The Long-term Effects of Early Childhood Lead Exposure: Evidence from the Phase-out of Leaded Gasoline

J Peter Nilsson[♠]
Uppsala University and IFAU
November 2009

Abstract

From the late 1970s through mid 1990s blood-lead levels decreased drastically in Swedish children due to the sharp phase-out of leaded gasoline. Exploiting the distinct geographical variation in early childhood lead exposure induced by the regulations together with micro data on all children in nine birth cohorts I show that reduced lead exposure early in life improves scholastic performance, cognitive ability, and labor market outcomes among young adults. At the relatively low levels of exposure considered, the analysis reveals a nonlinear relationship between local air lead levels in early childhood and adult outcomes, indicating the existence of a threshold below which further reductions no longer improve adult outcomes. Importantly, the effect is greater for children of lower socioeconomic status (SES), suggesting that pollution is one mechanism through which SES affects long-term economic outcomes and that environmental policies potentially can reduce the intergenerational correlation in economic outcomes.

Keywords: Environmental policy, early-life conditions, earnings

[♠] I would like to thank to Per-Anders Edin, Matz Dahlberg, Lena Hensvik, Per Johansson, Patrik Hesselius, Hans Grönqvist, Katharina Janke, Per Pettersson-Lidbom, Staffan Skerfving, Ulf Strömberg, Germund Tyler and seminar participants in Uppsala and at the RTN Microdata methods and practices meeting at the Institute for Fiscal Studies in London, and Bristol for useful comments and discussions. Björn Öckert kindly shared data with me as did Olle Nordell at Landskrona municipality and Staffan Skerfving at Lunds University. Correspond via peter.nilsson@nek.uu.se. Any remaining errors are my own.

1 Introduction

From the end of the 1960s government air pollution regulations have become increasingly stricter throughout the developed world. This has lead to sharply improved air quality in many countries and regions.[1] Recently, the air pollution reductions following some of these regulations have been shown to improve neonatal health and to reduce infant mortality (Chay and Greenstone, 2003a; Currie and Neidell, 2005; Lüchinger, 2009). However, these and previous studies have not been able to asses the potential long run effects of exposure to poor air quality in early childhood on the surviving infants and children.[2] Since children who are on the life/death margin at birth only constitute a small fraction of all children, the total cost of air pollution in terms of its impact on child health could potentially be much higher if also the sub-clinical effects on the general population of children are taken into account. For example, many of the pollutants released are neurotoxicants that potentially impair children's development in early life even at low levels of exposure. Such effects, even if not apparent at birth or in early childhood, can cause psychological or behavioral problems that first become apparent later on. Moreover, if skills beget skills, as suggested by Cunha and Heckman (2007) even if the direct health damage inflicted by pollutants early in life is fully reversible over time, long-run outcomes could still be affected through dynamic complementarities in human capital accumulation.

In order to shed light on how exposure to poor air quality early in life affects adult outcomes this study focuses on the causal impact of early childhood air lead exposure on subsequent cognitive skills, educational attainments, and labor market outcomes among young adults in Sweden. By merging unique data on local air lead levels in early childhood with comprehensive population micro data, it is possible to follow all children in nine birth cohorts from birth throughout school and examine their experiences on the labor market as young adults. The outcomes considered include scholastic performance (Grade 9 GPA), cognitive ability test scores

[1] See e.g. Giussani (1994) for a thorough report of the impact and history of pollution regulations in the UK. In the developing world along with increased economic growth pollution levels are still increasing rapidly. China stands out as a stark example where air pollution levels as measured between 1983 and 1993 were up to 5 times higher than what was observed in the US *before* the passage of the 1970 Clean Air Act Amendments (Almond et al., 2009).

[2] A notable exception is Reyes (2007) who focuses on the relationship between lead exposure in childhood and violent crime rates. I describe her study (and the differences to this study) in detail below.

(males, age 18), educational attainments and early social and labor market outcomes. These outcomes have previously been shown to be predictive of subsequent outcomes throughout the life cycle, and should therefore be particularly interesting from a public policy perspective.

The local lead pollution data stems from a previously unexplored data source. Since the early 1970s the Swedish environmental protection agency has used moss (bryophyte) samples covering the whole of Sweden to examine regional differences and trends in heavy metal air pollution levels. Mosses are particularly useful as air pollution biomonitors since they lack roots and therefore solely absorb heavy metal depositions from the air. The use of moss as biomonitors for ambient heavy metal air pollution is well established and the program has gradually been expanded; first to the rest of the Scandinavian countries, and since 1995 most other parts of Europe. In a companion study Nilsson et al. (2009) show that moss lead levels is a good predictor of blood lead levels in children.

Although a consensus exists on the health impact of high levels of lead exposure on adult health, the association between lower levels of lead exposure in childhood and cognitive development is still under debate (Canfield et al., 2003; Lanphear et al., 2000). The main reason is that lead exposure is not randomly distributed across locations, and hence confounding is a serious concern as highlighted by e.g. Bellinger (2004a). For example, parents with higher incomes or preferences for cleaner air are likely to sort into areas with better air quality and hence their children are less likely to be exposed to high levels of lead pollution. Failing to account for residential sorting of this kind can result in an upwardly biased estimate of the effect of lead exposure on children's subsequent outcomes. On the other hand, pollution tends to be higher in densely populated areas and at the same time metropolitan areas often attract highly educated parents with more resources, contain better access to quality child care, schools, health care and other amenities that are positively associated with adult outcomes. Such local amenities could in turn result in an underestimated role of childhood lead exposure if not properly taken into account.

To mitigate these and similar concerns this study focuses on children born from the early 1970s until the mid 1980s. The reason is that during the 1970s, along with many other developed countries, Sweden initiated a gradual phase-out of leaded gasoline in order to protect the environment and public health. In Sweden, the main reduction in gasoline-lead levels occurred between 1973 and 1981 when the maximum allowed lead level per litre of gasoline dropped by 79% (Table 1). Since gasoline lead was the main source of lead exposure in the general population[3], as shown in Figure 1, children's

[3] 80% of the air lead levels in the late 1980s where due to traffic (MOENR, 1994).

blood-lead levels (B-Pb) decreased drastically from the 1970s until the mid 1990s when leaded gasoline finally was banned.[4]

Table 1 Changes in maximum allowed gasoline lead levels

Date of policy change:	Maximum lead content:
1 Jan 1970	Max 0.7 g/L (2.65 g/gal)
1 Jan 1973	Max 0.4 g/L (1.51g/gal)
1 Jan 1980	Max 0.15 g/L (0.56g/gal) for regular
1 Jan 1981	Max 0.15 g/L (0.56g/gal) for premium
1 Jan 1986	Leaded regular gasoline is prohibited
1 March 1995	Total ban on lead for all gasoline grades

Source: The Swedish Petroleum Institute.

Due to large differences in initial lead levels the phase-out of leaded gasoline induced substantial variation across localities in the reduction of lead exposure. In the main analysis I exploit the differential changes in early childhood lead exposure for the cohorts born between 1972 and 1984. I compare changes in outcomes for children born in municipalities experiencing large reductions in lead exposure with changes in outcomes of children born in municipalities with only minor changes in air lead levels. By exploiting these differential changes in exposure across birth cohorts within the same municipalities unobserved time-invariant differences between the municipalities is taken into account.

[4] Similar large reductions in blood lead levels associated with the phase-out of lead from gasoline have been documented in many countries (c.f. Thomas et al., 1999). Other sources of lead exposure such as leaded paint was banned in the early 1920's and are therefore believed not to have caused the reductions in blood lead levels during the 70's and 80's. The costs associated with phasing out lead have been shown to be low, c.f. OECD (1999). After 1995 children's blood lead first seemed to stabilize at around 2µg/dL, but since 2000 it has continued to decrease, albeit at a slower speed in absolute terms, c.f. Strömberg et al. (1995, 2001, 2008).

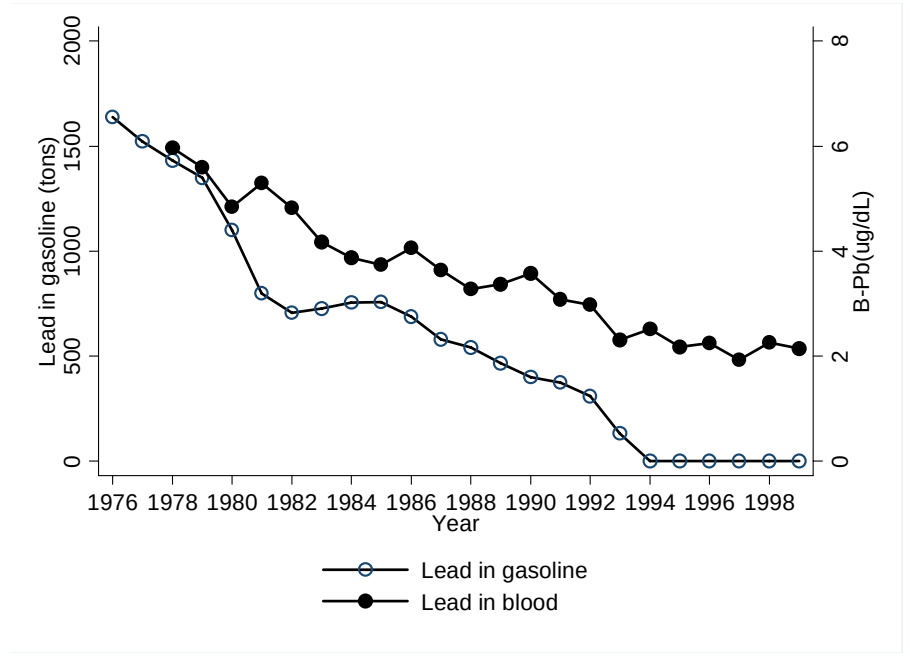

Figure 1 Mean blood-lead levels among primary school children and tons of lead added to gasoline 1976-99. *Source*: Strömberg et al. (1995) and Strömberg et al. (2003).

The importance of taking such unobserved characteristics into account is highlighted by a cross-sectional analysis showing that several *predetermined* parental characteristics that are strongly correlated with children's adult outcomes also are strongly correlated with their children's lead exposure. That is, higher lead exposure for the *child* is associated with lower educational attainments among the *parents*. This result suggests that cross-sectional estimates of the role of early childhood lead exposure are likely to overestimate the relationship between early lead exposure and subsequent outcomes due to omitted variable bias. In contrast, the within municipality variation in lead exposure induced by the gasoline lead level regulations is *not* significantly correlated with the predetermined parental characteristics, which provides support for the validity of the main identification strategy. In addition, besides several important individual, parental and municipality of birth control variables the data also contain unique family identifiers which enable a comparison of outcomes of full siblings with different early childhood lead exposure levels. By comparing differences in adult outcomes among siblings it is possible to take into account additional unobserved characteristics which the siblings have in common and that also influence adult outcomes.

The World Health Organization estimates that globally 20% the urban children have blood lead levels exceeding 10µg/dL (Fewtrell et al.,2003); the

level above which the Center for Disease Control and Prevention (CDC) recommends that actions to reduce lead exposure should be initiated.[5] Naturally, the relevance of the current limit of concern hinges on the relative effects of lead exposure above and below the limit. Since the average blood lead levels of Swedish children at its peak in the early 1970s on average already were *lower* than 10µg/dL, the Swedish experience is particularly interesting since it provides a direct test of the relevance of the concurrent limit. The combination of population micro data, relatively low initial exposure levels and considerable differences in changes in exposure induced by government regulations provides a compelling setting to search for a threshold of the relationship between early childhood lead exposure and adult outcomes.

The main results suggest that low levels of lead exposure early in life have both statistically significant and economically important effects on future educational attainments and labor market outcomes. A key finding is a clear nonlinear relationship between local air lead levels in childhood and long-term outcomes at the relatively low levels of exposures considered. Above an estimated municipality average early childhood blood lead level of 5µg/dL, reductions in lead exposure have a consistently positive and significant impact on long-term outcomes. Below this level reductions no longer seems to affect adult outcomes in a consistent or significant direction. Importantly, the results are insensitive to a number of specification changes, such as the inclusion of family fixed effects, measures of other pollutants, measures of lead exposure later on in childhood, and various sample restrictions.

Further analysis reveals that children from poorer families seem to have benefited most from the gasoline lead reductions. Although data constraints prohibit a full differentiation of the mechanisms behind the socioeconomic status (SES) differences, a key finding is that residential segregation within municipalities (and thereby potentially differential neighborhood lead exposure levels) *does not* seem to be able to entirely explain the SES-gradient in the effects of lead. Instead differential avoidance behavior, differences in sensitivity to the same levels of exposure or differences in the ability to compensate for the effects of early lead exposure seems to be more plausible explanations for the SES-gradient. Whichever of these pathways that matters most, these results indicate that environmental policies may be able to reduce the intergenerational correlation in economic outcomes and

[5] In the US, approximately 310,000 children aged 1-5 years have higher blood lead levels than the level of concern (CDC, 2005), and average childhood blood lead levels in the adult US population will have decreased from 10µg/dL in 2002 to below 3µg/dL in 2018 (Reyes, 2007). The acceptable limit has been revised downwards several times since the 1970 level of 60µg/dL as a result of increasing evidence of an association between lower lead levels and health.

potentially function as a redistributive instrument, since it seems to disproportionally improve long-term outcomes among low SES children.

The remainder of the paper is structured as follows: section 2 gives a brief summary of previous studies linking childhood lead exposure to adult outcomes. Section 3 describes the data. In section 4 the empirical strategy is explained and section 5 presents the results. Sections 6 and 7 discuss the policy implications and conclude.

