km cut-off	4 km cut-off	5 km cut-off	10 km cut-off	15 km cut-off
Treat • After	0.0030	−0.0029	−0.0021	−0.0041	−0.0051**	−0.0033	−0.0040*
	(0.0048)	(0.0034)	(0.0041)	(0.0033)	(0.0024)	(0.0021)	(0.0023)
R^2	0.021	0.021	0.021	0.021	0.021	0.021	0.021
Observations	65,668	65,668	65,668	65,668	65,668	65,668	65,668
Year fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Country fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: Dependent variable is the probability of dying before age 1. Each column presents results from a separate regression, where the treatment variable is alternatively defined based on mothers living less than 1, 2, 3, 4, 5, 10, and 15 km away from a major road. Control variables include dummies for birth order (2nd, 3rd, 4th, 5th, 6th or higher), mother's age at birth (21–25, 26–30, 31–35, 36 or higher), multiple birth, whether the mother has at least one year of education, and household wealth (quintiles). Standard errors (in parentheses) are clustered at the district–treatment-group level (for a total of 106 clusters). * indicates statistical significance at the 10% level, ** at the 5%.

The reason for such a pattern in the estimates becomes clear in the following sub-section: the phase-out of leaded gasoline affected infant mortality only for mothers residing between 2 and 5 km of major roads. For that reason, I focus only on the treatment and control groups constructed using the 5 km cut-off distance in the remainder of the analysis. The size of the effect in Table 2

implies that the phase-out led to a drop in infant mortality in areas within 5 km of major roads by around 7.4%, during the subsequent two years.

3.5.2 Dose response

As described in Section 3.3.2, lead pollution declines linearly with distance to roads (after the initial rapid decline within the first 50 meters). Therefore, one would expect the effect of the leaded-gasoline phase-out on infant mortality to be higher in areas closer to major roads. To investigate whether this is the case, I estimate Equation 1 with six treatment groups instead of one. Each treatment group includes mothers living within a certain distance range from a major road: < 1 km, 1–2 km, 2–5 km, 5–10 km, 10–15 km, and 15–30 km. The control group includes mothers residing more than 30 km from a major road.

The results, presented in Table 3, suggest that the phase-out did not affect infant mortality rate in areas within 1 km of major roads. However, there is a large and statistically significant negative effect 1–2 km from major roads, and a smaller, but still statistically significant (at the 10% level), negative effect at the 2–5 km distance. The effect disappears at the greater distances, presumably due to very low lead concentration levels in those areas. Therefore, the results show a declining dose-response pattern, but only after 1 km from major roads. They suggest that infant mortality fell by 16.6% in areas 1 to 2 km from major roads and by 8.7% in areas 2 to 5 km from major roads following the phase-out.

One potential explanation to finding no effect within the first kilometre is that parents residing in these areas may have better opportunities to protect against and mitigate the negative effects of lead pollution. Indeed, as seen in Table A.1a, mothers residing within 1 km of a major road are more likely to have some education and are on average wealthier than mothers who live farther away. As will be seen in Section 3.5.4, mother's education and wealth are important factors determining whether lead pollution affects a child's health. Another possibility is that individuals living that close to roads take more rigorous measures to protect themselves (and their children) from traffic pollution since they are more aware of the presence of highly trafficked roads in the vicinity (for example, due to traffic noise).

Table 3: Effect by distance to a major road

Distance to a major road	Treat • After	Observations in the bin
<1 km	0.0007	11,780
	(0.0033)	
1–2 km	–0.0123***	6,411
	(0.0041)	
2–5 km	–0.0061*	10,227
	(0.0033)	
5–10 km	0.0012	9,921
	(0.0043)	
10–15 km	–0.0028	6,126
	(0.0018)	
15–30 km	0.0022	9,472
	(0.0048)	
R^2	0.021	
Observations	65,668	
Year fixed effects	Yes	
Country fixed effects	Yes	
Controls	Yes	

Notes: Dependent variable is the probability of dying before age 1. Control variables include dummies for birth order (2nd, 3rd, 4th, 5th, 6th or higher), mother's age at birth (21–25, 26–30, 31–35, 36 or higher), multiple birth, whether the mother has at least one year of education, and household wealth (quintiles). Standard errors (in parentheses) are clustered at the district–treatment-group level (for a total of 106 clusters). * indicates statistical significance at the 10% level, *** at the 1%.

3.5.3 Testing the validity of the research design

This section explores the validity of the chosen research strategy. From this section forward, I focus on the control and treatment groups constructed based on the 5 km cut-off, which is motivated by the finding that the leaded-gasoline phase-out affected infant mortality only within 5 km of major roads (see the previous section).

I start by analysing whether the phase-out affected the composition of mothers living close to major roads. Detecting a change in mothers' characteristics following the phase-out would cast doubt on the unbiasedness of the estimates in Table 2. For example, if improvements in air quality close to roads attracted mothers with characteristics that are positively related to their children's health, the estimated effect of the lead pollution on infant mortality would be biased upward. To test for such selective migration, I estimate Equation 1 with different maternal characteristics as outcomes. More specifically, I look at the effect of the phase-out on the probability of being a teenage mother, the probability of her getting at least one year of education, and

on her wealth level as captured by the wealth index. As seen from Table 4, none of the estimates are statistically significantly different form zero. Although this does not rule out the possibility of maternal sorting based on unobservable characteristics, an absence of an effect on such important observables is reassuring.

Table 4 – Effect of the leaded-gasoline phase-out on mothers' characteristics

	Teenage mother	Educated mother	Mother's wealth
Treat • After	0.0011	0.0048	0.0206
	(0.0019)	(0.0043)	(0.0166)
Observations	65,668	65,668	65,668
Year fixed effects	Yes	Yes	Yes
Country fixed effects	Yes	Yes	Yes
Controls	No	No	No

Notes: The coefficients are estimates of α_3 in Equation 1. The treatment variable is defined based on the 5 km cut-off. Standard errors (in parentheses) are clustered at the district–treatment-group level (for a total of 106 clusters).

Next, I examine whether the effect is robust to different specifications and sample restrictions. The results are presented in Table 5, where Row (1) shows the estimate from the baseline regression for comparison. In Row (2), I exclude all individual and maternal characteristics and control only for country- and year-specific effects. We can see that the point estimate is very similar to that in Row (1), further suggesting that the main findings are not driven by changes in underlying characteristics.

In Row (3), I include linear trends that are allowed to vary by treatment status and by period relative to the phase-out (i.e., before/after). The results of this specification should be treated with extreme caution, however, since the trends include only two years in each period. We see that the coefficient is no longer statistically significant. However, it still suggests a large negative effect of the phase-out on infant mortality rate and thus provides evidence that the trends in infant mortality are similar between the treatment and control groups.

Rows (4) and (5) test if the results are robust to changing the time window around the phase-out. The estimate remains unchanged when the time window is increased to nine years before and four years after the phase-out; the effect rises somewhat if the sample is restricted to the births that occurred one year before and one year after the phase-out. Both estimates are statistically significant.[35]

[35] I also ran a regression that included the group-specific linear trends in the model with the increased time window. This should give a more reliable test of similarity in trends between control and treatment groups compared to Row (3). The resulting coefficient (0.0074) is a

Table 5: Robustness checks

Specification	Coefficient	Observations
(1) Baseline specification	–0.0051**	65,668
	(0.0024)	
(2) Exclude controls	–0.0059***	65,668
	(0.0019)	
(3) Include group x period trends	–0.0095	65,668
	(0.0093)	
(4) Increase time window	–0.0056***	249,134
	(0.0010)	
(5) Decrease time window	–0.0083**	33,780
	(0.0034)	
(6) Ethiopia, Kenya (restricted)	–0.0010	16,882
	(0.0047)	
(7) Ethiopia, Kenya (not restricted)	–0.0011	18,085
	(0.0062)	
(8) Placebo test	–.0001	76,325
	(0.0027)	
Year fixed effects	Yes	
Country fixed effects	Yes	

Notes: The coefficients are estimates of α_3 in Equation 1. The treatment variable is defined based on the 5 km cut-off. Standard errors (in parentheses) are clustered at the district-treatment group level (for a total of 106 clusters). ** indicates statistical significance at the 5% level, *** at the 1%.

As mentioned in Section 3.3.1, the results in this study may be biased by my use of the mother's residential location at the time of the survey as a proxy for the child's place of birth. To examine whether there is a bias, I use the question available in some of the surveys asking mothers how many years they have lived in the current place of residence. Only two of the surveys (for Ethiopia in 2005 and for Kenya in 2008–2009) have this information. Using these two surveys, I exclude births that occurred before the mother migrated to the current place of residence (if she did), which further reduces the sample size by 6.6%. This way, I focus only on children for whom the place of birth is known and who are, therefore, assigned the treatment status correctly. The regression results are presented in Row (6) of Table 5; Row (7) shows the estimated effect when using the full sample of the two surveys for comparison purposes. As can be seen, the estimates are very imprecisely estimated as the sample size is reduced considerably compared to the baseline specification. The effect is also much smaller in size. However, when comparing the estimates between Rows (6) and

little larger compared to the estimate in Row (4) and is statistically significant at the 1% level, which is reassuring.

(7), we see that they are almost identical. The results, therefore, provide no evidence that measurement error is a serious concern.

As the final check of the estimation strategy, I run placebo regressions in which the phase-out is moved two years back in time. As in the baseline specification, I focus on cohorts born two years before and two years after this "phase-out." As expected, there is no effect on infant mortality. The results in Row (8) of Table 5 show that the point estimate is very close to zero and is not statistically significant.

3.5.4 Heterogeneity

This subsection explores whether some population groups are more vulnerable to lead pollution than others. First, I investigate whether there are differences in the effects by gender. Such differences could arise due to physiological differences between genders, but also as a result of differential parental behaviour. For example, if parents are more likely to protect a son from ambient pollution or to seek medical care for his health problems, then the beneficial health effects of the phase-out would be stronger for girls. Rows (1) and (2) of Table 6 show the estimates of the effect separately for boys and girls. The results suggest that the leaded-gasoline phase-out significantly reduced infant mortality among girls, but had no effect on boys. This aligns with several previous studies on air pollution and child mortality in the developing world reporting stronger negative effects for girls (Jayachandran, 2009; Tanaka, 2015).

Next, I analyse how the estimated effects differ depending on the mother's socioeconomic status. There are several channels through which SES can influence the intensity of the harmful effects of pollution. First, children of lower SES may be exposed to more pollution to begin with; for example, poorer mothers may spend more time outdoors due to the nature of their work or location of the household (e.g., in rural areas). Second, the same level of pollution may have a stronger negative effect on poor children (or children with uneducated parents): for example, they may have lower health in general and be, thus, more sensitive to pollution. Third, higher-income or more educated parents may have more resources and knowledge to avoid pollution, such as by moving away or staying inside during times when ambient pollution levels are high (see Neidell, 2004). Finally, wealthier and more educated parents may be more successful in mitigating negative effects of pollution by seeking medical care or making other investments in the child's health.