2 Early childhood lead exposure and adult outcomes

Exposure to lead has previously been linked to a number of adverse effects on health. Prospective cohort and cross-sectional studies of children have demonstrated associations of lead exposure, measured by various indices, and cognitive skills. In a series of meta-analyses, using data from some of the cross-sectional studies of school-age children (Skerfving and Bergdahl, 2007), it was concluded, that a decrease of one (1) IQ point was seen for every 2-4μg/dL increase in concurrent blood-lead levels (B-Pb).

There are however good reasons to suspect that lead exposure *in utero* or in early childhood could have a stronger effect compared to the effect of blood lead levels later on. First, the developing nervous system is more vulnerable to the toxic substances than the mature brain (Dobbing 1968; Schwartz, 1994; Lidsky and Schneider, 2003). Secondly, this sensitive period in human development coincides with a period of particularly high uptake of lead. B-Pb levels typically follow an inverted u-shaped pattern between ages 6 and 60 months, reaching its peak around age 24 months due to the intense hand-mouth activity common at these ages (Canfield et al., 2003). In a recent study a 10μg/dL decrease in B-Pb was estimated to increased cognitive ability at age 3 by 7.4 IQ points (cf. Lanphear et al., 2005).

Besides the effects on cognitive development, an association between early lead exposure and anti-social behavior has also been found. For example, using time-series data from early to late 20th century Nevine (2000) find that the consumption of lead in the general population in the first year of life co-varies with teenage pregnancy (18 years later) and crime rates (20 years later). Finally, lead exposure is also associated with poorer pre- and postnatal growth, hearing impairment, reduced effectiveness of the kidneys, and lower skeletal growth among children.[6]

However, all of these estimates stem from observational studies, and while many of the studies try to account for important potential confounders e.g. maternal education, home environment etc., it is important to realize that

[6] Bellinger (2004b) provides a thorough review of the literature on the association between childhood lead exposure and childhood outcomes.

unless *all* factors correlated with both lead exposure and e.g. cognitive test scores are accounted for, the estimated impact of lead will be biased; most likely upwards.[7] [8] The bias is furthermore likely to become more important when studying the relationship between lower levels of exposure and less obvious non-clinical outcomes, such as cognitive development. While many randomized control studies on animals supports a causal link between lead exposure and cognitive ability, it is not evident that the results from such studies are easily generalized to human subjects.[9]

A notable exception is Reyes (2007) that addresses the omitted variables problem by focusing on the impact of exogenous state-year specific changes in gasoline lead levels in the US on state level violent crime rates around 20 years later. The panel data employed allow for controls of fixed unobserved state-specific characteristics correlated with both childhood lead exposure and crime. Reyes finds a strong relationship between state level lead exposure in early childhood and state level violent crime rates, suggesting that the sharp reduction in lead in gasoline following the Clean Air Act Amendments in the early 1970s could explain as much as 50% of the sharp drop in violent crime that occurred in the US during the 1990s.

Although compelling, Reyes' analysis suffers from the use of aggregated data since it is not known whether the individuals exposed in early childhood are actually still living in the state where they were born 20 years later when the outcomes (also measured at the state level) are realized. Since in the US between 25-40% of the children migrate from the state of birth before age 22, this is clearly a source of concern. Reyes attempts to account for inter-state migration rates, however, since it is not obvious how early childhood lead exposure affects migration propensities it is not clear to what extent correcting for general migration patterns solves this problem. Moreover, the mechanisms through which early childhood lead exposure affect crime remains unexplored in Reyes' study.

This study distinguishes itself from and complements Reyes study in at least two important ways. First and foremost this study focuses on children with blood lead levels *below* the concurrent 10μg/dL level of concern. The subjects in Reyes' study on violent crime were estimated to have a blood lead level between 10 and 20μg/dL. Hence, the results from this study are potentially more informative about the long-term effects of early childhood lead exposure at levels that are still common in the US and in many other countries today.

---

[7] The importance of omitted variable bias has lately been recognized also in the epidemiological literature (cf. Bellinger, 2004b).

[8] Similarly the main problem with using time-series data is that there are many things which possibly co-vary with both lead consumption during childhood and unsocial behavior later in life.

[9] For example they do not take avoidance behavior into account such as staying indoors on days with high pollution (see Neidell, 2004).

Secondly, an improvement of this study compared to previous work using aggregate data is that the data employed follow individuals rather than states/counties/cities across time, but still makes use of the plausibly exogenous changes in local air lead levels induced by government regulations. The comprehensive population micro data derived from administrative registers enable me to follow children from birth throughout school, and to examine their early experiences on the labor market virtually without any attrition, which is typically a common problem in prospective studies.

3 The data

3.1 Measuring lead exposure in childhood

The measure of local lead exposure levels used in this study has not previously been explored in the literature. With a bi-decennial interval since 1975 the Swedish Environmental protection agency has monitored heavy metal air pollution using a nationwide grid of moss (bryophytes) samples. The use of mosses as biomonitors of heavy metal pollution was developed in Sweden at the end of the 1960s in pioneering work by Rühling and Tyler (1968, 1969) and is by now well established.[10] On a national scale, the use of moss as air pollution monitors expanded to Norway and Finland in 1985, and since 1995 28 countries participate in a bi-decennial moss survey designed to study regional differences and time trends in heavy metal deposition using around 7,000 sample locations throughout Europe in each round.[11]

Moss is particularly suitable for biomonitoring of air pollution levels for several reasons. (1) The lack of roots implies that moss solely depend on surface absorption of pollution through precipitation or dry deposition of airborne particles.[12] (2) The absorption and retention of metals is high, and (3) it can be found in abundance in nearly all environments. (4) The annual

[10] See Onianwa (2001) for a recent and comprehensive review of this literature.
[11] The European biomonitoring program is described in greater detail in Rühling, ed., (1994).
[12] The close-set leaves of the carpet-forming moss species enable them to filter the air efficiently. The contact with the underlying mor layer and soil is negligible for most species, and the risk of contamination by metals from the substrate is thus insignificant. A non-negligible part of the lead deposition levels has its origin in other regions or even further away (Rühling and Tyler, 1973). While the analysis in this paper takes into account the fixed characteristics of localities (such as the yearly precipitation rate, distance from the contributing pollution sources in other parts of Europe etc.), it is still likely that parts of the variation in lead exposure are due to the phase-out of leaded gasoline in other parts of continental Europe. This implies that part of the effects provided here reflect the total impact of phasing-out lead from gasoline, not only in Sweden, but in other parts of Europe as well. For ease of exposition the total deposition level, that is the sum of local air lead levels and deposition from precipitation is simply denoted air pollution levels.

growth of the moss species included in the surveys is easily distinguishable and, since the transportation of metal between the yearly growth segments is minimal, it is possible to distinguish temporal patterns in pollution levels.

Biomonitors also have several advantages over regular pollution monitors; the main being its simplicity, accuracy and low cost which allows a large number of sites to be included in the surveys. In the Swedish moss survey samples from around 1,000 locations are collected. Additionally, unlike regular pollution monitors which often go in and out of operation as a response to prevailing changes in local pollution levels, the moss samples are collected all over Sweden using a systematic procedure. The sampling sites are chosen carefully; they should be located at least 300 m away from bigger roads and closed residential areas, or at least 100 m from smaller roads and single houses. At each site 5 to 10 subsamples are collected in an area of approximately 100 m^2. From each sampling site the growth over the last three years of all sub-samples is pooled and analyzed and hence reflects the average air lead level during the three years preceding the date of sampling.

This study focuses on the samples collected in 1975, 1980 and 1985, which reflects the average lead deposition levels during the years 1972-1974, 1977-1979, and 1982-1984. The selection of these years is made for two reasons. First, between these years the maximum allowed grams of lead per litre of gasoline decreased particularly sharply. Second, since the main outcomes focused on are educational attainments and labor market outcomes it is necessary to restrict the sample to those cohorts that have completed their compulsory education and for whom the exposure level in early childhood is known.

Although the principles for choosing the location of the specific sampling sites and how to collect the samples is well defined it should be made clear that the sampling locations are not always identical across the survey years.[13] Hence, in order to construct a measure of municipality lead exposure I follow a similar approach as Neidell (2004) and Neidell and Currie (2004): first I calculate the centroid of each municipality. Then I measure the distance between the sampling site and the center of the municipality. Finally I calculate a weighted average air lead exposure level using the lead levels at the five closest sampling points (i.e. altogether between 25 and 50 samples), with the inverse of the distance to the sampling point as weight. This is done for each time period and municipality.

Figures 2 and Figure 3 display the lead concentrations in the municipalities in 1975 and 1985 using this definition of exposure. Figure 4 displays a kernel density plot of the distribution of the municipality lead

[13] However, it should be remembered this is not unique to the moss biomonitoring of pollutants. Traditional pollution monitors also go in and out of operation. Presumably to a higher extent due to changes in pollution than in the case of moss biomonitors.

exposure levels as measured in 1975, 1980 and 1985. From these figures it is clear that entire lead exposure distribution shifted drastically in between the years. Similarly a *within* municipality comparison of the lead levels clearly display the tremendous differences across municipalities in the reduction of lead exposure that took place between 1975 and 1985 (Figure 5). These sharp within municipality differences in the reduction of early childhood lead exposure across the cohorts is a key feature of the main identification strategy in this study as discussed further below.

Three important questions regarding the local lead exposure definition should be addressed before proceeding with the empirical analysis. The first concerns the arbitrary choice of using the five nearest sampling sites to define municipality of birth lead exposure. To test the sensitivity of the analysis to this assumption I have also used the 3 nearest sample points instead. The differences between these definitions are small and they are highly correlated (corr. coeff.> 0.9). [14]

Secondly, to get an idea on how accurate the five nearest sample approach is in predicting the actual exposure level, I estimate the level of lead at each sampling point, as opposed to municipality, pretending as if the sampling point of interest was not there. That is, I estimate the air lead level at a given sampling point based on the air lead levels at the five nearest sampling points. I do this for all sampling points in the data, and then calculate the correlation between the actual and the estimated air lead levels. The correlation between these two measures is high (corr. coeff.=0.80), which clearly indicates that the pollution assignment method employed provides reasonably accurate predictions of actual air pollution levels, and suggests that it does not seem to be a major concern for the analysis.

[14] The results on adult outcomes when using the 3 nearest samples definition rather than the 5 nearest sample definition are qualitatively similar.

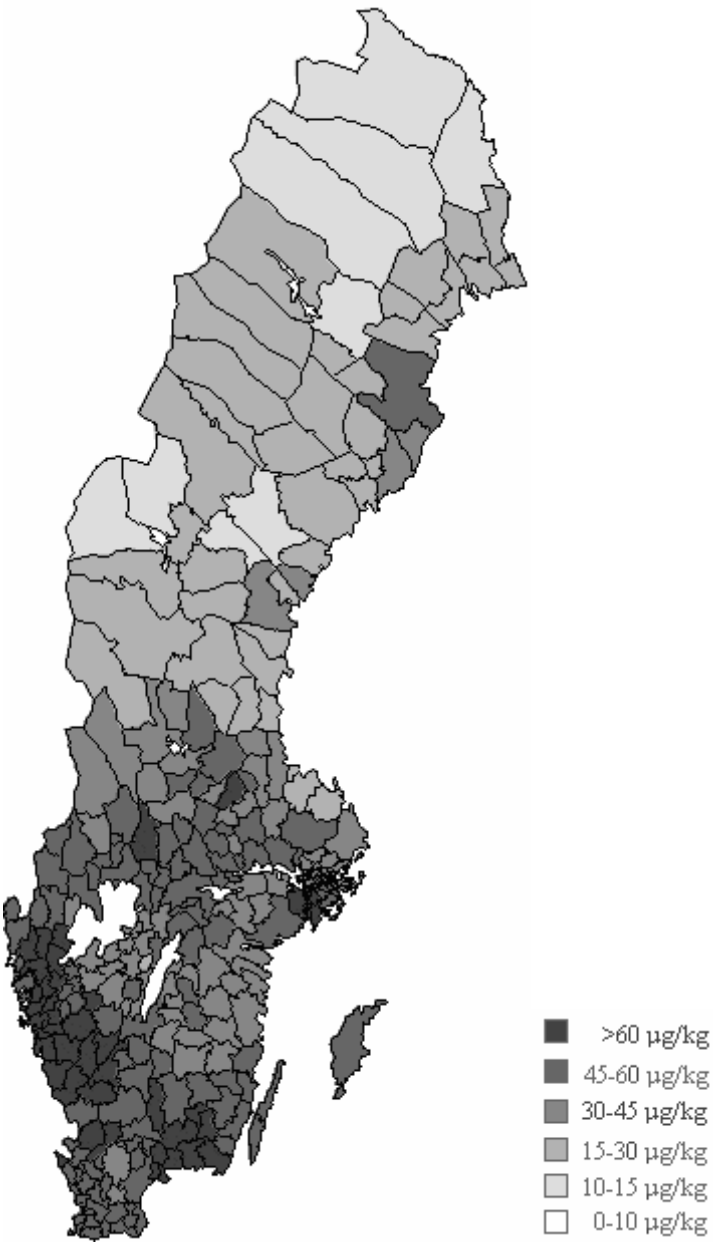

Figure 2 Moss lead levels (µg/Kg) in Swedish municipalities in 1975.Source: Authors calculation using data from the Swedish Environmental Research Institute (IVL).

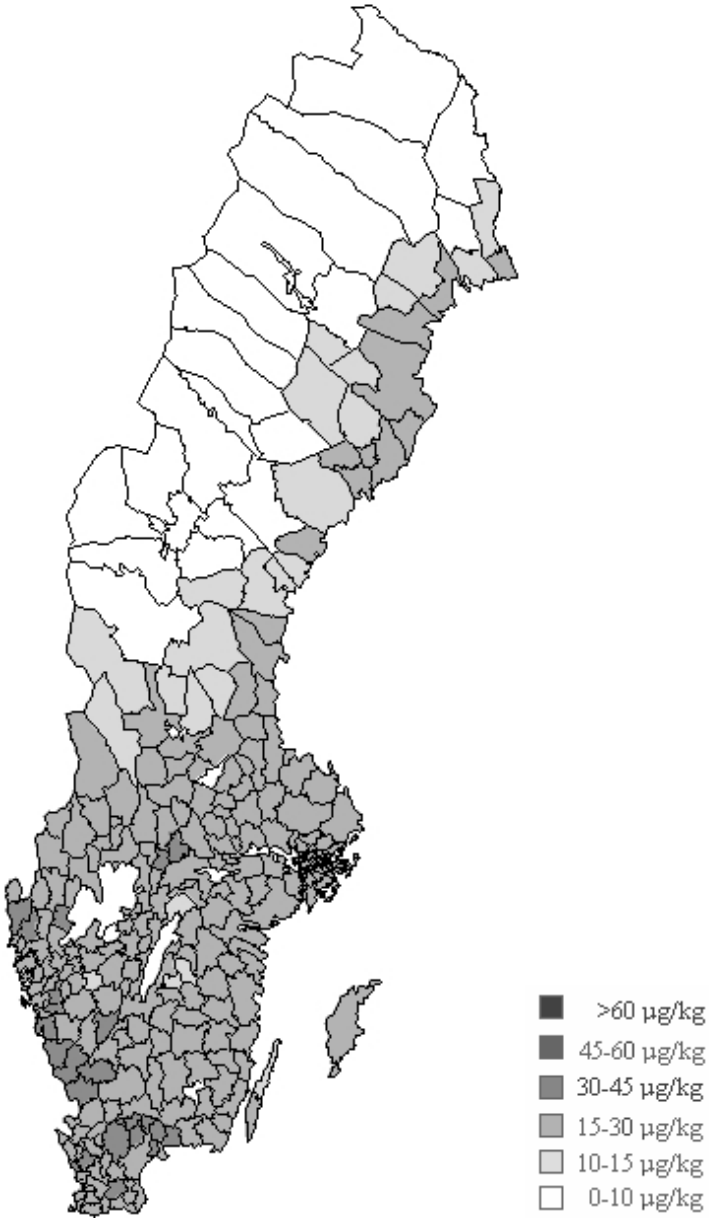

Figure 3 Moss lead levels (µg/Kg) in Swedish municipalities in 1985.Source: Authors calculation using data from the Swedish Environmental Research Institute (IVL).

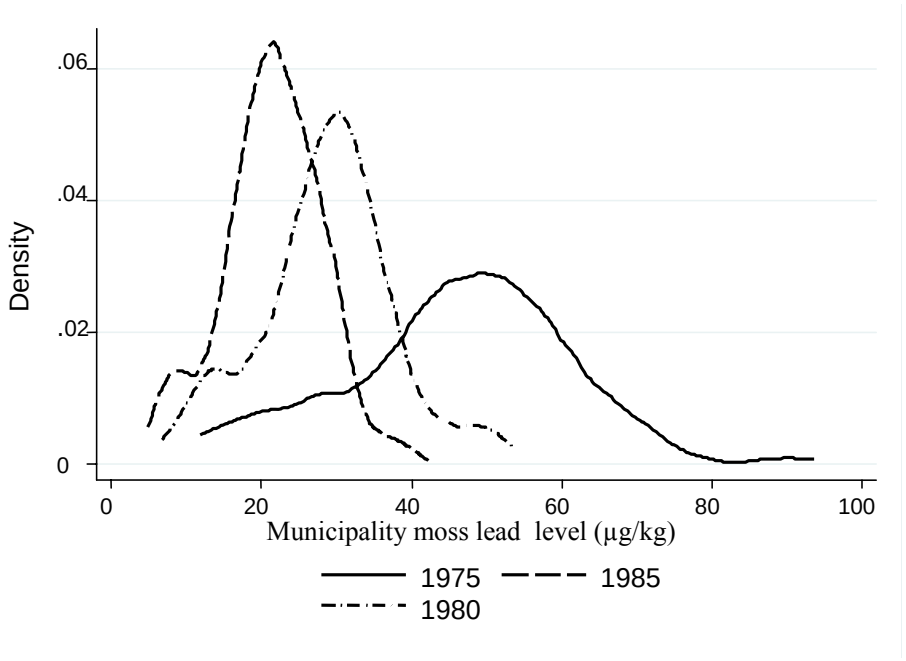

Figure 4 Kernel density distributions of moss lead levels in Swedish municipalities

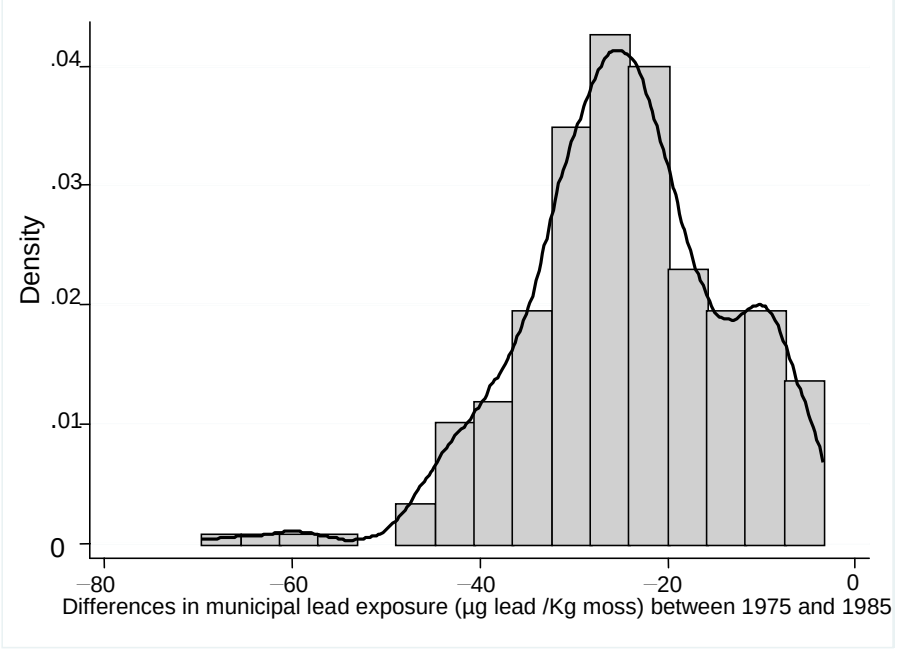

Figure 5 The distribution of within municipality lead level changes between 1975 and 1985

Finally, as in any study using data on local exposure levels rather than individual exposure an important question is how well the lead levels in moss predicts the actual blood lead levels in children. Unfortunately there exist no data that monitors the trends in blood lead levels among young children or the population in general in Sweden during this time period. However, since 1978 in two municipalities in southern Sweden, blood samples have been collected with a two year interval from about 120 primary school children (age 7-10) per annum. The results from these studies on the trends in childhood lead exposure are described in detail elsewhere (Strömberg et al., 1994, 2003). At the same time the department of environment (Miljöförvaltningen) in one of these municipalities (Landskrona) has at three time points (1984, 1995 and 2006) collected around 50 moss samples all over the municipality following the same procedure as the national monitoring program.[15]

Most previous studies using aggregate data on pollution have been forced to assume that local air pollution exposure is a valid proxy for actual exposure. However, the two datasets in Landskrona provide a unique opportunity to assess the strength of the relationship between local air lead exposure and children's lead exposure. Nilsson et al. (2009) do precisely this and link the average lead level of the five nearest moss samples to the children using their home addresses and estimate the elasticity between lead in moss and lead in children. Controlling for important individual characteristic, time and locality fixed effects they establish a Blood-Pb/Moss-Pb elasticity for the pre-gasoline lead free period (i.e. before 1995) of 0.44. This elasticity implies that a 10% reduction in Moss-Pb corresponds to a 4.4% decrease in primary school children's B-Pb. This estimate implies that the drop in air lead exposure between e.g. 1982 and 1994 can account for as much as 50% of the change in children's blood lead levels. Appendix A, gives a further review of the main findings in Nilsson et al. (2009).[16]

However, it is important to remember, as found in many previous studies, that the relationship between environmental lead exposure and very young children's blood lead levels is significantly higher. For example, Reyes (2007) finds that the elasticity between lead in gasoline and blood lead in children aged 0-6 is around 30% higher than among children aged 6-12,

[15] I am great full to Olle Nordell Landskrona miljöförvaltning for collecting and providing me with the data on lead in moss in Landskrona municipality. See also Nordell (2007) for (a Swedish) description of the sampling procedure and description of the moss data from Landskrona. To attain the moss lead levels which are comparable to those from the National survey it is necessary to calibrate the lead levels with a factor of 0.44 as described by Folkeson (1979), since they are measured in two different moss species.

[16] Assuming that this estimate functions as a valid proxy for the relationship between lead in moss and lead in children for the general population of children, in the last part of this paper I back out the elasticity between the adult outcomes focused on here and the children's blood lead levels.

which is important to remember later on when trying to estimate the relationship between the adult outcomes and early childhood blood lead levels.

3.2 Outcome measures

The individual outcome data stems from two data sources in the Educational database at the Institute for Labor Market Policy Evaluation (IFAU) in Uppsala.[17] In the main analysis I use all individuals born in Sweden in the three years prior to the year the moss samples were collected; that is all those born in 1972-1974, 1977-1979 and 1982-1984. Again the reason for the 1972 and 1984 constraint is that many of the individuals born after 1985 are less likely to have finished schooling in 2004 and that the first lead exposure measure available reflects the situation in 1972-1974 (i.e. mosses collected in 1975). As explained above the lead levels in the mosses measure the local lead deposition when they were between 0-3 years old. As discussed above, this age interval corresponds to a particularly sensitive period in human development and a period with particularly high lead uptake rates.

It is important to recognize that this assignment of exposure does not reflect an exact definition of timing of exposure for the cohorts. For example, taking the measure of air lead in moss literarily, for children born in June 1972 the lead exposure levels approximately reflect average lead exposure from the second trimester (starting January 1972) until about age 30 months (December 1974). For children born in June 1974, the moss lead exposure level reflects the exposure from conception until age 6 months. To check whether the results are sensitive to this deviation of exposure within cohorts, separate regressions including only the children born in the middle of each exposure measure period, i.e. those born in 1973, 1977 and 1983, where also tested, which yielded very similar results.[18] For these children the exposure levels reflect the average exposure level from conception until age 2. Finally, I focus on children who were living in Sweden in 2004, who have completed compulsory schooling (9 years education) and who were born in Sweden, so that their municipality of birth (and hence childhood lead exposure) is known.

The outcome variables considered are grade point averages in grade 9 (GPA at end of the 9 year compulsory school), whether the GPA was below (above) the 25%-tile (75%-tile) of the GPA distribution, the cognitive test score as measured for all men at military enrollment, whether the score was

[17] I am grateful to Björn Öckert for assembling the data and for sharing it with me.

[18] While the estimates of the parameters are essentially unchanged the precision also decreases since the sample is reduced by 2/3. These results are not reported but available upon request from the author.

below (above) the 25%-tile (75%-tile) of the cognitive test score distribution, whether having completed high school, ever enrolled in college education, the number of years of schooling completed, (ln) labor market earnings, welfare dependency and finally whether or not having become a teenage mother. The labor market and educational outcomes outcome variables are measured in 2004 (at ages 20-32). All of these outcomes have previously been shown to be predictive of other outcomes throughout life.

The military enrollment test scores are Stanine (Standard Nine) test scores which is similar to the AFQT in the US. The score is an evaluation of cognitive ability based on several subtests of logical, verbal and spatial abilities and a test of the draftees' technical understanding. The results on these subtests are combined to produce a general cognitive ability ranking on a 1-9 scale. All men were obliged by law to go through the military draft. However, due to reforms in the military enrollment procedures affecting the latest cohorts (i.e. those born during the 80s) the cognitive outcomes are only used for those born before 1980. Before that about 90 percent of all men in each cohort went through the draft procedure almost exclusively (99%) at age 18 or 19.[19] The test score is percentile ranked within each cohort of draftees to account for any minor changes in the tests over time.[20] Teenage motherhood is included as an outcome since it has previously been shown to be correlated with early childhood lead exposure using time-series data (see Nevin, 2000). Table B1 provides the definitions of the outcome and control variables and descriptive statistics for the outcome variables, individual and parental characteristics as well as some municipality of birth background characteristics.

4 Empirical method

4.1 Empirical model

As discussed above a number of factors complicate the estimation of causal effect of early childhood lead exposure on adult outcomes. Under the assumption that the effects of the covariates are additive and linear it is possible to remove the influences of many potential confounding factors by estimating a linear regression model that accounts for unobserved differences in municipalities and cohorts,

[19] In principle only the physically and mentally handicapped was exempted.
[20] The test has been subject to evaluation by psychologists and appears to be a good measure of general intelligence (Carlstedt, 2000).

$$y_{ijtc} = \alpha + \beta_1(\text{Lead exposure})_{jc} + X_{ijtc}'\beta_x + \gamma_t + \phi_j + \varepsilon_{ijtc} \quad (1)$$

where *y* is either a continuous measure or an indicator variable of the adult outcome of individual *i*, born in municipality *j* in year *t* and belonging to cohort *c* where $c \in \{1975,1980,1985\}$. Lead exposure is the continuous early childhood lead exposure measure (µg/kg moss) as described above; *X* is a vector children's own, parental and municipality of birth characteristics. They are indicators for child sex, month of birth, number of siblings, year of compulsory school graduation, maternal educational attainments (7 levels), maternal age at birth, indicators for parental earnings (quartiles) for sum of parental earnings in 1990, the average income of the parents in the municipality, share of parents that have completed high school/ university, the share with missing paternal indicator, the share of boys in the same cohort, cohort size, and the average family size. Finally γ_t and ϕ_j are nine year of birth and 287 municipality of birth specific effects respectively. ε_{ijtc} is the error term.