Table 6: Heterogeneity in the effect

	Specification	Coefficient	Observations
(1)	Boys	0.0009	33,314
		(0.0025)	
(2)	Girls	–0.0107**	32,354
		(0.0045)	
(3)	Mother has education	–0.0004	26,299
		(0.0022)	
(4)	Mother has no education	–0.0123**	39,369
		(0.0054)	
(5)	High *wealth* household	0.0028	35,402
		(0.0038)	
(6)	Low *wealth* household	–0.0091***	30,266
		(0.0017)	
Year fixed effects		Yes	
Country fixed effects		Yes	
Controls		Yes	

Notes: The coefficients are estimates of α_3 in Equation 1. The treatment variable is defined based on the 5 km cut-off. Control variables include dummies for birth order (2nd, 3rd, 4th, 5th, 6th or higher), mother's age at birth (21–25, 26–30, 31–35, 36 or higher), multiple birth, whether mother has at least one year of education, and household *wealth* (quintiles). Standard errors (in parentheses) are clustered at the district-treatment group level (for a total of 106 clusters). ** indicates statistical significance at the 5% level, *** at the 1% level.

To analyse the differences in the effects between socioeconomic groups, I estimate Equation 1 separately for mothers with at least one year of education and mothers with no education, and separately for mothers whose household wealth is relatively high (within the third quintile or higher) and those with low wealth (first two quintiles). Rows (3) through (6) present the results. The effect is much stronger for mothers with no education compared to the estimate for the whole sample (see Table 2). The effect for educated mothers, on the other hand, is very small and statistically insignificant. Similarly, the effect increases (compared to the estimate in Table 2) when looking only at infants born to mothers with low wealth and disappears when focusing on infants from high-wealth households. These results suggest that only children from poorer and low-educated families were negatively affected by traffic-related lead pollution in SSA and, therefore, benefited from the phase-out of leaded gasoline.

3.6 Conclusion

Previous research on the effects of environmental pollution on child health has predominantly focused on developed countries. It is in developing countries, however, where the majority of modern pollution occurs. Moreover, the health effects of pollution are likely to be much larger in the developing world due to presence of other health shocks and limited possibilities to avoid pollution or mitigate its negative effects. The shortage of studies for developing countries is largely driven by the lack of data on pollution concentrations, with lead pollution being no exception.

This paper is the first to study the effect of lead pollution on infant mortality in developing countries. It does so by evaluating an environmental regulation that completely eliminated the major source of lead pollution – leaded gasoline. The five SSA countries under study went from using leaded to unleaded gasoline within less than four years, which helps identify the causal effect of lead exposure on infant mortality. To estimate the effect, I employ a difference-in-difference method comparing the changes in infant mortality rates in areas close to major roads (affected by the leaded-gasoline phase-out) to those further away from major roads (unaffected). The findings suggest that the phase-out of leaded gasoline in these countries reduced infant mortality in areas within 5 km of major roads by at least 7.4% during the subsequent two years. The effect is driven by infants born to mothers with low socioeconomic status.

The results of this paper provide a good example of how a relatively simple and inexpensive environmental protection measure can lead to tremendous improvements in population health in developing countries. This change would not have occurred, however, without our prior knowledge of the detrimental health effects of lead (which was the reason for the phase-out). This is why further research on the effects of pollution on health and well-being is crucial. Humans are exposed to a large and increasing number of chemicals, the health effects of which are often poorly understood.[36] An enormous research effort is required to fill these knowledge gaps.

[36] For example, more than 85,000 chemicals are listed under the US EPA Toxic Substances Control Act (which excludes pesticides, food additives, drugs, cosmetics, and nuclear materials).

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Appendix

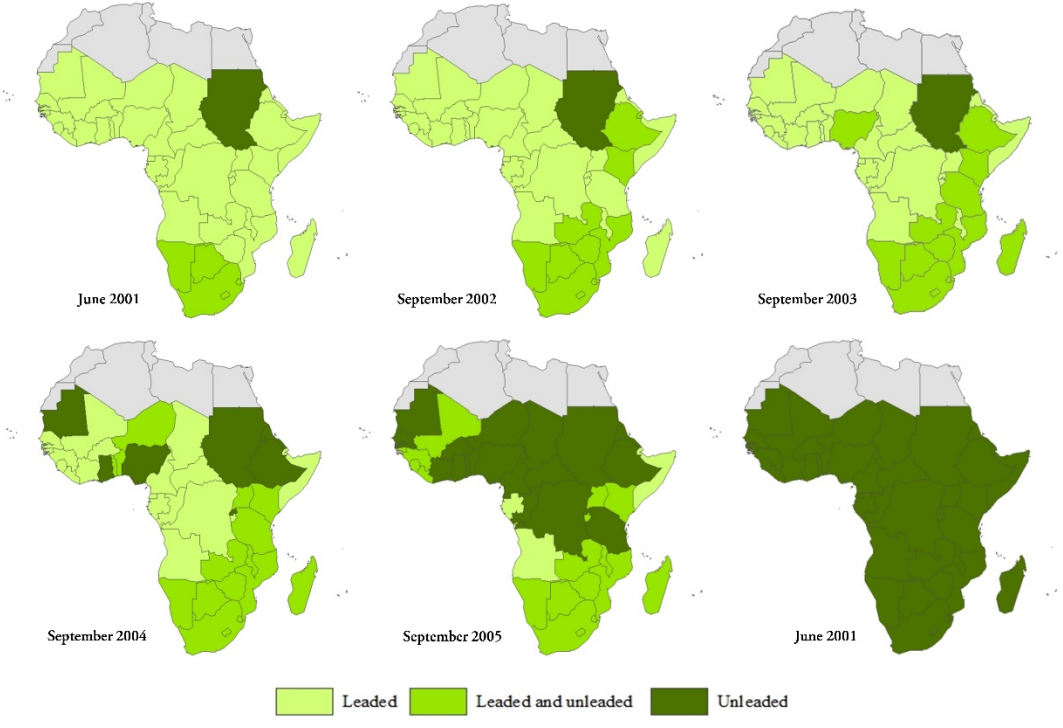


Figure A.1: Progress of leaded-gasoline phase-out in Sub-Saharan Africa

Source: Dumitrescu (2006).

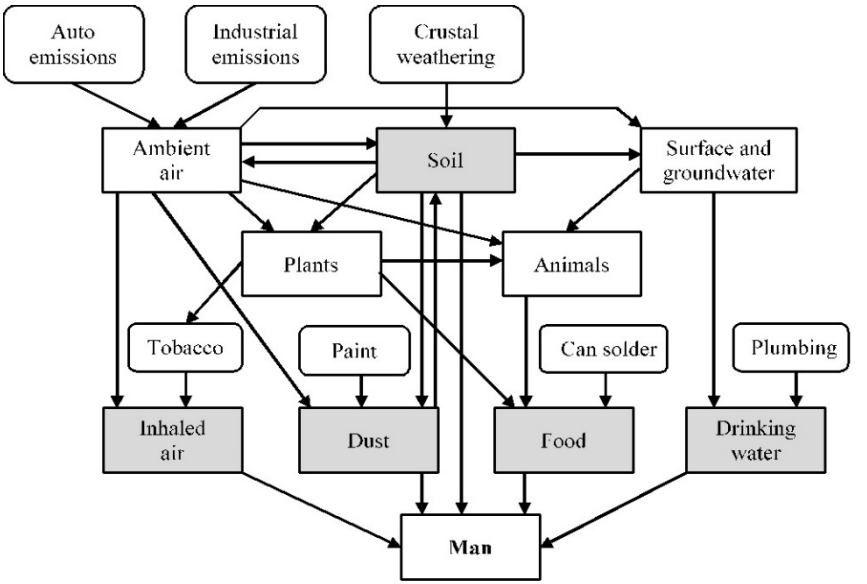


Figure A.2: Main sources and routes of lead exposure

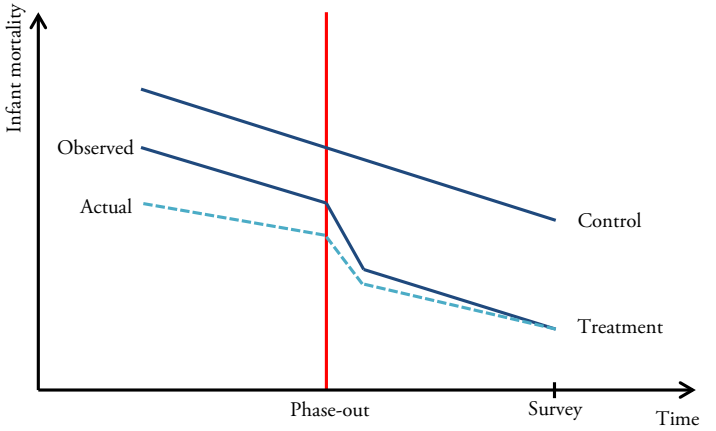
Source: Skerfving & Bergdahl (2007).

Analysing the direction of the bias associated with mothers' migration

As explained in Section 3.1, the information on the place of residence that is collected in the surveys corresponds to mother's current location and does not necessarily represent child's place of birth due to possible migration of the mother. This section discusses how non-random maternal migration between control and treatment groups may affect the estimated effect of the leaded-gasoline phase-out on infant mortality. I focus on the more likely scenario when mothers tend to move to areas closer to roads as these areas are likely to be more urbanized and thus offer better prospects (e.g., better access to education, health care, jobs, etc.) compared to more remote areas. In this case, children are born far from roads (control areas) but observed close to roads (treatment areas) and are thus wrongly assigned to the treatment group. Since the control group on average has a higher infant mortality rate (see Section 3.3.3), the observed infant mortality rate in the treatment group (which includes births that actually belong to the control group) is now higher than the actual one. As we go backwards from the survey year, it is more likely that the births are wrongly assigned and the gap between the actual and observed mortality rate is higher. The direction of the bias in the main analysis depends on where on the timeline the surveys were conducted. If there is no survey conducted in the pre-phase-out period (as in Ethiopia, Kenya, and Uganda), the actual drop in mortality due to the phase-out is smaller than observed one and we get an upward bias (see Figure A.3.a). This bias will be smaller, however, if the migrating mothers are more likely to have healthier babies than an average mother in the control group (which is possible, for example, if the migrating mothers have higher education level).

On the other hand, if there are surveys conducted right before the phase-out (as in Burkina Faso and Zimbabwe), the measurement error is larger after the phase-out rather than before, biasing the estimates downwards (see Figure A.3.b).

a – When there is no DHS survey before the phase-out.



b – When there exists a DHS survey before the phase-out.

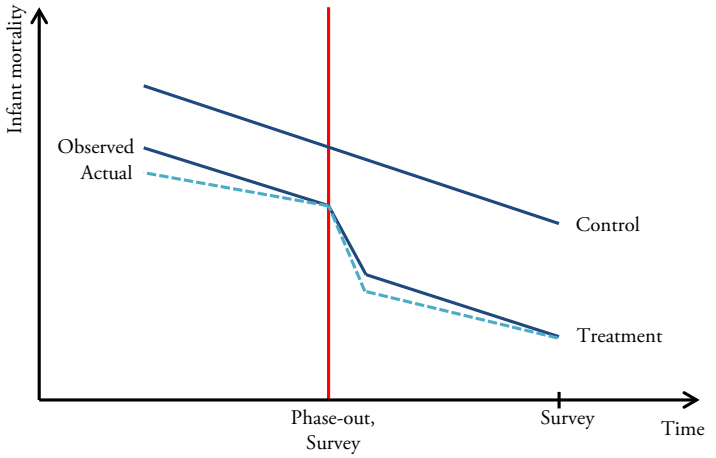


Figure A.3: Measurement error due to mothers' migration from control to treatment areas

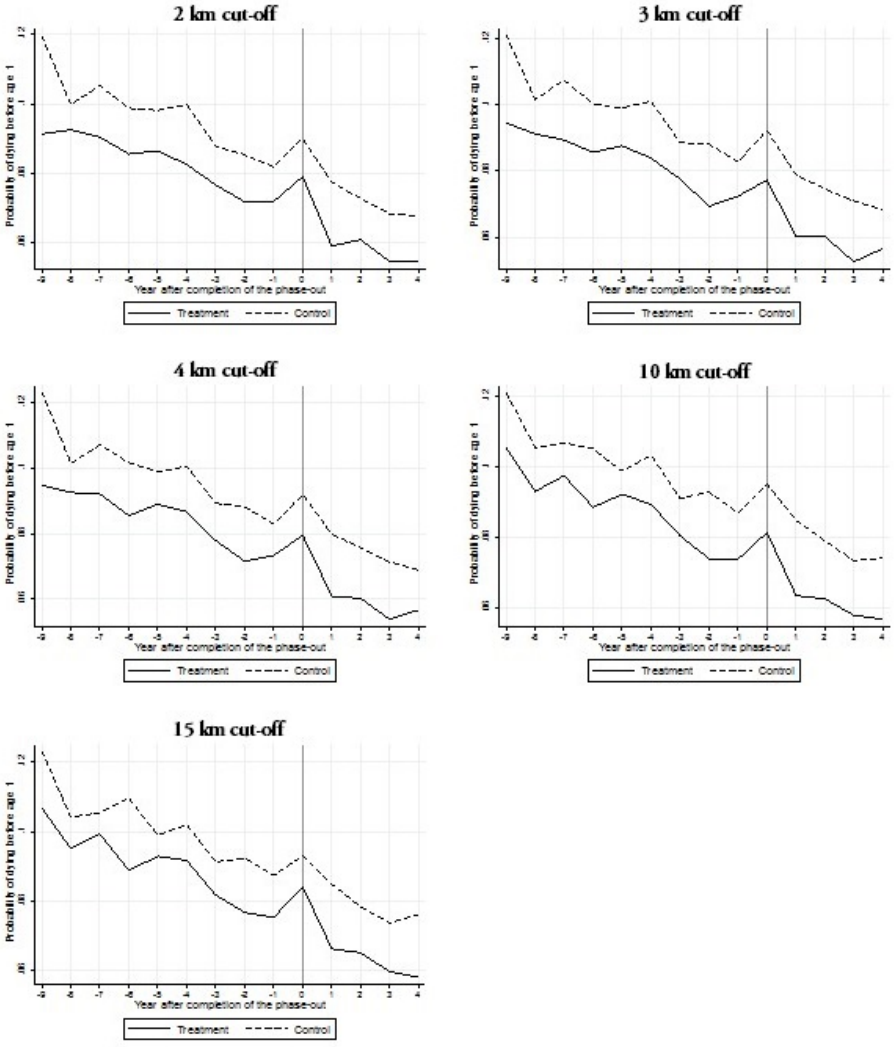


Figure A.4: Trends in infant mortality rate (2, 3, 4, 10 and 15 km cut-offs)

Table A.1: Summary statistics for treatment and control groups constructed based on 1 and 2 km cut-offs

	<1 km from major road		>1 km from major road		<2 km from major road		>2 km from major road	
	Before	After	Before	After	Before	After	Before	After
Infant mortality rate	0.070	0.059	0.086	0.074	0.075	0.060	0.086	0.075
	(0.256)	(0.236)	(0.280)	(0.261)	(0.264)	(0.237)	(0.280)	(0.264)
Mother age (years)	29.52	30.23	30.07	30.87	29.47	30.11	30.17	31.01
	(7.48)	(7.58)	(7.73)	(7.81)	(7.51)	(7.576)	(7.743)	(7.837)
Mother has at least 1 year of education	0.584	0.569	0.371	0.351	0.571	0.559	0.347	0.324
	(0.493)	(0.495)	(0.483)	(0.477)	(0.495)	(0.497)	(0.476)	(0.468)
Birth order	3.331	3.358	3.911	3.929	3.410	3.407	3.958	3.989
	(2.343)	(2.343)	(2.534)	(2.555)	(2.376)	(2.368)	(2.544)	(2.569)
Multiple birth	0.032	0.030	0.029	0.030	0.032	0.031	0.028	0.029
	(0.175)	(0.170)	(0.167)	(0.170)	(0.177)	(0.174)	(0.165)	(0.168)
Household "wealth" (quintiles)	3.804	3.796	2.585	2.588	3.707	3.714	2.461	2.452
	(1.388)	(1.398)	(1.363)	(1.382)	(1.416)	(1.420)	(1.301)	(1.316)
Observations	6,472	5,308	30,258	23,630	9,994	8,197	26,736	20,741
Burkina Faso	1,104	851	6,675	4,335	1,676	1,266	6,103	3,920
Ethiopia	2,974	2,678	13,806	12,968	4,418	4,026	12,362	11,620
Kenya	669	729	1,623	1,632	1,066	1,162	1,226	1,199
Uganda	1,184	802	4,864	3,037	1,928	1,303	4,120	2,536
Zimbabwe	541	248	3,290	1,658	906	440	2,925	1,466

Notes: The table presents means and standard deviations (in parentheses) of the variables. The before period includes two years preceding the year when the country was lead-free, while after includes two years following the completion of the phase-out (see Figure 1). The household wealth variable refers to the DHS "wealth index" as described in Section 3.3.1.

Table A.2: Summary statistics for treatment and control groups constructed based on 3 and 4 km cut-offs

	<3 km from major road		>3 km from major road		<4 km from major road		>4 km from major road	
	Before	After	Before	After	Before	After	Before	After
Infant mortality rate	0.075	0.060	0.087	0.077	0.076	0.061	0.087	0.078
	(0.263)	(0.238)	(0.282)	(0.266)	(0.266)	(0.239)	(0.282)	(0.268)
Mother age (years)	29.56	30.196	30.19	31.057	29.682	30.29	30.165	31.07
	(7.528)	(7.645)	(7.759)	(7.827)	(7.582)	(7.687)	(7.747)	(7.817)
Mother has at least 1 year of education	0.553	0.540	0.333	0.310	0.548	0.534	0.319	0.295
	(0.497)	(0.498)	(0.471)	(0.462)	(0.498)	(0.499)	(0.466)	(0.456)
Birth order	3.476	3.469	3.981	4.018	3.537	3.534	3.983	4.020
	(2.413)	(2.402)	(2.544)	(2.572)	(2.442)	(2.442)	(2.540)	(2.565)
Multiple birth	0.031	0.030	0.029	0.030	0.031	0.029	0.028	0.030
	(0.172)	(0.170)	(0.167)	(0.170)	(0.174)	(0.168)	(0.165)	(0.171)
Household "wealth" (quintiles)	3.591	3.594	2.391	2.383	3.489	3.489	2.358	2.353
	(1.427)	(1.431)	(1.273)	(1.291)	(1.441)	(1.450)	(1.261)	(1.281)
Observations	12,521	10,190	24,209	18,748	14,352	11,643	22,378	17,295
Burkina Faso	2,168	1,584	5,611	3,602	2,354	1,708	5,425	3,478
Ethiopia	5,518	5,052	11,262	10,594	6,394	5,850	10,386	9,796
Kenya	1,255	1,355	1,037	1,006	1,482	1,585	810	776
Uganda	2,391	1,611	3,657	2,228	2,692	1,767	3,356	2,072
Zimbabwe	1,189	588	2,642	1,318	1,430	733	2,401	1,173

Notes: The table presents means and standard deviations (in parentheses) of the variables. The before period includes two years preceding the year when the country was lead-free, while after includes two years following the completion of the phase-out (see Figure 1). The household wealth variable refers to the DHS "wealth index" as described in Section 3.3.1.

Table A.3: Summary statistics for treatment and control groups constructed based on 10 and 15 km cut-offs

	<10 km from major road		>10 km from major road		<15 km from major road		>15 km from major road	
	Before	After	Before	After	Before	After	Before	After
Infant mortality rate	0.077	0.063	0.091	0.082	0.08	0.066	0.09	0.082
	(0.267)	(0.243)	(0.287)	(0.275)	(0.271)	(0.248)	(0.286)	(0.274)
Mother age (years)	29.83	30.49	30.18	31.12	29.94	30.69	30.06	30.9
	(7.66)	(7.75)	(7.72)	(7.796)	(7.675)	(7.802)	(7.711)	(7.714)
Mother has at least 1 year of education	0.496	0.488	0.284	0.255	0.478	0.464	0.262	0.237
	(0.5)	(0.5)	(0.451)	(0.436)	(0.5)	(0.499)	(0.44)	(0.426)
Birth order	3.689	3.682	3.978	4.023	3.738	3.742	3.957	3.997
	(2.484)	(2.49)	(2.539)	(2.565)	(2.497)	(2.51)	(2.535)	(2.555)
Multiple birth	0.031	0.03	0.027	0.029	0.03	0.03	0.028	0.029
	(0.173)	(0.171)	(0.163)	(0.168)	(0.17)	(0.171)	(0.165)	(0.167)
Household "wealth" (quintiles)	3.195	3.22	2.244	2.237	3.091	3.106	2.188	2.189
	(1.452)	(1.458)	(1.234)	(1.26)	(1.445)	(1.456)	(1.236)	(1.264)
Observations	21,477	16,862	15,253	12,076	24,882	19,583	11,848	9,355
Burkina Faso	3,793	2,562	3,986	2,624	4,429	2,934	3,350	2,252
Ethiopia	9,562	8,616	7,218	7,030	11,236	10,304	5,544	5,342
Kenya	1,831	1,937	461	424	1,944	2,039	348	322
Uganda	4,191	2,621	1,857	1,218	4,768	3,029	1,280	810
Zimbabwe	2,100	1,126	1,731	780	2,505	1,277	1,326	629

Notes: The table presents means and standard deviations (in parentheses) of the variables. The before period includes two years preceding the year when the country was lead-free, while after includes two years following the completion of the phase-out (see Figure 1). The household wealth variable refers to the DHS "wealth index" as described in Section 3.3.1.

Chapter 4
Early-Life Lead Exposure and Cognitive Skills of School-Age Children in Uganda: Evidence from the Phase-Out of Leaded Gasoline

4.1 Introduction

It is now well established that exposure to lead in childhood has a negative effect on intellectual performance (see, e.g., Skerfving and Bergdahl, 2007, for a review of the literature). Most of the existing literature, however, focuses on concurrent effects of lead pollution, while few papers investigate the effects of early-life exposure to lead on later cognitive abilities. Since the human brain undergoes rapid growth and development in utero and during early childhood, exposure to lead during this period has the potential to cause long-lasting or permanent damage to the nervous system. In addition, even if lead neurotoxicity is temporary, its occurrence early in life may negatively affect later cognitive abilities through dynamic complementarities in skill production (Cunha and Heckman, 2007; Aizer and Cunha, 2012).