The inclusion of *X* controls for many of the important background characteristics that varies across cohorts and municipalities and the municipality of birth specific fixed effects ϕ_j accounts for persistent differences between municipalities that could be correlated with the children's future outcomes and childhood lead exposure. The month of birth dummies is included since both adult outcomes, but potentially also early childhood lead exposure can be influence by the season of birth. The nine year of birth dummies γ_t control for all general trends in the outcomes of interest.

The main parameter of interest is β_1 and the main hypothesis to test is whether $\beta_1 = 0$, that is if early childhood lead exposure has no effect on adult outcomes. Under the identifying assumption that the error term is uncorrelated with the lead exposure, $\hat{\beta}_1^{OLS}$ reflects the causal impact of the local air lead level (an additional gram of lead per Kg moss) has on subsequent adult outcomes. That is after conditioning on individual, parental, observable and fixed unobservable municipality characteristics, the main identifying assumption requires that there are no unobserved characteristic that are correlated with both childhood lead exposure and adult outcomes.

4.2 An indirect test of the main identifying assumption

Although this identifying assumption is fundamentally untestable, it is possible to indirectly assess the plausibility of this assumption by looking at the correlation between factors that are expected to be correlated with adult outcomes of the child but not with the child's lead exposure if the identifying assumption is valid. It is particularly informing to contrast this correlation in a traditional cross-sectional analysis with the within-municipality analysis, since this may reveal how well the within municipality analysis can reduce the potential bias induced by omitted variables.

Candidate factors qualifying for such a test directly available in the data are predetermined parental characteristics; such as parents educational attainments. These parental characteristics can be considered to be predetermined in the present context since the lion share of the parents (>95 percent) were born before 1960, i.e. before environmental lead exposure became a serious environmental problem in Sweden.[21] Therefore assessing whether predetermined *parental characteristics* are correlated with municipality lead levels during their *children's early childhood* should give a hint of whether omitted variables (associated with parents characteristics) is a major concern in the within municipality analysis.

The first panel of Table 2 first provides estimates of β_1 from a regression of the parents' educational attainments (or earnings) on their children's early childhood lead exposure using data on all cohorts but without controlling for municipality fixed effects.[22] The columns present the estimated impact on whether at least one of the parents had completed high school, university, the total parental earnings and finally the same outcomes for the mother and father separately, and an indicator for if the father is not known/missing. The model only controls for year of birth and cohort size of the child. In this cross-sectional analysis many of the parental predetermined characteristics are statistically significant and generally indicate that poorer educational and

[21] Using mosses collected from 1860 until 1968 Rühling and Taylor (1968) show that in the southern part of Sweden (the most highly exposed in the present sample) the increase in lead concentrations in moss were restricted to two distinct periods: a first increase towards the end of the nineteenth century, and a second increase during the 1960s (80-90 µg/kg in 1968). Before that the average lead level in Skåne (the southernmost regions in Sweden with the highest lead level in the data used in this study) was around 40-50 µg/kg moss. They conclude that the first rise is probably due to industrial pollution, possibly due to the increase use of coal, and that the second rise is *more than likely* caused by the rapid increased use of lead gasoline. The exact same pattern is found in a study by Rehnberg et al. (2000) who use extraordinary data on lead levels in lake sediments to examine regional trends in lead depositions in Sweden over a period of *4,000 years*. In particular the lead concentrations in the lake sediments increased by 50% between 1960 and the peak year of 1970. Hence the parents of the children were exposed to relatively low levels during their own childhood and therefore the lead levels in during the parents childhood is not expected to be able to influence the children's adult outcomes to any large extent.

[22] The parent's outcomes are measured in 1990 when the average mother was 40 years old.

labor market outcomes of the parents are strongly correlated with their children's childhood lead exposure levels.

In the second panel of Table 2 the same set of estimates is presented when only using the within municipality variation in childhood lead exposure. After controlling for municipality specific effects, for the majority of the outcomes, the magnitude of the relationship between lead and predetermined parental characteristics decreases typically by at least an order of magnitude, sometimes changes sign and are no longer statistically significant. The exception is father's high school completion which switches sign compared to the cross-sectional analysis and now indicates that higher exposure is correlated with higher probability of having a father that has completed high school education. However, it is only marginally statistically significant, and given the number of outcomes considered it is not surprising that at least one coefficient is significant at the 10% level.

This exercise highlights the problems with using cross-sectional research designs to make causal inferences. It furthermore provides supports for the validity of the main identifying assumption since the within municipality analysis seems to be able to reduce the importance of observed and hence also most likely unobserved omitted variables considerably.

Table 2 Parents predetermined characteristics and their childrens' lead exposure

PANEL A:	Parents Earnings	Parents High school	Parent College	Mom's College	Mom's High school	Father College	Father High School	Father earnings	Young mother
Lead exposure (µg/Kg)	.0004	-.0011**	-.00035	-.00023	-.0011***	-.0002	-.0009**	-.0001	-.0005*
	(.0005)	(.00029)	(.0004)	(.0003)	(.0003)	(.0004)	(.0004)	(.0004)	(.0003)
R-squared	0.02	0.01	0.02	0.01	0.01	0.03	0.02	0.02	0.03
Municipality fixed effects?	No	No	No	No	No	No	No	No	No
PANEL B:	Parents Earnings	Parents High school	Parent College	Mom's College	Mom's High school	Father College	Father High School	Father earnings	Young mother
Lead exposure (µg/Kg)	.0002	.00012	-.00003	0.00001	.00012	-.00005	.00046*	.00008	-.00017
	(.0004)	(.00025)	(.00029)	(.0002)	(.00025)	(.0002)	(.00025)	(.0003)	(.0002)
R-squared	0.04	0.03	0.06	0.06	0.02	0.05	0.03	0.04	0.03
Municipality fixed effects?	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
# observations	797,889	797,889	797,889	797,889	797,889	768,644	768,644	768,644	797,889

Notes: Standard errors are clustered at the municipality level. Controls for year of birth of the child and cohort size.

5 Results

In the following sections I look at the impact of childhood lead exposure levels on future outcomes. To preview the central results, in the baseline specification early childhood lead exposure have a negative impact on virtually all future outcomes considered. The number of years of schooling, having a low GPA at the end of the compulsory school, high school graduation, and being on welfare are all statistically significantly correlated with early childhood lead exposure. The estimated impact on the remainder of the outcomes is too imprecise to draw definite conclusions. However, further analysis reveals that the poor precision of the baseline estimates seems to be due to that the relationship with long-term outcomes are nonlinear. Reductions in lead exposure from high initial levels have consistently significant effects on virtually all of the outcomes, but similarly sized reductions from initially low levels of exposure only yield inconsistent and insignificant effects on the outcomes considered. A number of specification checks reveals that the estimated effects of exposure reduction from the highest levels is robust and that children from disadvantaged families seem to have benefited most from the reductions in lead exposure.

5.1 Baseline results

5.1.1 Cross-sectional estimates

Before proceeding with the main fixed effects analysis it is useful to replicate the results from a conventional cross-sectional analysis. For each cross section (1975, 1980, 1985) the results from estimations of equation (1) (but without the municipality fixed effects) on all outcomes considered in the main analysis is presented in Column (1)-(3) of appendix Table B2. These cross-sectional estimates in general points in the expected direction, although there is considerable variability in the magnitude of the estimates both within a given year for different outcomes but also across years for a given outcome. The estimates are furthermore only occasionally statistically significant at conventional significance levels. After pooling the data (column 4), except for earnings, all the estimates indicated that reduction in early lead exposure improves long-term outcomes.

If considering the statistically significant estimates from the pooled cross-sectional model it seems as if lead exposure particularly impairs development among children in the lower tail of the ability distribution. Both the risk of ending up in the lower quartile of the GPA and IQ-test score distribution are significantly affected, although only at the 10% significance

level. The estimated effects on these outcomes suggest that the average reduction in air lead exposure (60%) that occurred between the early 1970's and early 1980s reduced the incidence of ending up in the lower tail of the grade and IQ distribution by around 1 percentage point or by about 4%. Although, as shown above in Table 2, these cross-sectional estimates are likely biased by unobserved characteristics correlated with both high levels of lead exposure and the adult outcomes of the children. Overall the cross-sectional results in Table B2 provide little evidence of a significant relationship between adult outcomes and early childhood lead exposure. However, as will become clear, the pattern with stronger effects in the lower part of the ability distribution remains throughout the empirical analysis, even after unobserved heterogeneity has been taken into account.

5.1.2 Municipality of birth fixed effects

Next the analysis proceeds by focusing on the fixed effects estimates which under the present conditions potentially give a more accurate picture of the relationship of interest. The first column in Table 3 presents the results from the estimates of equation (1) for the percentile ranked GPA, now including the municipality of birth fixed effects. The estimate for GPA presented in column (1) implies that when the average lead exposure during early childhood increases with 1 μg/kg the grade point average decreases with 0.017 percentiles. Similarly, the probability of ending up in the lower quartile of the grade distribution increases with 0.024 percentage points per 1μg/kg increase in lead exposure. For males the average IQ level also decreases with 0.010 percentiles. An inverse relationship between lead exposure and the probability of ending up in the higher end of the grade distribution is also found. The probabilities of ending up in the top or lower part of the IQ distribution are also affected as expected. Table 4 present the estimated impact on educational attainments, early labor market and social outcomes. Again all point estimates suggest that higher levels of lead exposure are detrimental for subsequent outcomes.

However, in general the precision of the estimates presented in Tables 3 and 4 is poor. Only the probability of ending up in the lower tail of the grade distribution, high school completion rates, the number of years of schooling and the welfare dependency rates are significant at conventional significance levels. At first examination the estimated effects may seem small but it is important to recall that these reduced form estimates imply that the reductions in lead exposure during the observation period implies that the probability of ending up in the lower end of the GPA distribution decreased by 3.3 percent, increased high school completion increased by 0.9 percent, years of schooling completed in 2004 increased by 0.05 years and the prob-

Table 3 Grade point averages and cognitive test scores

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	ALL	ALL	ALL	Males	Males	Males
Lead exposure (µg/Kg)	-.0171	.00024*	-.00017	-.0109	.00016	.00002
	(.0104)	(.00014)	(.00013)	(.0135)	(.00019)	(.00018)
R-squared	0.22	0.12	0.13	0.17	0.09	0.1
Mean of dep. var.	50	0.25	0.25	50	0.25	0.25
Individual characteristics	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
Fixed municipality	yes	yes	yes	yes	yes	yes
Mean of dependent variable	50	0.25	0.25	50	0.25	0.25
Observations	797,889	797,889	797,889	262,283	262,283	262,283

Notes: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. In addition the estimated model includes controls for parental characteristics, and municipality characteristics (see section 4.1 for details). Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

Table 4 Alternative long-run outcomes

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare	Teen mother
Sample	ALL	ALL	ALL	ALL	ALL	Women
Lead exposure (µg/Kg)	-.00022**	-.00029	-.00142*	-.00021	.00016***	0.00001
	(.00010)	(.00020)	(.00084)	(.00036)	(.00005)	(.00005)
R-squared	0.06	0.18	0.2032	0.1301	0.03	0.03
Mean dep. var.	0.89	0.33	12.7	176,400	0.04	0.04
Individual characteristics	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
Fixed municipality	yes	yes	yes	yes	yes	yes
Observations	797,889	797,889	797,889	718,843	797,889	387,576

Notes: In addition the estimated model includes controls for parental characteristics, and municipality characteristics (see section 4.1 for details). Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

ability of welfare dependency in 2004 decreased by 0.6 percentage points. Again, the effects seem to be stronger in the lower tail of the ability/skill distribution.

5.2 Nonlinear effects in lead exposure

Since most neurotoxins follow a hockey stick shaped effect, with a much lower marginal effect below some threshold, one potentially important reason for the relatively poor precision of the estimates in Table 3 and 4 could be that the relationship is nonlinear or discontinuous at the levels of exposure considered. As discussed above identifying the threshold of such nonlinear effects are, of course, highly interesting from a public policy perspective. However, neither the biological nor the epidemiological literature provides a strong theory and only very limited evidence that could give any guidance in the search for a threshold when it concerns lead (c.f. Needleman, 2004). Indeed most studies have failed to identify a lower threshold for effects on cognitive skills, although an important reason is presumably that the sample sizes at the lowest exposure levels have been relatively small, and that confounding most likely becomes even more acute when studying the sub-clinical effects of low exposure levels.

In order to examine the presence and influence of nonlinearities in this case the same model as in equation (1) is estimated, but now the single continuous linear lead exposure measure is replaced by linear splines with breakpoints at each quartile of lead exposure. This setup mimics the approach taken by Reyes (2007), who find no/only weak nonlinearities in the lead exposure-violent crime relationship. However, again the average blood lead levels in her sample were considerably higher than in this context. By using splines it is possible to examine if the effect of a similar sized reduction in lead exposure within the different quartiles of exposure has heterogeneous impacts on adult outcomes.