This paper estimates the causal effect of early-life lead exposure on the cognitive skills of school-age children in Uganda. I exploit a sharp reduction in lead pollution resulting from the phase-out of leaded gasoline in Uganda in late 2005. In my empirical strategy, I compare the changes in outcomes of same-aged children born (on average) two years apart and thus having a two-year difference in the length of exposure to lead pollution in early childhood. The control group consists of children living in districts with very low traffic density, which presumably experienced very low, if any, change in lead pollution

following the phase-out. I define three different treatment groups comprised of children living in districts with low, medium, and high traffic densities, which had low, medium, and large drops in lead pollution, respectively. The measure of the average traffic density in a district takes into account the density of the road network as well as the annual average daily traffic of roads in the district. These road-network data are taken from the African Infrastructure and Country Diagnostic programme. The individual level data come from the Uwezo surveys, containing information on child age, district of residence, and math and English test scores, my measures of cognitive skills.

I find a strong and statistically significant positive effect of the phase-out of leaded gasoline on test scores of children aged 6 to 9 in districts with high traffic density. The effect varies between 0.15 and 0.24 standard deviations (SD) for math scores and between 0.16 and 0.22 SD for English scores. It declines with child's age, which, in this setup, implies that lead pollution is more harmful during earlier years of life.

The main identifying assumption of this research design is that the control and treatment groups would have experienced similar changes in child test scores had the phase-out not occurred. I argue that this assumption holds since I do not find any effect of the phase-out on test scores of older children (aged 10 to 16), who experienced the decline in lead pollution at later ages and are thus less likely to be significantly affected. I also show that the phase-out of leaded gasoline is unlikely to have affected the composition of mothers in the control and treatment groups, and that the main results are robust to exclusion of control variables and using alternative measures of district traffic density.

The findings in this paper contribute to the literature on the effects of early-life health shocks on later outcomes (see Almond and Currie, 2010, and Currie and Vogl, 2013, for overviews of existing studies). It also adds to our understanding of how environmental pollution affects child health in the context of a developing country. Most of the economic literature on the adverse effects of environmental toxicants focus on developed countries (see Graff Zivin and Neidell, 2013, a Currie et al., 2014, for extensive reviews). However, several recent studies have shown that the negative health effects of pollution may be much stronger in developing countries (Tanaka, 2015; Arceo et al., 2016).[37]

[37] There are several reasons why pollution may have a much stronger negative effect on child health in developing countries compared to developed countries. First, children in developing countries are more likely to experience other shocks to their health, which may enhance the negative effects of pollution. For example, lead absorption (and therefore toxicity) increases with present deficiencies of calcium, iron, and zinc (Skerfving and Bergdahl, 2007). Second, parents in developing countries may have less opportunity or knowledge for avoiding

The present study is the first to demonstrate that this is also the case with regard to cognitive effects of lead pollution.

The rest of the paper is structured as follows. Section 4.2 provides background on the link between lead pollution and child neurological health, reviews some of the previous studies, and describes the process of the phase-out of leaded gasoline in Uganda. Section 4.3 discusses the data used and describes the process of constructing control and treatment groups. Section 4.4 explains my empirical approach. Section 4.5 presents the results and robustness checks. Finally, Section 4.6 concludes.

4.2 Background

4.2.1 Lead: exposure pathways and neurotoxicity

The vast majority of vehicular lead is emitted in particulate (inorganic) form. Most of these particles are of very small size (<1 μm aerodynamic diameter) and thus can easily enter the human body through inhalation (Harrison and Laxen, 1984). Lead particles that are deposited on the ground accumulate in soil and thus contaminate drinking water and enter the food chain, leading to exposure through ingestion (Skerfving and Bergdahl, 2007). A small part of vehicular lead emissions is in organic form. Organic lead is absorbed through the skin and is much more toxic to the brain and central nervous system than inorganic lead (World Health Organization [WHO], 2010). When lead enters the body, it travels by blood to the soft tissues and organs. After several weeks, most of the lead gets deposited in bones and teeth where it can stay for decades. As there is a constant turnover of the skeleton, some lead is released again into the bloodstream causing repeated exposure.

Foetuses are not protected from lead pollution since lead readily crosses the placenta. Moreover, during pregnancy, lead stored in the mother's skeleton is mobilized and transferred into the foetus. Infants are also exposed to lead through breast milk, which contains lead from recent exposure as well as lead mobilized from the mother's skeleton during lactation (Skerfving and Bergdahl,

pollution; they may also have lower possibilities to mitigate or compensate for the negative health shocks to their child's health.

2007). In addition, young children are at higher risk of ingesting lead particles through dust and soil due to their hand-to-mouth behaviour.[38]

Lead has long been recognized as a neurotoxin that, at high levels of exposure, attacks the central nervous system causing coma, convulsions, and death. However, recent research has also shown that lead causes damage to the nervous system even at low levels of exposure, resulting in, for example, loss of cognition, reduced attention span, and changes in behaviour (Emory et. al, 2003; Chiodo et al., 2004; Surkan et al., 2007). Young children are more susceptible to the damaging effects of lead than adults since their absorption of lead is much higher and their brains undergo rapid growth and development which are easily disturbed (WHO, 2010).

One of the main mechanisms through which lead affects the nervous system is by mimicking or inhibiting the action of calcium, an important cofactor in many biological processes. One example of such action by lead is premature activation of protein kinase C, which may lead to impaired microvascular formation and function in the brain and disruption of the blood–brain barrier. This may partially explain why infants and young children (whose blood–brain barrier is not fully developed) are particularly vulnerable to lead's neurotoxicity. Other important channels of lead neurotoxicity include its substitution for zinc in some enzymes and zinc-finger proteins, and disruption of heme synthesis (Agency for Toxic Substances and Disease Registry, 2007; Skerfving and Bergdahl, 2007).

4.2.2 Related literature

The link between early-life lead exposure and later school-age intellectual performance has been examined in several epidemiological studies. Most of these studies find strong associations between blood lead level (BLL) and IQ, but the results are inconsistent regarding whether early or more recent exposure has the largest effect. For example, Bellinger et al. (1992) examine the relationship between child BLL, measured throughout the period between 6 and 57 months of age, and the WISC-R and K-TEA intelligence tests performed at 10 years of age, and find that only BLL at age 24 month is significantly associated with lower test scores (which is also the age when BLL tends to peak). Similarly, Wasserman et al. (2000) show that lead exposure between 0 and 2 years of age is

[38] Several studies have shown that mean blood lead concentrations in children rapidly increase until the age of 24 months and then gradually decline (Dietrich et al., 1993; Canfield et al., 2003).

most strongly associated with decrements in IQ at later ages (between 3 and 7 years), although prenatal and later-life exposure (at ages 2 to 4) also have statistically significant negative effects on IQ. On the other hand, Dietrich et al. (1993) and Chen et al. (2005) find that concurrent BLL (in children 5 to 7 years of age at the time of IQ test) has stronger negative association with intelligence than BLL at earlier ages.

One of the main concerns in these studies is that BLLs are highly correlated across ages, which makes it difficult to disentangle the size of the effect at a specific age. Also, since lead accumulates in the body, the associations between concurrent BLL and IQ may actually reflect the effect of cumulative exposure during early childhood. Another important limitation of these studies is that they do not account for endogeneity of lead exposure. For example, children with health-cautious or highly educated parents may be exposed to lower levels of lead pollution if these parents live in cleaner areas or take larger steps to avoid pollution (Neidell, 2004). If these parents also invest more in their children's health and cognitive development, then the estimated effect of lead exposure on cognitive skills will be biased upward. On the other hand, since highly educated people tend to reside in urban areas, where traffic (and thus lead pollution) is higher but opportunities to positively affect child's health/intelligence are more abundant, the true effect of lead pollution on cognitive skills may be underestimated.

A study by Nilsson (2009) addresses the latter issue by exploiting exogenous variation in lead pollution resulting from a sharp phase-out of leaded gasoline in Sweden during the 1970s and 1980s. Due to differences in initial levels of lead pollution, the drop in lead exposure varied significantly between municipalities. The results suggest that exposure to lead in early childhood has a negative effect on one's later cognitive ability and labour market outcomes. For example, the reduction in lead pollution during the study period lowered the probability of ending up in the lower end of the GPA distribution by 3.3%, increased high school completion by 0.9%, increased years of schooling by 0.05 years, and decreased the probability of welfare dependency by 0.6 percentage points.

To my knowledge, the only paper that attempts to estimate the causal effect of early-life lead exposure on later cognitive skills in a context of a developing country is by Rau at al. (2013). The authors compare scores in math and language tests (conducted during college enrolment) between individuals residing in the same city but at different distances from a toxic waste site in Chile. They argue that since the waste site was established and abandoned before the city of Arica was built, and the residents were unaware of its high levels of toxicity, the endogeneity of exposure is reduced. The findings suggest that an

increase of BLL during childhood by 1 µg/dL reduces math scores by 0.15 SD and language scores by 0.21 SD. An important limitation of this analysis is that the results may capture the effects of other hazardous materials located at the waste site (some of which are known neurotoxins). This problem is avoided in the present study since the leaded-gasoline phase-out affects only the levels of lead pollution.

4.2.3 Phase-out of leaded gasoline in Uganda

The transition to unleaded gasoline in Uganda was initiated at the Dakar Conference, organized by the World Bank Clean Air Initiative in Sub-Saharan Africa (SSA), in June 2001. The conference aimed at 1) raising awareness about negative health effects of leaded petrol and 2) discussing technical and financial feasibility of the phase-out process.[39] In the resulting Dakar Declaration, the 25 participating SSA countries agreed to phase out leaded fuel by the end of December 31, 2005 (Todd and Todd, 2010).

Implementation of the phase-out programme in Uganda was quick and occurred at the end of 2005. In September 2005, the Ministry of Energy and Mineral Development wrote to Kenya Pipeline Company (the main supplier of petrol in Uganda) requesting that only unleaded petrol be supplied to the Ugandan market. This request was met in December 2005 (Ministry of Energy and Mineral Development, 2006).

[39] There was a common misconception regarding the necessity of adding lead to gasoline. Lead additives had been used since 1930s to enhance gasoline's octane rating. However, new technologies were available to easily and cost-effectively enhance octane ratings (see Lovei, 1998, for more technical details). Another reason for adding lead was that it provided lubrication for the engine valve seats. However, several tests carried out in the United States and Europe had shown that unleaded gasoline does not cause recession of soft valve seats under normal driving conditions (Weaver, 1986). In addition, alternative gasoline additives existed to lubricate the engine (Lovei, 1998).

4.3 Data

4.3.1 The Uwezo surveys

My empirical analysis is based on data from the Uwezo surveys.[40] These are large-scale household surveys conducted in three East African countries (Uganda, Kenya, and Tanzania) annually since 2009. Their main aim is to assess competencies in literacy and numeracy among school-age children. The surveys are representative at the district level for each country. Uwezo employs a two-stage stratified-sampling design (with districts as strata). In the first stage, 30 enumeration areas (typically villages) are selected in each district with probability proportional to population size (of subcounty). In the second stage, 20 households are selected in each enumeration area using systematic random sampling. To account for this sampling design, I apply sample weights throughout the analysis.

As part of the survey, the head of each selected household is asked a set of basic questions about the household (e.g., size of the household, whether the household has direct access to clean water, type of lightning used, etc.), about the children in the household (e.g., age, gender, school grade, etc.), and their parents (age and education level). Next, all children in the household aged between 6 and 16 years (regardless of whether they attend school or not) are administered short literacy and numeracy tests. The literacy test is based on the English language and involves reading a letter, reading a word, reading a paragraph, and reading and comprehending a short story. The numeracy test assesses child's ability to count, recognize numbers, and perform basic operations of addition, subtraction, multiplication, and division. The score for each test is based on the highest level achieved and ranges between 1 and 5 for the language test and between 1 and 7 for the numeracy test. Children of all ages receive tests of the same difficulty level, which corresponds to the level to be attained after two years of schooling.

This paper focuses on the Uwezo surveys for Uganda.[41] The data are available for the survey rounds 2010 to 2014. However, since the 2010 and 2014 surveys cover a small number of districts (27 and 28, respectively), I exclude them from

[40] For more information on the Uwezo initiative, see www.uwezo.net.

[41] Although Kenya and Tanzania phased-out leaded gasoline during the same time, it was not possible to include them in the analysis due to lack of reliable data on district boundaries during the survey years.

the analysis to keep the selection of districts consistent. The 2011, 2012 and 2013 surveys were carried out in (the same) 80 districts out of 112 existing districts.[42]

The Uwezo surveys do not record the child's birthdate. Only the child's age at the time of the survey is known (but not the date when the survey took place). Since the surveys are usually conducted during two months (February and March) every year, for each child there is a span of 14 possible birth months. For example, a child that is 6 years old at the time of the 2011 Uwezo assessment was born somewhere between February 2004 and March 2005. Figure 1 depicts estimated year of birth for all the children in the 2011–2013 surveys (with actual birthdate lying between February of that year and March of the next year). As can be seen from the figure, only one cohort of children (6 year olds in Uwezo 2013) was born after the leaded gasoline phase-out. 6 year olds in Uwezo 2012 were born both before and after the phase-out, while in Uwezo 2011 they were all born at least 9 months before the phase-out. In order to have clear-cut groups of 6 year olds that are exposed and not exposed to lead pollution in utero and during the first year of life, I therefore focus on the 2011 and 2013 rounds of the surveys.[43] For consistency, I exclude the 2012 surveys from the analysis on older children as well.

	1994	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007
												Phase out		
Uwezo 2011	16	15	14	13	12	11	10	9	8	7	6			
Uwezo 2012		16	15	14	13	12	11	10	9	8	7	6		
Uwezo 2013			16	15	14	13	12	11	10	9	8	7	6	

Figure 1: Year of birth of children aged 6 to 16 years old at the time of Uwezo surveys in 2011–2013

Note: Since the Uwezo surveys are conducted during February and March, the actual birthdate lies somewhere between the start of February of the year specified and the end of March of the following year.

[42] Uganda was divided into 80 districts when the 2010 survey was conducted. It had been planned to cover all 80 districts during the following survey rounds. However, since some of the Ugandan districts have been split in two, the surveys in 2011, 2012 and 2013 were conducted in the 55 unsplit districts and in the mother districts (ones that remained with their old names after division), thus covering all 80 of the old districts.

[43] Some of the 6 year olds in the 2013 survey have still been exposed to lead pollution in utero to some extent. Children in this cohort with the longest exposure in utero were born in early February of 2006 and have been exposed to lead for the first 8 months of pregnancy. Children that are born later in the year were exposed for fewer months respectively, and those born after the beginning of October were not exposed at all.

In my analysis (see Section 4.4 for details) I compare children in Uwezo 2011 to children of the same age in Uwezo 2013. For the 6 year olds, this implies that the comparison is done between children not exposed to lead pollution after birth (and in utero to some extent) and children exposed for 1 year and 4 months after birth on average (ranging between 9 months and 1 year and 11 months). For older ages this implies comparison of children with different lengths of exposure after birth. For example, 7 year olds were exposed for 2 years and 4 months after birth on average in Uwezo 2011 and for about 4 months on average in Uwezo 2013. Table 1 lists ranges of exposure durations for children of all the ages covered in Uwezo 2011 and Uwezo 2013. Since exposure to lead pollution is most dangerous during early childhood, I focus on the 6-to-9-year-old children in the main analysis for the sake of brevity (the 9 year olds were exposed to lead pollution for a maximum of 4 years and 11 months after birth). I look at other ages when testing the validity of the research design in Section 4.5.2.

Table 1: Estimated durations of exposure to lead pollution after birth for children of different ages surveyed in the 2011 and 2013 rounds of Uwezo assessments.

	Duration of exposure to lead pollution after birth (range of possible durations)	
Age at survey	*Uwezo 2011*	*Uwezo 2013*
6 years	9 months–1 year 11 months	not exposed
7 years	1 year 9 months–2 years 11 months	not exposed–11 months
8 years	2 years 9 months–3 years 11 months	9 months–1 year 11 months
9 years	3 years 9 months–4 years 11 months	1 year 9 months–2 years 11 months
10 years	4 years 9 months–5 years 11 months	2 years 9 months–3 years 11 months
11 years	5 years 9 months–6 years 11 months	3 years 9 months–4 years 11 months
12 years	6 years 9 months–7 years 11 months	4 years 9 months–5 years 11 months
13 years	7 years 9 months–8 years 11 months	5 years 9 months–6 years 11 months
14 years	8 years 9 months–9 years 11 months	6 years 9 months–7 years 11 months
15 years	9 years 9 months–10 years 11 months	7 years 9 months–8 years 11 months
16 years	10 years 9 months–11 years 11 months	8 years 9 months–9 years 11 months

4.3.2 District traffic-density calculation

In the absence of data on actual levels of lead pollution, I calculate average traffic density in a district as a proxy for lead exposure before the phase-out. To do this, I use the road network data from the World Bank's African Infrastructure and Country Diagnostic programme. It provides comprehensive road data in Uganda (as well as 23 other countries in Africa) as of the period between 2001 and 2006.[44] Importantly, it contains information on annual average daily traffic (AADT) per road segment (usually a piece of the road network between two road junctions that is homogenous in terms of road type, condition and traffic flows).

I use the following procedure to estimate district traffic density. First, I generate a raster (grid cell) map of traffic densities with a resolution of 1 km using the kernel density tool in ArcGIS (see Figure 2). The calculation involves first fitting a kernel surface over each road segment with values gradually declining between the road segment (where it is highest) and the specified search radius (after which the value of the surface is equal to 0). The surface values at any specific distance from the road segments (within the search radius) are higher for road segments with higher AADT values.[45] The traffic density at each output raster cell is then calculated by adding the values of all kernel surfaces that overlay the centre of the cell. This approach is in line with how ambient air concentrations of vehicular lead pollution are spatially distributed, i.e. increasing with traffic volume, declining with distance to roads, and increasing with the density of the road network (Daines et al., 1970). In the main analysis, I use a search radius of 5 km in the kernel density estimation since lead pollution spreads many kilometres from roads (Harrison and Laxen, 1984). The choice of the radius, however, should not make a substantial difference as district traffic densities calculated using different search radiuses are highly correlated (see Table A.1).[46] Finally, I use the resulting raster map to compute the traffic density for each district by averaging the cell values situated within its borders.

[44] The data can be accessed from: www.infrastructureafrica.org.

[45] The surface is defined so the volume under the surface equals the product of the length of the road segment and its AADT value.

[46] I test whether the results are sensitive to the choice of search radius in Section 4.5.2.

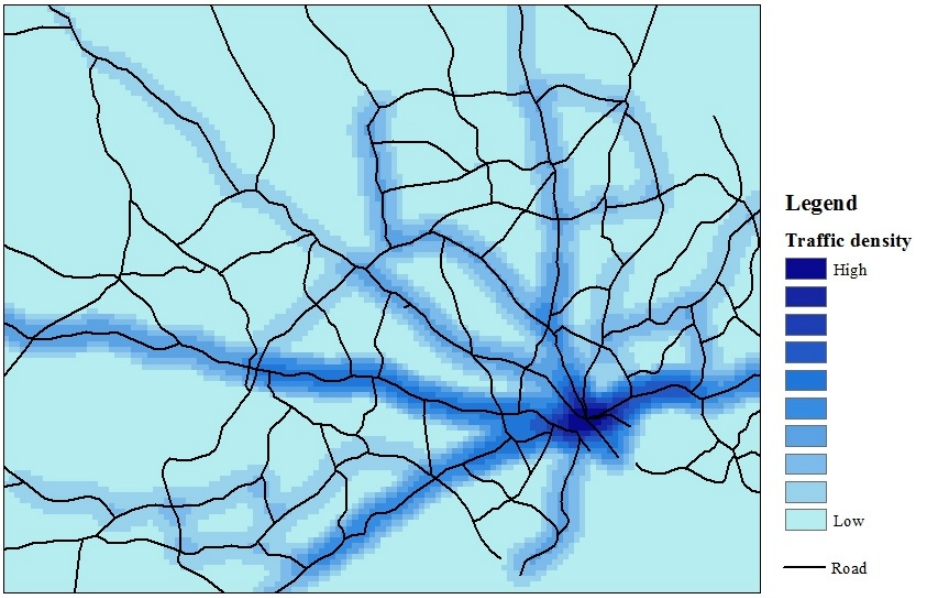


Figure 2: Example of kernel-density estimation of traffic density using a 5 km search radius

Figure 3 depicts estimated traffic density levels in the districts included in the analysis. It can be seen from the figure that most of the districts have relatively low levels of traffic density (below 50) and one district (that of the capital city Kampala) has an extremely high level of 1,500. The remaining 14 districts have traffic densities between 50 and 250.

4.3.3 Descriptive statistics

To perform the analysis, I first allocate the children into groups based on the quartiles of the traffic density values assigned to them based on their district of residence. In the paper, I refer to these groups as very low traffic-density (VLTD), low traffic-density (LTD), middle traffic-density (MTD), and high traffic-density (HTD) groups.[47]

[47] An alternative approach would be to divide the children into groups where the group average traffic-density levels are evenly spaced. However, such approach would yield a disproportionally large number of observations in the group with the lowest traffic density and too few observations in the other groups.

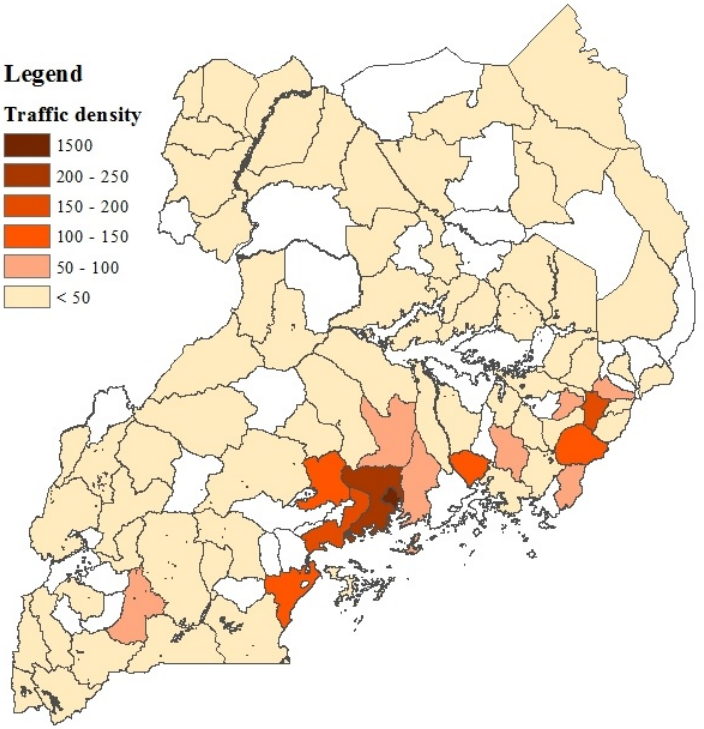


Figure 3: District traffic densities estimated using the kernel density estimation method with 5 km search radius

Table 2 shows summary statistics for the four groups as well as the two survey years. The table reveals some interesting patterns. The average test scores in both math and English increase with the district traffic density. They also increase with time in all four groups. The increase in math test scores with time is generally greater in higher traffic-density districts, which could possibly be explained by the sharper drop in lead pollution in those areas after the leaded-gasoline phase-out. Such tendency is not observed for English test scores. It is important to remember, however, that these results are based on averages for all the children in the dataset. This implies that they may mask stronger effects of lead exposure on test scores expected for the youngest children. Another important observation is that the share of mothers with formal education is higher in higher traffic-density districts, which (together with the above observation of the positive relation between traffic density and test scores) may point to better economic conditions in these regions.