Table 5 presents the results from this specification for the GPA and the cognitive test scores, and Table 6 for the other outcomes. In contrast to the analysis using a single linear measure of exposure, the estimates based on changes within the different quartiles of exposure show a strikingly consistent pattern. A 1 µg/kg reduction in moss lead in the municipality of birth in early childhood has a highly significant and consistent adverse effect on basically all outcomes considered; but only within the highest quartile of exposure (i.e. >48µg/kg). Below this level similarly sized reductions in lead exposure has inconsistent and generally insignificant effects on long-term outcomes.

Table 5 Nonlinear effects of early childhood lead exposure: GPA and cognitive test scores

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	ALL	ALL	ALL	Males	Males	Males
Lead in 1st quartile	-.0463	.00006	-.0008	-.0166	-.0010	-.0010
	(.0426)	(.0005)	(.0006)	(.0529)	(.0006)	(.0008)
Lead in 2nd quartile	-.0421	.00024	-.0010*	-.0269	.0013**	.0002
	(.0452)	(.00059)	(.0006)	(.0404)	(.0006)	(.0005)
Lead in 3rd quartile	.0128	-.00008	.00015	.0216	-.0003	.00045*
	(.0301)	(.00041)	(.0003)	(.0175)	(.0003)	(.00025)
Lead in 4th quartile	-.0350**	.00046***	-.00025	-.0283***	.00025	-.0003**
	(.0136)	(.00018)	(.0002)	(.0106)	(.00016)	(.0001)
R-squared	0.22	0.12	0.13	0.17	0.09	0.10
Mean of dep. var.	50	0.25	0.25	50	0.25	0.25
Individual characteristics	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
Fixed municipality	yes	yes	yes	yes	yes	yes
Observations	797,889	797,889	797,889	262,283	262,283	262,283

Notes: The coefficients shown reflect the average effect of a 1µg/kg increase within each quartile. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels. The sample for the IQ test scores are reduced and only include children born before 1980 in order to reduce the impact of changes in the enrollment procedures for men born after 1980.

Table 6 Nonlinear effects of early childhood lead exposure: Alternative long-run outcomes

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare	Teen mother
Sample	ALL	ALL	ALL	ALL	ALL	Women
Lead in 1st quartile	-.0002	-.0006	-.0043	-.0004	-.00039	.00015
	(.0005)	(.0011)	(.0039)	(.0020)	(.0003)	(.00025)
Lead in 2nd quartile	.0004	-.0005	.0003	.0044**	.00035	.00023
	(.0005)	(.0011)	(.0040)	(.0021)	(.00025)	(.00027)
Lead in 3rd quartile	-.0003	-.0001	-.0001	.0004	.00004	-.00025*
	(.0003)	(.0005)	(.0023)	(.0007)	(.00013)	(.00014)
Lead in 4th quartile	-.00026**	-.0005	-.0027**	-.0016***	.00019***	.00017*
	(.00013)	(.0003)	(.0013)	(.0005)	(.00006)	(.00009)
R-squared	0.06	0.18	0.20	0.1302	0.03	0.03
Mean of dep. var.	0.89	0.33	12.7	176,400	0.04	0.04
Individual characteristics	yes	yes	yes	Yes	yes	yes
Year of birth	yes	yes	yes	Yes	yes	yes
Fixed municipality	yes	yes	yes	Yes	yes	yes
Observations	797,889	797,889	797,889	718,843	797,889	387,576

Notes: The coefficients shown reflect the average effect of a 1μg/kg increase within each quartile. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

This analysis reveals a clear nonlinear effect of reduction in early childhood lead exposure on long-run outcomes. Since the average blood-lead level in the cohorts considered already initially were lower than the limit of concern, these results also suggest that if anything the threshold of the adverse effects of lead exposure on the long-term outcomes lie at a level that is significantly lower than a blood lead level of 10µg/dL.[23] In section 6 I use the elasticity provided by Nilsson et al. (2009), to estimate the childhood blood lead level corresponding to the moss lead level found in the analysis in this section.[24]

5.3 Robustness checks

Next, since the previous estimates primarily show an effect at the highest quartile of lead exposure, in order to increase precision, the remainder of the analysis concentrates on children born in municipalities with a moss lead level above the 1st quartile of initial exposure (>37 µg/kg in 1975). This

[23] The earliest systematic blood lead levels sampled among children in Sweden was conducted in 1978 by Strömberg et al. (1995). At that time the blood lead level was just below 6 µg/dL on average in two locations in southern Sweden (where the air lead levels were among the highest in Sweden at the time). Needleman report blood lead levels in the US in the same year of on average around 14 µg/dL. Before this point in time in order to assess the lead levels of the children I use the estimates of previous work by Reyes (2007). She finds that an increase of 1 gram lead per gallon of gasoline increases blood lead level with approximately 3.3 µg/dL in the general population. Taking her baseline specification literally (B-Pb=9.316+3.325*GRAMSLEAD/GALLON) and combining it with the data in Table 1 suggest that at the year of birth of the first cohort born in 1972-74 the average blood lead level would have been on average 6µg/dL. A second way to assess the initial blood lead levels is to use the model developed in Nilsson et al. (2009) and use the average moss lead level in 1975 to predict the blood lead levels in primary school children at that time. This approach provides a predicted initial blood lead level of around 3µg/dL in primary school children. After adjusting the blood lead moss lead elasticity using the age specific blood lead blood gasoline elasticity estimated by Reyes (2007) (30% higher for children aged 0-6 than for children aged 6-12) and under the additional assumption that the additative separable specification used in the estimation hold for both populations, the relevant blood lead level in for children aged 0-6 would on average correspond to about 5µg/dL. A third way to estimate the initial blood lead level is to use the estimates in Strömberg et al., who based on repeated blood lead measurements find that individual blood lead levels in primary school children decreases on average by around 6% per year. Given the average level in 1978 this would imply that the average blood lead levels among 1-4 year olds in 1972 (since the samples are taken from children aged 7-10 in 1978) would be on average 8.5µg/dL. Since these samples were taken in a region with the highest lead exposure (based on the moss lead values) it seems reasonable to assume that this level represent a higher bound of blood lead levels in the general population of children in these cohorts. Hence all three approaches provide estimates that suggest that initial blood lead levels were below 10µg/dL for the cohorts born between 1972 and 1974.

[24] One concern with this analysis is that the apparent nonlinear effect could be due to that the precision of the estimates are poor at the lower quartiles of exposure simply because the changes in exposure within these quartiles are not large enough. However, the pattern in Table (6) and (7) is if anything reinforced if the children growing up in municipalities with the least (<10 %-tile) changes in exposure between 1975 and 1985 are excluded.

decreases the number of municipalities included in the analysis to 210 and the number of children to 670,000.

To make sure that excluding the lowest initial level municipalities in this way does not introduce any major bias it is informative to compare the baseline model estimates for the reduced sample with the baseline linear spline estimates. Column (1) of Table B4 in appendix B report the estimates from the original specification in equation (1) estimated on the reduced sample (Tables B2 and B3 report the full results). As expected, these estimates are higher and more precisely estimated compared to the baseline OLS estimates displayed in Tables 3 and 4, and are reasonably similar to the estimates for reductions in the highest quartile of exposure in Tables 5 and 6. For further comparison, in Table B4 I have also included the estimated effect when children in the highest quartile of initial exposure are excluded (column 3). After excluding the highest quartile of initial exposure the estimated parameters in virtually all cases are insignificant and when they are significant (earnings, teen pregnancy) they are always pointing in the unexpected direction. By comparing the results in columns (1) and (3) in Table B4 it again becomes clear that the initial level of exposure is important and that the relationship between early childhood lead exposure and long-term outcomes seem to be nonlinear. Columns (2) and (4) of Table B4 also report estimates after splitting the sample based on *changes* in exposure between 1975 and 1985. The resemblance between the high initial and high changes municipality estimates is striking.

Table 7 continues the analysis by testing the sensitivity of the reduced sample estimates. First, for ease of comparison, column (1) reviews the baseline estimates for the children under risk of being affected. In column (2) family fixed effects estimates are reported. This model accounts both for the fixed characteristics of the biological parents and the municipality of birth. The family identifier used is a combination of the unique mother and father identifiers and hence in the analysis the comparison is made between full biological siblings only (in total 123,324 families).

Table 7 Robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Specification/ Sample:	Baseline	+ Family fixed effects	+ Linear time trends	+ cadmium exposure	Log exposure	Excl. 3 largest cities	+ Child-care enroll.	Municipality level data
EXPOSURE:	*Lead*	*Lead*	*Lead*	*Lead*	*ln (lead)*	*Lead*	*Lead*	*Lead*
OUTCOMES:								
GPA	-.0356**	-.0175*	-.0294	-.0453***	-1.290**	-.0246**	-.0356**	-.0289**
	(.0143)	(.0102)	(.0218)	(.0172)	(.5575)	(.0125)	(.0145)	(.0148)
Low GPA	.0006***	.0004**	.00034	.0006***	.0162**	.0004***	.0006***	.00045**
	(.0002)	(.0002)	(.0003)	(.0002)	(.0074)	(.0002)	(.0002)	(.0002)
High GPA	-.0003	-.00014	-.00043*	-.00044**	-.0154**	-.00018	-.00032*	-.00025
	(.0002)	(.00018)	(.00025)	(.0002)	(.0071)	(.00018)	(.00018)	(.00018)
IQ (Men)	-.0336***	-.0664**	-	-.0403***	-1.129***	-.0352***	-.0349***	-.0272**
	(.0128)	(.0290)		(.0105)	(.4004)	(.0106)	(.0092)	(.0128)
Low IQ (Men)	.0003**	.0009*	-	.00044**	.0141**	.00034**	.0003**	.00031
	(.00015)	(.0004)		(.00015)	(.0058)	(.00015)	(.00015)	(.00025)
High IQ (Men)	-.00026**	-.0009*	-	-.0003*	-.0064	-.00024*	-.00028**	-.00031*
	(.00012)	(.0005)		(.00015)	(.0059)	(.0001)	(.00012)	(.00017)

Table 7 CONT'D

High school	-.00033*	-.0003**	-.0002	-.0003	-.0066	-.0001	-.0002	-.0002
	(.00017)	(.00014)	(.00028)	(.0002)	(.0067)	(.00015)	(.00014)	(.00015)
University	-.0002	-.0003	.0002	-.00033	-.0071	-.00024	-.0002	-.0002
	(.0002)	(.00019)	(.0003)	(.00028)	(.0093)	(.00028)	(.0002)	(.0002)
Years of schooling	-.0022*	-.0019**	-.0007	-.0022*	-.0644	-.0015	-.0015	-.0009
	(.0012)	(.0008)	(.0015)	(.0013)	(.0447)	(.0012)	(.0010)	(.0008)
Welfare	.00012	.00017**	.00004	.00005	.0017	.0002***	.0001	.00011
	(.0001)	(.00008)	(.0001)	(.0001)	(.0035)	(.00006)	(.0001)	(.00008)
Earnings	-.0009**	-.0011**	.0012**	-.0015**	-.0123	-.0009*	-.0007*	-.0007*
	(.00045)	(.0005)	(.0005)	(.0007)	(.0237)	(.0005)	(.0004)	(.0004)
Teenage mom	.0001	.0005**	.00013	.00014*	.0038	.0001	.0001	.00007
	(.0001)	(.0002)	(.0001)	(.00008)	(.0034)	(.0001)	(.0001)	(.00005)

Notes: Each row and column represent a separate regression. Column (2) presents the results estimates from a family fixed effects model; column (3) introduces municipality of birth specific time trends; column (4) replaces the linear exposure measure with log lead exposure, column (5) excludes the 3 largest cities; column (6) checks to what extent the estimated baseline effect of exposure to lead is confounded by effects of changes in other pollutants; column (7) adds controls for public childcare enrollment; finally column (8) reports estimates from a model where the individual data have been aggregated to the municipality level. In addition all specifications includes controls for municipality, cohort fixed effects and individual and parental controls. The reported estimates is the marginal effect of one (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood, except in column (4) which report estimates for a lin-log specification. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two time periods and hence adjust standard errors at the time period-municipality level). The municipality level regressions (column (7)) are weighted by the number of children in each municipality/period cell. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects. The baseline sample estimates refers to the linear estimates after excluding the municipalities below the 1st quartile of initial (1975) lead exposure level (full results in appendix B). In the family fixed effect model only families with less than four children are included.

The municipality fixed effect in this model is identified by families that report differing municipality of birth for their children.[25] After including family fixed effects, the effect of childhood lead exposure is identified by differences in exposure between siblings. As the estimates in column (2) show, conditioning on parental fixed effects in addition to the municipality fixed effects produces estimates that are similar to the baseline estimates, but for cognitive test scores the point estimates are larger in absolute terms. The high similarity between the sibling fixed effects results and the main results are striking, and provides further evidence for the validity of the estimated relationship.

In column (3) the baseline specification is augmented with municipality of birth specific linear time trends. This model addresses the concern that the main effects could partly result from trends in outcomes at the local municipal level. After introducing these time trends, the majority of the point estimates decreases somewhat. For earnings the point estimate changes sign. At the same time the precision of almost all estimates decreases significantly. However, the sharply decreased precision of the estimates after controlling for municipality linear time trends should come as no surprise. The parameter of interest in this specification is identified using only the residual variation of each municipality around its own time trend. Since the effective panel only stretches over three years (1975, 1980, 1985), this specification is likely to reduce the signal-to-noise ratio considerably and increase attenuation bias.