Table 2 – Summary statistics

	Uwezo 2011				Uwezo 2013			
	VLTD	LTD	MTD	HTD	VLTD	LTD	MTD	HTD
District traffic density	5.498	16.57	33.93	290.43	5.633	16.04	33.78	268.81
	(2.439)	(3.683)	(6.493)	(473.185)	(2.415)	(3.909)	(6.429)	(449.228)
Math score (1–7)	4.377	4.425	4.463	4.793	4.519	4.523	4.691	4.987
	(2.265)	(2.297)	(2.254)	(2.215)	(2.170)	(2.140)	(2.138)	(2.089)
English score (1–5)	2.725	2.758	2.835	3.166	2.992	2.909	3.022	3.394
	(1.518)	(1.552)	(1.537)	(1.549)	(1.501)	(1.526)	(1.518)	(1.482)
Child is male (0/1)	0.52	0.51	0.50	0.49	0.52	0.51	0.50	0.50
	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)	(0.50)
School grade (0–13)	3.45	3.58	3.53	3.99	3.450	3.357	3.346	3.585
	(2.012)	(2.166)	(2.148)	(2.390)	(1.876)	(1.912)	(1.918)	(2.093)
Child receives extra lessons (0/1)	0.20	0.25	0.24	0.31	0.12	0.20	0.19	0.25
	(0.397)	(0.433)	(0.429)	(0.463)	(0.328)	(0.398)	(0.393)	(0.434)
Number of children in household	5.39	4.62	4.09	4.93	3.94	3.99	4.04	3.84
	(5.042)	(3.015)	(2.378)	(4.454)	(1.764)	(1.839)	(1.931)	(1.907)
Mother's age	36.08	37.44	37.87	37.67	37.05	36.29	35.97	35.36
	(11.322)	(11.259)	(11.985)	(12.302)	(9.369)	(8.656)	(8.313)	(7.845)
Mother has formal education (0/1)	0.72	0.81	0.79	0.89	0.57	0.70	0.71	0.81
	(0.45)	(0.40)	(0.41)	(0.32)	(0.49)	(0.46)	(0.45)	(0.39)
Observations	21,226	23,013	22,809	21,475	22,458	20,984	20,212	19,370
Age 6	2,149	2,246	2,142	2,192	2,463	2,175	2,123	2,119
Age 7	1,867	2,006	2,059	1,920	2,153	2,059	2,022	1,962
Age 8	2,199	2,426	2,310	2,136	2,419	2,224	2,154	1,959
Age 9	1,818	2,005	1,971	1,908	1,921	1,808	1,823	1,775
Age 10	2,580	2,629	2,711	2,550	2,504	2,395	2,399	2,183
Age 11	1,565	1,786	1,839	1,792	1,480	1,425	1,443	1,345
Age 12	2,402	2,496	2,634	2,257	2,480	2,293	2,217	2,109
Age 13	1,823	2,062	2,135	2,077	1,962	2,056	1,970	1,892
Age 14	1,932	2,182	2,132	1,865	2,037	1,957	1,694	1,655
Age 15	1,482	1,659	1,511	1,477	1,450	1,308	1,259	1,245
Age 16	1,409	1,516	1,365	1,301	1,589	1,284	1,108	1,126

4.4 Empirical strategy

4.4.1 The model

To identify the effect of lead pollution on test scores, I employ a difference-in-difference method. Specifically, I compare children of a particular age surveyed in Uwezo 2011 with those of the same age surveyed in Uwezo 2013. Since children in the former group were born earlier, they were exposed to lead pollution for longer after birth compared to the latter group due to the leaded-

gasoline phase-out at the end of 2005 (see Section 4.3.1 for more detail). The control group includes children who reside in districts with very low traffic density as defined in Section 4.3.3. For these children the exposure to lead pollution prior to the phase-out is expected to be very low. I define three treatment groups comprised of children from low, middle, and high traffic-density districts, respectively. I estimate the following equation separately for ages 6 through 9:

$$Y_{ijt} = \alpha_1 + \alpha_2 \cdot Treat_j + \alpha_3 \cdot After_t + \alpha_4 \cdot \left(Treat_j \cdot After_t\right) + \alpha_4 \cdot X_{ijt} + \varepsilon_{ijt}, \quad (1)$$

where i indexes the child, j treatment status, and t the year of the survey. Y is a test score in math or English. To facilitate interpretation, the test scores are standardized with a mean of 0 and standard deviation of 1 separately for each age. *Treat* is a vector of treatment variables which take a value of 1 if the child resides in a district with a certain traffic-density level (low, moderate, or high) and 0 otherwise (the reference group consists of children residing in districts with very low traffic density). *After* is 1 if a child was surveyed in Uwezo 2013 and 0 if surveyed in Uwezo 2011. X is a vector of controls that include dummy variables for child's grade (1st, 2nd, 3rd, 4th and higher, grade is missing),[48] mother's age (25–30, 31–35, 36–40, 41 or higher, age is missing), mother's education level (primary, secondary and higher, education is missing), an indicator variable for whether the child is male, and an indicator variable for whether the child receives extra lessons. ε is the error term. Standard errors in all regressions are clustered at the village level. The regressions are weighted by households' inverse sample probability (provided in the dataset).

4.4.2 Bad controls

Two of the control variables in Equation 1 are potentially affected by the phase-out of leaded gasoline and may thus be bad controls. First of all, the age at which a child starts school (and thus the grade s/he is attending at the time of the survey) may depend on his/her health and abilities, which are affected by lead pollution. For example, a child with worse health or abilities may be sent to

[48] The reference group consists of children who have never enrolled in school.

school later or not at all.[49] Similarly, receiving extra lessons may be more likely for children with lower abilities or worse health (due to missed classes, for example). I test whether school grade and receiving extra lessons are affected by the phase-out by estimating Equation 1 with the variables under question as outcomes. The results in Table 3 show that the estimates are very small and mostly statistically insignificant. I, therefore, include the discussed variables in the main analysis to increase precision of the estimates. Moreover, it seems reasonable to compare children of the same school grade and access to extra lessons which directly affect the child's knowledge.

Another potential control variable that is available in the dataset is the number of children in a household. However, as seen from the Panel C in Table 3, it is affected by the phase-out and therefore not included in Equation 1. This result is of interest in itself as it implies that lead pollution affects fertility or child mortality (or both). It is interesting, however, that only children in the MTD and (to a lesser extent) LTD groups are negatively affected by lead pollution, while there is no effect in the HTD group. One potential explanation for this result is that more urbanized HTD districts offer better opportunities for parents to protect against and mitigate the negative effects of pollution on child health through better health care, education, and job prospects. It is important to note that a similar pattern in the effect is observed in Paper 2, where I find that the phase-out of leaded gasoline in five SSA countries had no effect on infant mortality within 1 km of major roads, but significantly reduced infant mortality in areas between 1 and 2 km and (to a lesser extent) 2 and 5 km of major roads.

The size of the estimates for the MTD treatment group suggests that children in Uwezo 2013 have, on average, one more sibling compared to children in Uwezo 2011. This corresponds to a 24.6% increase in the number of children per household. While this is a large effect, it is supported by similar results in Paper 2, where infant mortality fell by 16.6% in areas between 1 and 2 km from major roads and by 8.7% in areas between 2 and 5 km from major roads following the phase-out. While the effect in this study is larger, it also captures the effect of lead pollution on fertility, foetus survival, and child mortality after age 1 in addition to the effect on infant mortality.

[49] The minimum age at which a child can go to school in Uganda is six years (established in 2009), but there is no maximum age. Moreover, schooling became compulsory in Uganda only since 2009.

Table 3: Testing for bad controls

	Age 6	Age 7	Age 8	Age 9
Panel A. Grade				
Treat • After (LTD)	0.049	0.004	–0.033	–0.095
	(0.047)	(0.057)	(0.069)	(0.084)
Treat • After (MTD)	0.031	0.067	0.0004	–0.067
	(0.043)	(0.056)	(0.066)	(0.078)
Treat • After (HTD)	0.083	–0.014	–0.023	–0.081
	(0.052)	(0.062)	(0.076)	(0.093)
R^2	0.009	0.016	0.025	0.039
Observations	18,856	16,670	18,644	15,522
Panel B. Tuition				
Treat • After (LTD)	0.033	0.048*	0.006	0.024
	(0.025)	(0.028)	(0.029)	(0.035)
Treat • After (MTD)	0.011	0.023	0.008	0.026
	(0.024)	(0.025)	(0.025)	(0.028)
Treat • After (HTD)	0.048	0.03	–0.015	0.015
	(0.029)	(0.031)	(0.034)	(0.035)
R^2	0.010	0.018	0.021	0.018
Observations	20,704	17,706	19,351	15,971
Panel C. Number of children in household				
Treat • After (LTD)	0.523	0.627	0.973**	0.495
	(0.436)	(0.59)	(0.473)	(0.551)
Treat • After (MTD)	0.964**	1.000*	1.042**	0.902*
	(0.413)	(0.562)	(0.472)	(0.515)
Treat • After (HTD)	–0.168	0.399	–0.272	–0.259
	(0.569)	(0.632)	(0.704)	(0.708)
R^2	0.020	0.015	0.029	0.029
Observations	20,704	17,706	19,351	15,971
Controls	No	No	No	No

Notes: Each column corresponds to a separate regression. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities respectively. The *Grade* variable ranges between 0 and 13, with 0 being assigned to children who have never enrolled in school. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level and ** at 5%.

4.4.3 Interpretation

The parameter of interest in Equation 1 is α_4. It shows the difference in test scores between children with different lengths of exposure to lead pollution in the treatment groups less the corresponding difference in the control group. If reductions in lead pollution following the phase-out had a beneficial effect on child cognitive skills, we expect α_4 to be positive.

For parameter α_4 to have a causal interpretation, the control group must provide a valid counterfactual for changes in test scores in treatment groups had the phase-out not occurred. A good indicator that this requirement holds would be that the control and treatment groups exhibit similar trends in test scores before the phase-out. However, it is not possible to check whether this is the case since I do not have data for preceding years. I support the validity of my control group with an alternative check in Section 4.5.2.

Another issue that could bias my estimates is selective migration. For example, if improvements in air quality in high-traffic areas after the phase-out attracted families with characteristics positively related to a child's cognitive skills, the estimate of the effect of pollution on test scores would be overestimated. I address this concern in Section 4.5.2.

4.5 Results

4.5.1 Baseline results

Table 4 presents the results of estimating Equation 1. The results suggest that the phase-out of leaded gasoline had a statistically significant positive effect on test scores in both math and English, but mostly for the group with the highest traffic density. It is interesting to contrast these findings with the results for the number of children per household in Table 3, where the effect is also increasing with the traffic density of one's district of residence, but is absent for the highest traffic-density group. I hypothesized that the absence of the effect in the HTD group is due to the ability of parents in this group to prevent and compensate for the negative effects of lead pollution on child health. If this proposition holds, then the finding of a strong and statistically significant effect on test scores in the HTD group implies that parents in this group are not as successful at mitigating the negative effects of lead pollution on cognitive skills of their children. This conclusion would be in line with abundant existing evidence that negative neurological effects of early-life lead exposure are not fully reversible (Needleman et al., 1990; Cecil et al., 2008; Rogan et al., 2001; Dietrich et al., 2004).

The finding that the leaded-gasoline phase-out caused an increase in the number of children per household in the LTD and MTD groups offers two explanations to why there is no effect on test scores in these groups. First, in accordance with the "quantity–quality trade-off" theory of family size (see, e.g.,

Table 4: Effect of leaded gasoline phase-out on child test scores (in standard deviations)

	Age 6	Age 7	Age 8	Age 9
Panel A. Math				
Treat • After (LTD)	0.124	0.065	0.072	−0.021
	(0.077)	(0.078)	(0.059)	(0.067)
Treat • After (MTD)	0.07	0.026	0.087	0.156**
	(0.076)	(0.078)	(0.06)	(0.063)
Treat • After (HTD)	0.239***	0.202**	0.172**	0.154**
	(0.084)	(0.089)	(0.071)	(0.07)
R^2	0.143	0.256	0.284	0.318
Observations	17,182	15,654	17,429	14,745
Panel B. English				
Treat • After (LTD)	0.043	0.032	−0.024	−0.031
	(0.073)	(0.08)	(0.058)	(0.07)
Treat • After (MTD)	−0.058	−0.035	−0.03	0.012
	(0.073)	(0.081)	(0.058)	(0.07)
Treat • After (HTD)	0.164*	0.223**	0.083	0.123
	(0.098)	(0.098)	(0.076)	(0.076)
R^2	0.141	0.267	0.313	0.393
Observations	17,169	15,653	17,450	14,757
Controls	Yes	Yes	Yes	Yes

Notes: The table presents estimates of α_4 from Equation 1. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities respectively. The control variables include: child's grade (1st, 2nd, 3rd, 4th+, missing), gender, mother's age (25–30, 31–35, 36–40, 41+, missing), mother's education level (primary, secondary and higher, missing), and whether the child receives extra lessons/tuition. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level, ** at 5%, and *** at 1%.

Becker and Lewis, 1973; Becker and Tomes, 1976), an increase in the number of children leads to lower investments per child, given the limited family budget, and thus worse outcomes for individual children. Second, if the number of children per household increased due to higher survival rate of children and the children saved by the phase-out are, on average, weaker and have lower cognitive skills than the other children, then the estimates of the effect of the phase-out on test scores are biased downward.

Another important tendency observed in Table 4 is that the effect on math scores (in the HTD group) gradually declines with the child's age. This is a clear indication that lead exposure is more harmful during earlier years of life.[50] For

[50] Remember that, for the 6 year olds, the comparison is done between those who were exposed to lead pollution for about a year after birth and those not exposed. For the 9 year olds, on the other hand, the comparison is done between those exposed for about four years after birth and those exposed for about two years after birth. Therefore, the estimates for the 9 year olds capture the effect of exposure at a later age compared to the estimates for the 6 year olds.

the English test scores, the tendency is similar in the sense that the effect is strong and significant for the ages 6 and 7, but smaller and insignificant for the ages 8 and 9. The estimate for the 6 year olds is, however, smaller than for the 7 year olds. One possible explanation for this is that the effect for the 6 year olds is underestimated due to increased foetal survival after the phase-out. That is to say, if the phase-out of leaded gasoline increased the probability of foetal survival, and if the marginal surviving foetus has worse brain health compared to others, then for the 6 year olds in Uwezo 2013 this effect will be translated into lower test scores, thus biasing the estimates downwards.[51] The fact that the effect for English is only significant for ages 6 and 7 suggests that the brain functions employed during reading are mostly formed (or most susceptible to lead pollution) during first three years of life.

The size of the effect varies between 0.15 and 0.24 SD for math and between 0.16 and 0.22 SD for English test scores in the HTD group. For comparison, a 60% reduction of lead pollution in Sweden due to a leaded-gasoline phase-out increased Grade 9 GPA by 0.04 SD in the quartile with highest exposure (Nilsson, 2009).[52]

4.5.2 Testing the validity of the research design

This section addresses potential concerns regarding the validity of the research strategy. One such concern is that the control group does not provide a valid counterfactual for test results in the treatment group. For example, the strong and statistically significant improvement in test scores in the HTD group compared to the VLTD group found in Section 4.5.1 could have been caused by higher rate of improvement in education quality in the (more urbanized) HTD districts rather than by the phase-out of leaded gasoline. One way to test whether this is the case is to estimate Equation 1 for the older children. If improvements in test scores are driven by improvements in education quality (or other changes affecting test scores) in the HTD districts, then this effect would apply to children of all the ages. On the other hand, the effect of the leaded gasoline phase-out should be more pronounced for younger children since for them the estimates capture the effect of exposure during the period when the

[51]As discussed in Section 4.3.1, only the cohort of 6 year olds in Uwezo 2013 are either not exposed to lead pollution in utero or exposed only during some part of the gestational period

[52] Although the two studies are not directly comparable due to differences in methodology and outcomes, they are similar in terms of potential lead pollution levels as the initial maximum allowed lead levels in gasoline was 0.4 g/L in both cases.

nervous system is most susceptible to the toxic effects of lead pollution. Table 5 shows the results for children aged 10 to 16. We can see that the coefficients are very small and mostly statistically insignificant. Only one of the positive estimates is statistically significant at the 5% level. However, given the number of regressions, it is not surprising that some of the coefficients are statistically significant. Many of the estimates (especially for the later ages) are actually negative, suggesting that there may be a downward bias in the main results in Table 4 since the test scores in the treatment groups would have declined compared to the control group without the phase-out. Most of these estimates are statistically insignificant, however.

Table 5: Estimates for older children (in standard deviations)

	Age 10	Age 11	Age 12	Age 13	Age 14	Age 15	Age 16
Panel A. Math							
Treat • After (LTD)	0.069	0.020	0.032	0.047	–0.119*	–0.035	–0.089
	(0.06)	(0.067)	(0.058)	(0.062)	(0.069)	(0.089)	(0.078)
Treat • After (MTD)	0.063	0.125**	0.040	0.079	–0.066	0.093	0.035
	(0.058)	(0.063)	(0.058)	(0.061)	(0.066)	(0.086)	(0.072)
Treat • After (HTD)	0.100	0.099	0.057	0.027	–0.017	0.025	–0.120
	(0.065)	(0.071)	(0.058)	(0.066)	(0.072)	(0.095)	(0.0860)
R^2	0.299	0.286	0.267	0.204	0.155	0.140	0.124
Observations	19,574	12,454	18,534	15,668	15,195	11,200	10,502
Panel B. English							
Treat • After (LTD)	–0.047	–0.079	–0.040	–0.117*	–0.053	–0.048	–0.121
	(0.059)	(0.059)	(0.059)	(0.061)	(0.067)	(0.074)	(0.077)
Treat • After (MTD)	–0.06	–0.096	–0.023	–0.062	–0.043	–0.018	–0.060
	(0.057)	(0.059)	(0.058)	(0.060)	(0.066)	(0.072)	(0.076)
Treat • After (HTD)	0.040	0.054	0.074	–0.062	0.041	0.052	–0.054
	(0.063)	(0.062)	(0.061)	(0.065)	(0.069)	(0.081)	(0.087)
R^2	0.360	0.379	0.346	0.275	0.211	0.163	0.120
Observations	19,613	12,481	18,631	15,741	15,233	11,233	10,555
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: The table presents estimates of α_4 from Equation 1. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities, respectively. The control variables include child's grade (1st, 2nd, 3rd, 4th+, missing), gender, mother's age (25–30, 31–35, 36–40, 41+, missing), mother's education level (primary, secondary and higher, missing), and whether the child receives extra lessons. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level and ** at 5%.

Next, I investigate whether the phase-out affected the composition of mothers in the treatment and control groups, which could lead to biased estimates in Section 4.5.1. To do this, I estimate Equation 1 with two mother's characteristics as outcomes: mother's age and whether the mother has any formal education. The results are shown in Table 6. The estimates for mother's

Table 6: Effect of leaded gasoline phase-out on mother's characteristics

	Age 6	Age 7	Age 8	Age 9
Panel A. Mother has formal education				
Treat • After (LTD)	0.023	0.024	0.029	0.064*
	(0.032)	(0.032)	(0.035)	(0.034)
Treat • After (MTD)	0.059*	0.018	0.046	0.043
	(0.032)	(0.033)	(0.034)	(0.033)
Treat • After (HTD)	0.052*	0.024	0.049	0.073**
	(0.031)	(0.031)	(0.032)	(0.031)
R^2	0.055	0.049	0.053	0.044
Observations	19,039	16,280	17,731	14,699
Panel B. Mother's age				
Treat • After (LTD)	−1.920***	−2.034***	−2.051***	−2.307***
	(0.571)	(0.697)	(0.585)	(0.703)
Treat • After (MTD)	−2.750***	−2.545***	−2.698***	−3.382***
	(0.576)	(0.685)	(0.619)	(0.669)
Treat • After (HTD)	−3.171***	−3.283***	−3.436***	−3.814***
	(0.709)	(0.738)	(0.670)	(0.839)
R^2	0.008	0.011	0.010	0.010
Observations	19,139	16,334	17,691	14,572
Controls	No	No	No	No

Notes: The table presents estimates of α_4 from Equation 1. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities, respectively. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level and ** at 5%, and *** at 1%.

education are positive but mostly insignificant. The estimates for mother's age, however, are all statistically significant and negative. This is somewhat counterintuitive as we expect older mothers to sort into cleaner areas. One possible explanation is that these results are driven not by the leaded-gasoline phase-out, but by the general trend of migration of young, educated mothers from rural to urban areas (as higher traffic density implies higher level of urbanization). Such maternal sorting could still bias the estimates of the effect of lead pollution on child test scores if not controlled for.

Table 7 shows the results of estimating Equation 1 without control variables. Some of the point estimates for math test scores in the LTD and MTD groups increase in size and are now statistically significant. Given the possibility of maternal sorting discussed above, this is not surprising and shows the importance of controlling for maternal characteristics. Most of the estimates for the HTD group are nevertheless stable.