When trying to identify the effects of a particular polluting element it is important to address the concern that any association found between lead exposure in childhood and outcomes later in life in principle could be due to that the observed air lead levels simply proxy for other unobserved pollutants correlated with lead. If higher (unobserved) pollution also leads to poorer subsequent outcomes, this may bias the estimated impact of lead upwards. The focus on the changes in air lead levels induced by government regulations targeting gasoline lead levels in particular should mitigate much of this problem. Still, since the moss sample data also hold information on seven of the other most common heavy metal pollutants (As, Cd, Cr, Cu, Ni, V, Zn) it is easy to do an initial assessment of the potential severity of this problem.[26] Of all the observable environmental pollutants in the data, the only other air pollutant which displays even nearly as large and widespread changes during the observation period as lead does is cadmium (Cd).

Cadmium has previously been found to be associated with adverse health outcomes (kidney damage, bone disease). Early exposure to Cd has been

[25] In 19.8% of the two-child families the siblings have differing municipalities of birth.
[26] This is the data contained from the start of the moss survey. From 1985 the Iron (Fe) and mercury (Hg) levels also started to be assessed.

shown to be able to produce neurotoxic effects in laboratory experiments (Anderson et al., 1997; Peterson et al., 2004), and in a recent study cadmium air releases are shown to affect infant health in humans (Currie and Schmeider, 2009). Moreover, air cadmium and air lead levels display a fairly high correlation at the municipality level; both in the cross-section (corr. coeff.=0.8) and in changes between 1975 and 1985 (corr. coeff.=0.5). Hence, changes in air Cd levels could potentially at least partly explain the estimated relationship between lead and subsequent adult outcomes. However, in this context it is not likely that the baseline estimates for lead are driven by the changes in local air cadmium exposure rather than local air lead exposure. This is so since unlike lead, the primary exposure route of cadmium is dietary rather than respiratory (WHO, 1972; IPCS, 1992; Moon et al., 2003; Ohlsson et al., 2005).[27] Cadmium accumulates in crops, fish and livestock. But since only a small proportion of the food that children in Sweden (and elsewhere in most developed countries) eat is locally produced, *a priori* it is not expected that the intertemporal changes in *local* air cadmium levels in early childhood necessarily are associated with adverse future outcomes.

Nevertheless, to make sure that the impact of the changes in lead are not confounded by the changes in cadmium both the lead and cadmium exposure measures are entered into the same regression to assess to what extent controlling for cadmium has an effect on the precision and/or magnitude of the estimated effect of lead. The estimated lead coefficients are reported in column (4) of Table 7. For most outcomes the estimates effects of lead remain highly similar after the additional control for cadmium is included. These results clearly indicate that the effect of lead does not seem to be caused by the simultaneous changes in cadmium.[28]

In column (5) estimates are reported from a specification where the natural log of exposure has replaced the baseline linear exposure measure in

[27] For example, Moon et al. (2003) calculate the ratio of the dietary route uptake over the sum of the uptake via dietary and respiratory routes in a sample of non-smoking non-occupationally exposed mothers and their children. Cadmium intake was almost exclusively from food (98%), both in children and mothers. Dietary cadmium intake of children significantly correlated with that of their mothers. Dietary lead intake in children, however, did not correlate with that of their mothers. Lead uptake from ambient air tended to be higher (50%) in children than in their mothers (35%).

[28] The parameter estimates for cadmium are almost exclusively insignificant, and almost always point in an unexpected direction; i.e. higher cadmium improves adult outcomes. The discrepancy between the impact of Cd found for infant health in Currie and Schmeider (2009) and the lack of effects on long run outcomes found here could be due to that Currie and Schmeider examine releases of large doses at critical periods *in utero*. The changes in cadmium exposure in this setting potentially reflect more subtle changes at low levels in Cd exposure. Therefore the evidence here should not be taken as evidence that a temporary large dose of air cadmium exposure does not have an effect on long-run outcomes. The cadmium exposure estimates are retained due to space limitations, but are available upon request from the author.

order to investigate to what extent the model is sensitive to changes in functional form. A somewhat counter intuitive feature of the lin-log model, given the suggested nonlinear effect with the strongest effect at the highest exposure, is that it imposes decreasing marginal effects at the highest levels of exposure. Still the effects on GPA and IQ remain highly significant while the impact on schooling outcomes no longer is significant. However, the point estimates suggest effects of similar size as the baseline estimates. This could potentially indicate that exposure at the highest levels are particularly important for the later adult outcomes, while the more subtle effects on IQ and GPA remain even at lower levels of exposure.

Column (6) assess to what extent the exclusion of children growing up in the three largest cities (Stockholm, Göteborg and Malmö) affects the parameter estimates. For various reasons one may suspect that the lead exposure measure in these areas is a less good predictor of the children's blood lead levels than in other less densely populated areas. For example, exposure to motor vehicle exhaust is likely higher and at the same time the moss sampling sites may differ significantly from other areas. However, as shown in column (6) restricting the sample in this way only has limited effects on the precision and the magnitude of most of the estimates.

Approximately simultaneous with the sharp phase-out of leaded gasoline there was also a strong expansion in the public day-care system in Sweden. Hence, a concern is that the impact of the reductions in lead exposure could partly be confounded by increased day-care enrollment (see e.g. Baker, Gruber and Milligan, 2009). However, first of all a regression of the day-care enrollment rates on childhood lead exposure suggests no significant association between the two variables. Still, to make sure that the increase in day-care enrollment rates is not biasing the baseline estimates, column (7) reports the results from a model where cohort-municipality specific day-care enrollment rates (averaged over ages 0-6) has been included as additional controls.[29] Again as seen in column (7) the baseline results are in general not sensitive to this change in specification.

Finally in column (8) I have aggregated the data to the municipality level in order to address the concerns of biased inference due to the regression of a municipality level explanatory variable (lead exposure) on individual outcome data.[30] This alternative and conservative method in general provides

[29] Moreover, since left-wing local governments were more likely to expand public day-care, additional controls for the number of years during the index person's childhood that the municipal council had a left-wing majority is also added. Note that the ideological orientation of the municipality council potentially captures many different hard to observe characteristics of the parents and the municipalities besides childcare. The data on childcare exposure and municipality level political majorities were kindly provided by Per Pettersson-Lidbom.

[30] I follow Bertrand et al. (2004) and first regress all individual variables on the outcomes and then use the average residuals as the outcome variable in a regression on the municipality level controls and fixed effects. The reported standard errors are robust to within municipality correlation. The observations are weighted by the number of children in each municipality-

fairly similar results as the baseline model does which is reassuring. The estimates for the outcomes which no longer remain significant after aggregating the data are typically not significantly different from the baseline estimates.

To summarize, the weight of the evidence presented in this section first of all clearly displays the robustness of the main results to various specification changes. Several tests of alternative explanations for the observed effect of lead on adult outcomes suggest that neither observed nor unobserved changes in other important factors such as other pollutants, unobserved parental characteristics, or municipality specific factors seem to be able to explain the main results.

5.4 Heterogeneity

The analysis now proceeds by investigating the heterogeneity of the main effects. In section 5.4.1 the potential redistributive role that environmental policies may play is examined by checking if low SES and high SES children are differentially affected by the phase-out of leaded gasoline. Section 5.4.2 assess to what the extent there exists any difference in the susceptibility or impact of lead exposure early in life between boys and girls. Finally, in section 5.4.3 the sharp inter-temporal differences in lead exposure is exploited in order to test the validity of the assumption that early childhood lead exposure is more harmful than lead exposure later on in childhood.

5.4.1 Can differences in pollution exposure early in life explain parts of the SES-gap in economic outcome later in life?

Parental resources may potentially help mitigate some of the negative effects of adverse conditions in early life (see e.g. Currie and Hyson, 1999; Case et al., 2002; Cunha and Heckman, 2007). Moreover, several studies have found that low SES children are under higher risks of being exposed to environmental hazards; either through residential segregation or by less care taken by polluters in reducing the risk of exposure in neighborhoods with families of low political and/or economic influence.[31] Information differentials about the health effects of pollution exposure between low and high SES households could also result in differential childhood lead exposure even within the same localities, since it can induce differential avoidance behavior across social groups (c.f. Neidell, 2004). A SES gradient

---

period cell, rather than municipality year of birth cell which explains the differences between the individual level estimates and the estimates from the aggregated data.

[31] See e.g. Davidson and Anderton (2000) or Szasz and Meuser (1997) for a review of the environmental justice literature.

in the long-term effects of early childhood lead exposure could also be expected if parents from different social groups have different preferences for or possibilities to compensate the impact of lead on subsequent outcomes. Finally, since children from poorer backgrounds more often suffer from other health problems, a SES gradient in the effect of early childhood lead exposure could result from interactive effects of lead exposure and other health problems (c.f. Currie et al. 2009). In either case environmental policy initiatives that improve air quality are bound to benefit children in the poorest household the most.[32]

To assess whether the phase-out of leaded gasoline improved long term outcomes particular for low SES children, Table 8 report results from separate regressions by parents earning (below/above the median in 1990), and by parental education attainments (at least one of the parents has completed high school or not). As expected the parameter estimates are systematically larger and more precisely estimated for low SES children suggesting that the benefits of the reductions in lead exposure are particularly beneficial for the relatively disadvantaged children.

Given the data at hand, it is not possible to fully differentiate between the relative importance of the competing underlying mechanism behind the SES gradient. However, since the data also contain information on the parish of birth it is possible to examine if the same SES-gradient persists even if fixed parish of birth effects (2500 parishes) are included as additional controls.[33] A parish on average corresponds to the size of a US census tract (≈ 4.500 individuals). Hence, the parish of birth fixed effects regression compares children growing up in the same "neighborhood" within the municipality, and thereby the importance of differential exposure between low and high SES children induced by residential segregation *within* municipalities should be reduced. While parishes are not an ideal measure of the relevant neighborhood, it is the finest locality of birth data available and can be assumed to work as a decent proxy for it.[34]

[32] Chay and Greenstone (2003b) find suggestive evidence of a SES gradient in the impact of Total Suspended Particulates (TSP) on infant mortality.

[33] Unfortunately, I do not have access to polygons for parish of birth, and hence can not calculate exposure levels for the parishes. However, it should be noted that even if they were available it is not evident that parish exposure would be a preferable measure compared to municipality exposure since parish boarders are likely to be crossed in regular day to day activities to a higher extent than municipality boarders.

[34] A better neighborhood definition than the parish of birth would be to use the SAMS areas (9,000 locations), which are very well defined neighborhoods. However, unfortunately information on the SAMS of residence are not available before 1985, and therefore the parish of birth was preferred in order to minimize the risk of attaining biased estimates due to endogenous parental migration between the year of birth and 1985. However, I have also estimated the same models using SAMS fixed effects instead and the results were *highly* similar.

Table 8 Estimates by socioeconomic status

Specification	(1)	(2)	(3)	(4)
Sample:	Low education parents (no high school)	Educated parents	Below median earnings	Above median earnings
OUTCOMES	N=196,359	N=472,550	N=329,076	N=339, 847
GPA	-.0425**	-.0329**	-.0469***	-.0249*
	(.0166)	(.0144)	(.0152)	(.0151)
Low GPA	.0007***	.0005**	.0008***	.00034*
	(.00024)	(.0002)	(.0002)	(.0002)
High GPA	-.0003	-.00025	-.0004**	-.0002
	(.0002)	(.00020)	(.0002)	(.0002)
IQ (Men)	-.0613***	-.0156	-.0307	-.0378**
	(.0165)	(.0113)	(.0193)	(.0144)
Low IQ (Men)	.0006**	.0002	.00061*	.00006
	(.00026)	(.00016)	(.00034)	(.0002)
High IQ (Men)	-.00039**	-.00016	-.00008	-.00043*
	(.00017)	(.00015)	(.00022)	(.00023)
High School	-.00034	-.0003*	-.00038*	-.00017
	(.0002)	(.00016)	(.00019)	(.00014)
University	-.0004	-.00017	-.00041	-.00009
	(.00025)	(.00026)	(.00026)	(.00025)
Yrs of schooling	-.0028**	-.0016	-.0024**	-.0011
	(.0012)	(.0012)	(.0012)	(.0009)
Welfare	.00022	.00010	.00018	.00005
	(.00015)	(.00007)	(.00011)	(.00006)
Earnings	-.0018***	-.0009	-.00073*	-.0012**
	(.0006)	(.00065)	(.00040)	(.0005)
Teenage mother	-.00011	.00020	.00008	.00012*
	(.00014)	(.00007)	(.0001)	(.00007)
Individual char.	Yes	Yes	Yes	Yes
Parental char.	Yes	Yes	Yes	Yes
Year of birth FE	Yes	Yes	Yes	Yes
Mun. of birth FE	Yes	Yes	Yes	Yes

Notes: Each row and column represent a separate regression. The reported estimates is the marginal effect of a (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two timeperiods and hence adjust standard errors at the timperiod-municipality level). Parent's characteristics include maternal education (7-levels) and indicators for quintile of total parental earnings in 1990. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects.

The estimated differences in the impact of lead exposure early on between children of differential socioeconomic background persist even if they grew up in the same neighborhood. The point estimates generally decrease somewhat but not to the same extent as would have been expected if residential segregation would be the main cause of the SES-gradient (not reported). Next I limited the sample to children with parental earning more or less than the median earnings but, with and without having parents with a college/high school education. The parameter estimates from this matched comparison provide suggestive evidence that the income of parents seems to matter more than education of parents (not reported). The differences in the impact of lead on children in the two parental income groups are still large even after conditioning on parental educational attainments. Hence, although parental earnings seem to matter a lot for the influence of lead exposure on child outcomes an obvious candidate mediating mechanism for this income gradient, residential sorting within municipalities, does not seems to be the main mechanism at work. In addition, even after conditioning on parental education, the parental income gradient remains almost as strong.