Table 7: Excluding controls

	Age 6	Age 7	Age 8	Age 9
Panel A. Math				
Treat • After (LTD)	0.182**	0.108	0.126*	−0.035
	(0.08)	(0.083)	(0.069)	(0.075)
Treat • After (MTD)	0.135*	0.058	0.148**	0.166**
	(0.078)	(0.084)	(0.069)	(0.069)
Treat • After (HTD)	0.295***	0.137	0.198**	0.124
	(0.089)	(0.098)	(0.086)	(0.083)
R^2	0.024	0.034	0.035	0.032
Observations	17,182	15,654	17,429	14,745
Panel B. English				
Treat • After (LTD)	0.094	0.074	0.018	−0.030
	(0.076)	(0.084)	(0.07)	(0.074)
Treat • After (MTD)	0.007	0.003	0.024	0.015
	(0.076)	(0.085)	(0.068)	(0.077)
Treat • After (HTD)	0.213**	0.137	0.065	0.093
	(0.103)	(0.112)	(0.095)	(0.095)
R^2	0.048	0.063	0.065	0.067
Observations	17,169	15,653	17,450	14,757
Controls	No	No	No	No

Notes: The table presents estimates of α_4 from Equation 1 without controls. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities, respectively. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level, ** at 5%, and *** at 1%.

Finally, in Table 8, I test whether the results are sensitive to the chosen search radius when calculating district traffic density (see Section 4.3.2 for more detail). The left-hand panel shows results when using a smaller search radius of 1 km; in the right-hand panel, I use a larger 20 km radius. In both cases the estimates are very similar to the main results in Table 4, both in terms of size and statistical significance.

4.5.3 Heterogeneity

This section explores whether the effect of the phase-out on test scores differs across population subgroups. I start by analysing whether the effect is different by child gender. The previous literature provides conflicting evidence as to whether there are gender differences in susceptibility to lead. For example, Jedrychowski et al. (2009) document that prenatal lead exposure is negatively associated with cognitive function at age 3 in boys, but not girls. On the other

Table 8: Alternative search radiuses when calculating district traffic density

	1 km				20 km			
	Age 6	Age 7	Age 8	Age 9	Age 6	Age 7	Age 8	Age 9
Panel A. Math								
Treat • After (LTD)	0.099	0.073	0.061	−0.036	0.144*	0.080	0.076	0.003
	(0.079)	(0.080)	(0.060)	(0.067)	(0.078)	(0.079)	(0.059)	(0.066)
Treat • After (MTD)	0.061	0.039	0.075	0.139**	0.084	0.046	0.089	0.122*
	(0.078)	(0.081)	(0.062)	(0.065)	(0.077)	(0.080)	(0.061)	(0.064)
Treat • After (HTD)	0.222***	0.198**	0.165**	0.148**	0.230***	0.210**	0.173**	0.158**
	(0.085)	(0.090)	(0.071)	(0.070)	(0.085)	(0.093)	(0.072)	(0.070)
R^2	0.143	0.255	0.284	0.318	0.142	0.255	0.284	0.317
Observations	17,182	15,654	17,429	14,745	17,182	15,654	17,429	14,745
Panel B. English								
Treat • After (LTD)	0.060	0.054	−0.020	−0.0194	0.041	0.029	−0.019	−0.022
	(0.074)	(0.082)	(0.059)	(0.072)	(0.073)	(0.081)	(0.058)	(0.070)
Treat • After (MTD)	−0.036	−0.027	−0.030	−0.003	−0.067	−0.025	−0.044	0.010
	(0.075)	(0.083)	(0.059)	(0.073)	(0.075)	(0.083)	(0.059)	(0.072)
Treat • After (HTD)	0.166*	0.230**	0.078	0.140*	0.157	0.221**	0.097	0.130*
	(0.097)	(0.099)	(0.075)	(0.076)	(0.099)	(0.100)	(0.077)	(0.077)
R^2	0.142	0.268	0.314	0.393	0.139	0.265	0.313	0.390
Observations	17,169	15,653	17,450	14,757	17,169	15,653	17,450	14,757
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: The table presents estimates of α_4 from Equation 1. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities, respectively. The control variables include child's grade (1st, 2nd, 3rd, 4th+, missing), gender, mother's age (25–30, 31–35, 36–40, 41+, missing), mother's education level (primary, secondary and higher, missing), and whether the child receives extra lessons. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level and ** at 5%, and *** at 1%.

hand, Nilsson (2009) finds that the effect of early-life lead exposure on 9th-grade GPA and IQ-test scores is similar between boys and girls. Gender differences in susceptibility to lead may arise due to different rates of cognitive development in early childhood, but also as a result of different behavioural response by parents to both lead pollution and its health effects. The results in Table 9, however, do not suggest that the effect is different: some of the estimates indicate a stronger effect for boys, but many of the coefficients are similar between the genders and some of them are actually larger and more statistically significant for girls.

Next, I examine whether the effect differs by mother's education level. There are several reasons why children born to mothers with different education levels may be affected differently by pollution. First, children born to less-educated mothers may have lower initial health levels, making them more sensitive to pollution. Second, less-educated mothers may have less knowledge and opportunity to avoid pollution. Third, more educated mothers may have more

Table 9: Effects by child's gender

	Male				Female			
	Age 6	Age 7	Age 8	Age 9	Age 6	Age 7	Age 8	Age 9
Panel A. Math								
Treat • After (LTD)	0.118	0.074	0.079	−0.023	0.132	0.052	0.065	−0.012
	(0.103)	(0.087)	(0.075)	(0.077)	(0.084)	(0.103)	(0.072)	(0.095)
Treat • After (MTD)	0.077	−0.037	0.115	0.110	0.066	0.093	0.069	0.209**
	(0.104)	(0.087)	(0.076)	(0.075)	(0.081)	(0.103)	(0.072)	(0.091)
Treat • After (HTD)	0.258**	0.083	0.239**	0.169*	0.223**	0.333***	0.111	0.145
	(0.118)	(0.100)	(0.102)	(0.087)	(0.090)	(0.116)	(0.080)	(0.094)
R^2	0.121	0.240	0.273	0.311	0.169	0.276	0.298	0.326
Observations	8,658	8,080	8,541	7,501	8,524	7,574	8,888	7,244
Panel B. English								
Treat • After (LTD)	0.047	−0.078	−0.015	−0.022	0.038	0.135	−0.028	−0.037
	(0.086)	(0.087)	(0.073)	(0.078)	(0.085)	(0.101)	(0.074)	(0.095)
Treat • After (MTD)	−0.0005	−0.092	−0.014	0.014	−0.113	0.019	−0.044	0.010
	(0.093)	(0.089)	(0.073)	(0.077)	(0.080)	(0.101)	(0.074)	(0.093)
Treat • After (HTD)	0.304**	0.033	0.129	0.159*	0.027	0.429***	0.041	0.097
	(0.138)	(0.113)	(0.108)	(0.083)	(0.100)	(0.118)	(0.087)	(0.100)
R^2	0.138	0.256	0.320	0.380	0.152	0.286	0.308	0.408
Observations	8,633	8,054	8,573	7,501	8,536	7,599	8,877	7,256
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: The table presents estimates of α_4 from Equation 1. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities, respectively. The control variables include child's grade (1st, 2nd, 3rd, 4th+, missing), gender, mother's age (25–30, 31–35, 36–40, 41+, missing), mother's education level (primary, secondary and higher, missing), and whether the child receives extra lessons. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level and ** at 5%, and *** at 1%.

resources to mitigate and compensate for negative health shocks. All these factors imply a stronger negative effect of lead pollution for children from less-educated families. However, the effect could also be smaller if living in (poorer) polluted areas made individuals adapt to pollution by taking more stringent protective measures.

To analyse this, I estimate Equation 1 separately for mothers with and without formal education. The results, shown in Table 10, do not suggest that the effect is systematically larger for any of the groups. Instead, some of the estimates are larger for one group while others are larger for the other group.

Table 10: Effects by mother's education

	Mother has formal education				Mother has no formal education			
	Age 6	Age 7	Age 8	Age 9	Age 6	Age 7	Age 8	Age 9
Panel A. Math								
Treat • After (LTD)	0.079	0.037	0.145*	−0.068	0.311**	0.245*	−0.029	0.216**
	(0.088)	(0.089)	(0.074)	(0.085)	(0.133)	(0.125)	(0.089)	(0.110)
Treat • After (MTD)	0.040	0.011	0.152**	0.185**	0.054	0.10	−0.011	0.085
	(0.084)	(0.087)	(0.074)	(0.082)	(0.138)	(0.120)	(0.088)	(0.102)
Treat • After (HTD)	0.258***	0.118	0.163*	0.104	−0.199	0.342**	0.005	0.258**
	(0.093)	(0.10)	(0.086)	(0.087)	(0.175)	(0.150)	(0.131)	(0.122)
R^2	0.133	0.236	0.269	0.298	0.139	0.217	0.232	0.274
Observations	11,989	10,930	11,775	10,241	3,946	3,536	4,262	3,376
Panel B. English								
Treat • After (LTD)	0.024	−0.029	0.029	−0.034	0.109	0.180	−0.144	0.026
	(0.081)	(0.094)	(0.073)	(0.088)	(0.132)	(0.114)	(0.097)	(0.097)
Treat • After (MTD)	−0.042	−0.064	0.038	0.091	−0.157	−0.006	−0.19**	−0.17*
	(0.083)	(0.094)	(0.074)	(0.088)	(0.125)	(0.115)	(0.091)	(0.099)
Treat • After (HTD)	0.193*	0.089	0.053	0.110	−0.249	0.452***	0.030	0.094
	(0.113)	(0.118)	(0.096)	(0.093)	(0.152)	(0.138)	(0.123)	(0.128)
R^2	0.120	0.232	0.286	0.382	0.115	0.193	0.289	0.279
Observations	11,987	10,932	11,775	10,241	3,942	3,535	4,307	3,394
Controls	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: The table presents estimates of α_4 from Equation 1. LTD, MTD, and HTD represent groups of children residing in districts with low, medium, and high traffic densities, respectively. The control variables include child's grade (1st, 2nd, 3rd, 4th+, missing), gender, mother's age (25–30, 31–35, 36–40, 41+, missing), mother's education level (primary, secondary and higher, missing), and whether the child receives extra lessons. Standard errors (in parentheses) are clustered at the village level. * indicates statistical significance at the 10% level and ** at 5%, and *** at 1%.

4.6 Conclusion

Using Uganda's leaded-gasoline phase-out as a source of exogenous variation in lead pollution, I show that exposure to lead early in life has a lasting negative effect on one's cognitive skills. Specifically, I find that the phase-out improved test scores of children aged 6 to 9 residing in districts with high traffic density compared to children from districts with very low traffic density. The effect ranges from 0.15 to 0.24 SD for math scores and from 0.16 to 0.22 SD for English scores. It declines with child's age, indicating that lead pollution is more harmful during earlier years of life.

The effect is considerably larger than the one found in a similar study by Nilsson (2009) for Sweden. This is in line with the recent literature showing that the negative health effects of pollution are much stronger in developing

countries than in developed countries (Currie and Vogl, 2013; Tanaka, 2015; Arceo et al., 2016). Developing countries also tend to have much larger pollution levels than developed countries. Lead pollution is not an exception. According to the World Health Organization (2009), about 98% of adults and 99% of children affected by lead exposure live in low- and middle-income countries. Until recently, the vast majority of studies on pollution abatement benefits focused on developed countries. The considerations presented above, however, highlight the importance of directing research efforts to the developing world.

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Appendix

Table A.1: Correlation matrix of district traffic densities estimated with different search radiuses

	1 km	5 km	10 km	20 km	30 km	50 km
1 km	1					
5 km	0.9998	1				
10 km	0.9978	0.9989	1			
20 km	0.9721	0.9766	0.9855	1		
30 km	0.9136	0.9214	0.9382	0.983	1	
50 km	0.7777	0.7894	0.8153	0.8974	0.9613	1

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