Under the assumption that university education of the parents is indicative of a higher awareness of potential negative effects of air pollution (and thereby a higher degree of avoidance behavior), these two auxiliary results suggest either i) that children with parents with low earnings are more heavily affected by air lead levels, potentially due to heightened sensitivity (e.g. due to co-morbidities); ii) that poorer parents lack the resources needed to compensate for the initial insult to the child's development, or iii) that low and high SES parents have different preferences regarding the value of remedying investments in their children.

Whichever of the suggested reasons that are most valid, these results clearly indicate that environmental policies such as the ban of leaded gasoline not only has the ability to reduce the intergenerational transmission in economic outcomes. However, they also indicate that public and/or private resources/initiatives may potentially be effective in reducing the impact of early life insults on long-term outcomes, particularly among low SES children.

5.4.2 Effects of lead exposure by gender

Studies investigating gender differences in the impact of lead exposure has typically not detected differential effects of early childhood lead exposure on behavioral or cognitive outcomes among boys and girls (see e.g. Burns et al 1999). But again these studies typically focus on children with relatively high levels of exposure. It has been suggested that in general male fetuses and infants are more susceptible to damage from early insults to health.

Since boys tend to develop more slowly than girls, this could imply that exposure to lead may lead to greater damage in boys.[35]

To assess this notion Table 9 and 10 present the OLS estimates (except IQ and teenage pregnancies) on all outcomes from separate regressions on the male and the female samples respectively. In general the point estimates are highly similar for both boys and girls. The precision is somewhat better for the girls (particularly for the educational attainments) potentially reflecting the lower variance in these outcomes among girls.

Table 9 Women

Specification	(1)	(2)	(3)	(4)	(5)
	Women	*Women*	*Women*	*Women*	*Women*
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare
Sample	ALL	ALL	ALL	ALL	ALL
Lead exp. (µg/Kg)	-.00033**	-.0003	-.0023**	-.00085	.00014
	(.00014)	(.00026)	(.0011)	(.0006)	(.00009)
R-squared	0.06	0.17	0.19	0.06	0.03
Mean of dep. var.	.91	.38	12.9	141,437	.037
Outcomes:	(6) GPA	(7) LOW GPA	(8) HIGH GPA		
Lead exp. (µg/Kg)	-.0334**	.00043***	-.00038		
	(.0160)	(.00019)	(.00025)		
R-squared	0.19	0.09	0.13		
Mean of dep. var.	56	.18	.31		
Indiv. Char.	yes	yes	yes	yes	yes
Yr of birth FE	yes	yes	yes	yes	yes
Muni. F.E:	yes	yes	yes	yes	yes
# Obs.	324,694	324,694	324,694	291,002	324,694

Notes: The table reports results for separate regression on females. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

[35] It is also interesting to highlight the fact that studies that have investigated gender differences in blood lead levels has found that up until around age 10 the blood lead levels in boys and girls are generally highly similar, and then starts to diverge (see e.g. Strömberg et al., 1995).

Table 10 Men

Specification	(1)	(2)	(3)	(4)	(5)
	Men	*Men*	*Men*	*Men*	*Men*
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare
Sample	ALL	ALL	ALL	ALL	ALL
Lead exp. (µg/Kg)	-.0002	-.0002	-.0020	-.00098*	.00011
	(.0002)	(.00025)	(.0014)	(.00055)	(.00009)
R-squared	0.06	0.17	0.20	0.14	0.03
Mean of dep. var.	.87	.27	12.5	211,095	.037
Outcomes:	(6) GPA	(7) LOW GPA	(8) HIGH GPA		
Lead exp. (µg/Kg)	-.0380**	.00065***	-.00022		
	(.0161)	(.00023)	(.00019)		
R-squared	0.03	0.09	0.11		
Mean of dep. var.	44.8	.31	.18		
Indiv. Char.	yes	yes	yes	yes	yes
Yr of birth FE	yes	yes	yes	yes	yes
Muni. F.E:	yes	yes	yes	yes	yes
# Obs.	371,996	371,996	371,996	310,772	371,996

Notes: The table reports results for separate regression on females. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

5.4.3 Early childhood (age 0-2) vs. pre-primary school (age 5-7) lead exposure

In line with the epidemiological literature the analysis so far has assumed that children's development should be most strongly affected by early childhood lead exposure. As discussed above the motivation for the focus on this age period is that lead take up is higher and the rate of development is particularly rapid and critical in early life. Moreover, when it comes to insults to children's development it has been suggest that earlier insult should have a stronger effect than later insults on subsequent outcomes due to the potentially dynamic complementarities of human capital accumulation; that is if skills beget skills (Cunha and Heckman, 2007). Hence, disadvantages early on may induce children to fall behind and not catch-up to their healthier peers. If either of these notions is true then early exposure should play a greater role than exposure to lead later on.

On the other hand since the brain continues to develop until around age 20, and children potentially recover from early insults, more recent exposure to air pollutants might be more important (Currie et al., 2009). Indeed a few recent studies have suggested that lead exposure in ages 5-7 are more strongly correlated with IQ than early childhood exposure (c.f. Hornung et al., 2009) and the references cited therein).[36] Moreover, it has been suggested that for cognitive skills the most sensitive period is early childhood while the most sensitive period for noncognitive skills occur later in childhood (c.f. Heckman, 2007). Further evidence on the age of greatest vulnerability to lead is moreover of clear policy relevance. If later exposure is shown to produce the same effects as early exposure efforts to reduce blood lead levels should continue through out childhood (Hornung et al., 2009).

In order to differentiate between the impact of early and late childhood lead exposure one would ideally like to have measures of the lead exposure from birth until the outcome of interest is realized. But, since lead exposure at different ages will be highly correlated, a distinction between the impacts of early vs. late childhood exposure is difficult in most settings. With these caveats in mind with my data it is however still possible to estimate a horse race model between early childhood (ages 0-2) and the pre-school age (age 5-7) exposure since for the present cohorts the changes in lead exposure between the different ages are substantial.[37]

The estimates for the impact of lead at the different ages on adult outcomes are presented in Tables 11 and 12. From the results in these tables a clear pattern emerges. For all outcomes the baseline estimates for early exposure is highly similar to the baseline model estimates and in most cases significant. For virtually all outcomes the estimated impact for exposure later in childhood is smaller than the age 0-2 exposure, and not statistically significant. The only two exceptions to this rule is the estimated impact on welfare dependency and earnings where the point estimates is higher for later childhood exposure than early childhood exposure.

Moreover, in most cases the standard errors are not any larger for the later childhood estimates than for the early childhood estimates. Hence, it does not seem as if the later childhood exposure estimate is insignificant just because the precision decreases due to collinearity between the two measures of exposure. However, before concluding that early is much worse than later exposure, it should be kept in mind that there are at least two additional factors which potentially hamper the validity of this interpretation. First, the

[36] However, again previous studies in general investigate children with much higher lead exposure than the children in this setting, use small samples, typically look at cognitive test administered only in childhood and are susceptible to omitted variable bias.

[37] In order to implement this exercise data on lead exposure from the 1990 moss survey was added to the last three cohorts.

Table 11 Age of greatest susceptibility, GPA and cognitive test scores

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	All	All	All	Males	Males	Males
Lead exp.	-.0461***	.0007***	-.0004*	-.0264*	.0002	-.00023
(age 0-2)	(.0164)	(.0002)	(.0002)	(.0150)	.0002)	(.00018)
Lead exp.	-.0162	.00018	-.00013	-.0020	-.0001	-.00019
(age 5-7)	(.0185)	(.0002)	(.0002)	(.0177)	(.0003)	(.00027)
R-squared	0.21	0.12	0.14	0.17	0.09	0.1
Mean of dep. var.	50	0.25	0.25	49.8	0.22	0.26
Individual & parental char..	yes	yes	yes	yes	yes	yes
Yr of birth FEs	yes	yes	yes	yes	yes	yes
Muni FEs	yes	yes	yes	yes	yes	yes
Observations	668,909	668,909	668,909	220,498	220,498	220,498

Note: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate statistical significance at the 10/5/1 percent levels.

Table 12 Age of greatest susceptibility, alternative long-run outcomes

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	High school	Ever in College	Yrs. in School	Log earnings	Welfare	Teen mother
Sample	All	All	All	All	All	All
Lead exp.	-.00027*	-.0001	-.0015	-.0013***	.00016	.00014*
(age 0-2)	(.00015)	(.0003)	(.0010)	(.0005)	(.0001)	(.00008)
Lead exp.	.0002	.0004	.0021	-.0021***	.00027*	.00007
(age 5-7)	(.0002)	(.0003)	(.0016)	(.0008)	(.00015)	(.0001)
R-squared	0.06	0.18	0.21	0.13	0.03	0.03
Mean of dep. var.	0.89	0.32	12.7	177,283	0.037	0.042
Individual & parental char.	yes	yes	yes	yes	yes	yes
Yr of birth FEs	yes	yes	yes	yes	yes	yes
Muni FEs	yes	yes	yes	yes	yes	yes
Observations	668,909	668,909	668,909	601,774	668,909	325,010

Note: see Table 11

exposure measure used is local exposure rather than individual blood lead levels. Since lead uptake is higher in early childhood the differences in the impact on long run outcomes could be due to differential blood lead levels at the different ages. Second, the nonlinearity of the relationship between childhood lead exposure and adult outcomes suggested above could also provide a similar pattern since the lead exposure in the pre-primary school years typically has decreased below the relevant level of concern as estimated above.

Although definite conclusions regarding the most sensitive period is difficult to make with the data at hand at least the results in this section do not provide any direct support for the hypothesis that later childhood lead exposure should be more detrimental than earlier exposure. Instead, the weight of the evidence suggests that early childhood lead exposure is more influential than later exposure which is in line with most the theoretical mechanisms suggested in the literature and the previous empirical evidence.

6 Economic significance and policy implications

In order to attain a rough estimate of what blood lead level the critical moss lead levels correspond to, I use the model estimated in Nilsson et al. (2009) for the pre-ban of leaded gasoline period.[38] This yields an estimate suggesting that for the children aged 7-10 a local moss lead level of 50µg/kg (i.e. lower end of the 4th quartile of exposure used in section 5.2) correspond to a blood-lead level of around 3µg/dL under a log-normal distribution. After adjusting the blood-lead moss-lead elasticity using the age specific blood-lead gasoline-lead elasticity estimated in Reyes (2007) (30% higher for children aged 0-6 than for children aged 6-12) and under the additional assumption that the additive separable specification used in the estimation holds for both populations, the relevant blood lead level in this setting would correspond to about 4.8µg/dL.

This estimate suggests that the average early childhood blood lead level among children in more than 50% of the Swedish municipalities in the period 1972-1974 were high enough to affect their adult outcomes. Since these municipalities also are the most densely populated, a majority of the children in Sweden born in the late 1960s and early 1970s likely suffered from blood lead levels high enough to potentially affect their future adult outcomes. However, since this study use the average municipality lead levels as exposure measure, the average effects on cognitive ability are associated with a *municipality* average blood lead level above 4.8µg/dL. It is thus in principle possible that the entire effect could solely be caused by large effects on cognitive development among a few children with very high blood

[38] Evaluated at the mean of the explanatory variables

lead levels. However, since that the standard deviation in childhood blood lead levels is not extremely large this seems less likely. Still, the preciseness of this blood lead level "threshold" remains to be confirmed in future research using individual childhood blood lead levels *and* a credible empirical strategy that takes unobserved confounders into account. At the very minimum the evidence provided in this study gives a clear indication that while the current acceptable blood lead limit (10µg/dL) is set at a level above which acute effects of lead might be avoided, it is clearly not low enough to prevent more subtle damage on child development.

With these caveats in mind it is interesting to consider the effects on future GPA and earnings if early childhood blood lead levels would decrease from 10µg/dL to 5µg/dL. By combining the estimated average impact on GPA in the upper quartile (see Table 5 & 6) and assuming that the estimated elasticity between lead in moss and lead in children is constant in this interval (i.e. 0.57) a decrease in a child's blood lead level from 10 µg/dL to 5 µg/dL would imply an average increase in 9th grade GPA by 2.2 percentiles and an increase in the high school graduation rate by 2.3 %. In terms of labor market outcomes the same decrease would imply an estimated increase in earnings (average for ages 20-32) by 5.5%.

Although due to the strong life-cycle variations in income, concurrent earnings measured below age 30 is typically not a very accurate measure of life time earnings, and hence the earnings estimate should be interpreted with care (c.f. Haider and Solon, 2006; Lindqvist and Böhlmark, 2006). If instead regressing age 30-32 earnings (i.e. only for those born in 1972-1974) on a high school graduation dummy (or grade 9 GPA), gender, year of birth and family fixed effects, the Swedish high school premium is estimated to be about 17%, and a one (1) percentile rank increase in GPA is associated with on average 0.54% higher earnings at age 30-32.[39] If combining these estimates with the estimated effects of lead exposure on GPA and high school graduation rates, the effect of reducing early childhood blood lead levels from 10 to 5µg/dl implies that life time earnings would increase by 1.2% (2.2*0.54) using the GPA/earnings estimate and around 0.4% (17*0.023) using the high school graduation premium estimate.[40] Of course these alternative estimates only capture the part of the lead exposure effects on earnings that goes through educational attainments.

With these estimates it is for example possible to calculate the hypothetical gains of reducing the blood lead levels of the 310,000 children in the US (c.f. CDC, 2005) with a blood lead above 10µg/dL to 5µg/dL.

[39] In Sweden the life cycle bias in earnings are found to be minimal after age 33 which is why I estimated the impact of the educational attainments on earnings for the earliest cohorts only; see further Böhlmark and Lindqvist, (2006).

[40] The lower estimate for high school likely reflects that part of the earnings effects which goes through the impact of increased university *completion* (which is too early to estimate).

Since general equilibrium effects most likely is not an issue, under the assumption that the earnings effects are directly translatable to the US setting, and given an annual income of 30,000 USD, the benefits in terms increased labor market earnings from reducing the blood lead level in these children would hence be around USD $112 million annually after age 32 (30,000*310,000*0.012) using the GPA/earnings estimate, and around USD $37 million annually using the high school graduation/earnings estimate.[41] This reflect the effect on the average population of children, but since 60% of all children with blood lead levels above 10μg/dL are Medicaid eligible (see Currie, 2009) the expected effects on individual earnings could be larger.

7 Concluding remarks

This study use a new measure of early childhood lead exposure to estimate the long run effects of the rapid reductions in lead exposure following the phase out of leaded gasoline. The results are robust to a number of specification changes and suggest that in Sweden the reduction in children's blood lead levels that occurred between 1972 and 1984 has improved young adult outcomes for a majority of the population.

The main policy implication of the results concerns the evidence of the nonlinear effect of municipality air lead levels in early childhood on young adult outcomes. This nonlinear relationship provides suggestive evidence of the existence of a threshold below which further reductions in early childhood blood lead levels no longer improves long term outcomes. Given the wide use of heavy metal moss monitoring throughout Europe, the finding that reductions in lead exposure below 49μg/kg moss no longer seems to affect long-term outcomes is of clear policy relevance in itself. However, this study also provides an estimate suggesting that the critical moss lead level corresponds to an early childhood blood lead level of approximately 5μg/dL. This is well below the current blood lead limit of concern (10μg/dL) suggested by the US Center for Disease Control and Prevention (CDC). Since the CDC estimates that more than 310,000 children aged 1-5 in the US alone have blood lead levels exceeding 10μg/dL, and WHO estimate that globally 40% of the urban children suffer from blood lead levels that exceed 5μg/dL (Fewtrell et al., 2003), reductions in the recommended limit of concern and implementations of further programs designed to reduce lead exposure could potentially be cost effective

[41] Note that this calculation assumes that the lead levels in all the children are lowered from 10μg/dL to 5μg/dL, which implies that the gain is underestimated since a non-negligible share of the children has higher levels than 10μg/dL.

A second key result of this study is that while low SES children seem to suffer more heavily from lead exposure in early childhood, the SES differences *does not* seem to be solely caused by differences in pollution exposure due to residential segregation. The SES-gap persists even when comparing children growing up in the same neighborhood. This result indicate not only that environmental policies, such as the ban of leaded gasoline, could function as social policy, but also that public or private investments may mitigate some of the detrimental effects of early life exposure to lead.

In planned future work the same strategy will be used to investigate if early childhood lead exposure at low levels can yield similarly sized effects on violent crime rates as Reyes (2007) finds for considerably higher levels of exposure. Other relevant health outcomes such as birth outcomes will also be considered.

References

Almond, D., Y. Chen, M. Greenstone, H. Li, "Unintended Consequences of China's Huai River Policy", *American Economic Review: Papers and Proceedings,* 99,May, 184-190.

Andersson, H., K. Petersson-Grawe, E. Lindqvist, J. Luthman, A. Oskarsson, L. Olson (1997). "Low-level cadmium exposure of lactating rats causes alterations in brain serotonin levels in the offspring", *Neurotoxicol. Teratol.* 19, 105–115.

Baker M, J. Gruber, K., Milligan (2008), "Universal childcare, Maternal Labor Supply, and Family well-being", *Journal of Political Economy* 116(4), 2008, p. 709-745.

Bellinger DC. (2004a), "Assessing environmental neurotoxicant exposures and child neurobehavior: confounded by confounding?", *Epidemiology* 15:383–384.

Bellinger DC (2004b), "Lead", *Pediatrics*;113;1016-1022.

Burns J., P. Baghurst, M. Sawyer, A McMichael, S Tong (1999), "Lifetime low-level exposure to environmental lead and children's emotional and behavioral development at ages 11–13 years. The Port Pirie Cohort Study." *Am J Epidemiol*.;149:740–749.

Böhlmark A., J. Lindquist (2006), "Life-Cycle Variations in the Association between Current and Lifetime Income:Replication and Extension for Sweden", *Journal of Labor Economics,* vol. 24, no. 4

Case A., D. Lubotsky, C. Paxson, (2002), "Economic Status and Health in Childhood: The Origins of the Gradient," *American Economic Review*, American Economic Association, vol. 92(5), 1308-1334, December.

CDC (2005), Centers for Disease Control and Prevention, "Blood Lead Levels in Young Children---United States and Selected States, 1999-2002". *MMWR*. May 27, 2005, 54(20);513-516.

Canfield, R. , C. Henderson, R. Cory-Slechta, et al. (2003) **"**Intellectual Impairment in Children with Blood Lead Concentrations below 10 microgram per Deciliter**"** *New England Journal of Medicine,* 348: 1517-1526.

Carlstedt, B. (2000), *Cognitve abilities - aspects of structure, process, and measurement*, doctoral thesis , Department of Education, Göteborg : Acta Universitatis Gothoburgensis

Chay, K. , M. Greenstone, (2003a) "Air Quality, Infant Mortality, and the Clean Air Act of 1970," *NBER Working Paper* No.10053.

Chay, K. , M. Greenstone, (2003b), "The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession", *Quarterly Journal of Economics*, August, 1121-1167.

Cunha, F., J Heckman (2007), "The Technology of Skill Formation,"*American Economic Review*, May, *97* (2), 31–47.

Currie, J. (2009), "Healthy, Wealthy, and Wise? Socioeconomic Status, Poor Health in Childhood, and Human Capital Development," *Journal of Economic Literature*, 47 #1, March , 87-122.

Currie, J., R. Hyson (1999) "Is the Impact of Health Shocks Cushioned by Socioeconomic Status?: The Case of Birth Weight," *American Economic Review,* May, 89 #2, 245-250.

Currie, J., M. Neidell, (2005). "Air Pollution and Infant Health: What Can We Learn from California's Recent Experience?," *The Quarterly Journal of Economics*, vol. 120(3), pages 1003-1030, August.

Currie, J., J. Schmieder (2009) "Fetal Exposure to Toxic Releases and Infant Health," (2009), *American Economic Association Papers and Proceedings,* .

Currie J., M. Stabile, P. Manivong, L. Roos (2009), "Child Health and Young Adult Outcomes, forthcoming, *Journal of Human Resources*.

Davidson P., D Anderton (2000), "Demographics of dumping. II: A national environmental equity survey and the distribution of hazardous materials handlers" *Demography,* Nov;37(4):461-6.

Dobbing J. (1968), "Vulnerable periods in developing brain". In: Davison AN, Dobbing J, eds. *Applied Neurochemistry*. Philadelphia: Davis, 1968, 287–316.

Fewtrell L., R. Kaufman, A. Prüss-Üstün (2003), *Lead: assessing the environmental burden of disease at national and local level*., Geneva, World Health Organization., (WHO, Environmental Burden of Disease Series, No. 2)

Folkeson L. (1979), "Interspecies calibration of Heavy-metal concentrations in nine mosses and lichens: – Applicability to deposition measurements", *Water, Air and Soil Pollution,* **11** 253-260.

Giussani V. (1994), "The UK clean air act 1956: an empirical investigation", CSERGE Working Paper GEC 94-20.

Haider S., G. Solon (2006), "Life-Cycle Variation in the Association between Current and Lifetime Earnings", *American Economic Review*, 96 (4), September, 1308-1320.

Heckman, J. (2007), "The technology and neuroscience of capacity formation", *Proceedings of the National Academy of Sciences (PNAS)*, 104 #33, August 8, 2007: 13250-13255.

Hornung RW, BP Lanphear, KN Dietrich, (2009), "Age of greatest susceptibility to childhood lead exposure: a new statistical approach**",** *Environ Health Perspectives*. Aug;117(8):1309-12.

IPCS (1992), International Programme on Chemical Safety *Environmental health criteria 134. Cadmium.,* Geneva: World Health Organization.

Lanphear B., K. Dietrich, P. Auinger, C. Cox. (2000), "Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents", *PublicHealth Rep.* 115(6):521–29.

Lanphear B., R. Hornung and J. Kouhry et al.(2005), "Low-Level Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis", *Environ Health Perspect*. July; 113(7): 894–899.

Lidsky TI, Schneider JS. 2003. "Lead neurotoxicity in children: basic mechanisms and clinical correlates", *Brain,* 126:5–19.

Lopez R. (2002), "Segregation and Black/White Differences in Exposure to Air Toxics in 1990", *Environmental Health Perspectives*, 110(suppl 2):289–295.

Lüchinger, S. (2009), "Air Pollution and Infant Mortality: A natural experiment from Power Plant Desulfurization", mimeo LSE.

MOENR (1994), *The Swedish Experience - Taxes and Charges in Environmental Policy.* Ministry of the Environment and Natural Resources. Stockholm, Sweden.

Moon C.-S. J-M Paik, C-S Choi, D-H Kim, M. Ikeda (2003), "Lead and cadmium levels in daily foods, blood and urine in children and their mothers in Korea", *Int Arch Occup Environ Health* 76: 282–288

Neidell, M. (2004), "Air pollution, health, and socio-economic status: the effect of outdoor air quality on childhood asthma", *Journal of Health Economics*, Volume 23, Issue 6, November, 1209-1236.

Neidell, M. (2009), "Information, Avoidance Behavior, and Health: The Effect of Ozone on Asthma Hospitalizations." *Journal of Human Resources*, forthcoming.

Nilsson, J P, S. Skerfving, E. Stroh, U. Strömberg (2009), "The Relationship Between Ambient Air Pollution and Pollution Exposure in Children", mimeo, Uppsala University.

Nevin, R., (2000), "How Lead Exposure Relates to Temporal Changes in IQ, Violent Crime, and Unwed Pregnancy," *Environmental Research,* **83**(1), p. 1-22.

Nordell, O., (2007), "Tungmetaller i mossor i Landskrona kommun 1983, 1995 och 2006", Rapport 2007:3, Miljöförvaltningen Landskrona

OECD (1999), *Phasing lead out of gasoline: an examination of policy approaches in different countries*, Paris; 1999

Olsson I-M, Eriksson J, Öborn I, Skerfving S, Oskarsson A., (2005), "Cadmium in food production systems: A health risk for sensitive population groups". *Ambio*;34:344-351.

Onianwa P.C. (2001),"Monitoring Atmospheric Metal Pollution: A Review of the Use of Mosses as Indicators", *Environmental Monitoring and Assessment*, **71,** pp 13-50.

Petersson-Grawe, K., Teiling-Gardlund, A., Jalkesten, E. and Oskarsson, A. (2004), "Increased spontaneous motor activity in offspring after maternal cadmium exposure during lactation", *Environ. Toxicol. Pharmacol.* 17, 35–43.

Rankin, J., T. Pless-Mulloli, S. Glinianaia, (2007) "Does the effect of air pollution on pregnancy outcomes differ by gender? A systematic review", *Environmental Research*, Volume 105, Issue 3, November, Pages 400-408.

Rehnberg, I. , M. Brännvall, R. Bindler, O. Emteryd (2000), "Atmospheric Lead Pollution History during Four Millennia (2000 BC to 2000 AD) in Sweden"**,** *Ambio* Vol. 29 No. 3, May.

Reyes, Jessica W. (2007), "Environmental Policy as Social Policy? The Impact of Childhood Lead Exposure on Crime," *The B.E. Journal of Economic Analysis & Policy*: Vol. 7: Iss. 1 (Contributions), Article 51.

Rühling, Å., and G. Tyler (1968), "An ecological approach to the lead problem", *Botaniska Notiser***121**, 321–342.

Rühling, Å. and Tyler, G.: (1969), 'Ecology of heavy metals – a regional and historical study', *Botaniska Notiser* **122**, 248–259.

Ruhling, Å, (ed.), (1994), "Atmospheric heavy metal deposition in Europe. Estimations based on moss analysis". *Nord 1994, 9* (Nordic Council of Ministers, Copenhagen).

Schwartz, J. (1994), "Low-level lead exposure and children's IQ: A meta-analysis and search for a threshold". *Environ.Res.* **65** ,42–55

Skerfving S, Bergdahl IA. Chapter 31. "Lead", In: Nordberg GF, Fowler BA, Nordberg M, Friberg LT. *Handbook on the Toxicology of Metals*, Academic Press, Elsevier, p.599-643. ISBN 978-0-369413-3.

Strömberg U, A Schütz, S Skerfving, (1995) "Substantial decrease of blood lead in Swedish children, 1978-94, associated with petrol lead". *Occup Environ Med* 52:764-9.

Strömberg U, T. Lundh, A. Schütz, et al. (2003), "Yearly measurements of blood lead in Swedish children since 1978: an update focusing on the perol lead free period 1995-2001". *Occup Environ Med*;60:370-2

Smodis B. and R. Parr (1999), "Biomonitoring of air pollution as exemplified by recent IAEA programs", *Biological Trace Element Researc*, ***1,*** pp. 257-266.

Thomas, V., R. Socolow, J. Fanelli, T. Spiro (1999); "Effects of Reducing Lead in Gasoline: An Analysis of the International Experience", *Environ. Sci. Technol.,* **33** (22), 3942 -3948.

World Health Organization (1972). Sixteenth Report of the Joint FAO/WHO Expert Committee on Food Additives. *WHO Technical Report Series* no. *505*. Geneva: WHO.

Appendix A: The relationship between lead exposure and blood lead among children

This section briefly review the results from Nilsson, Skerfving, Stroh and Strömberg (2009) that provide the estimated elasticity between lead levels in moss and blood-lead levels in children. The interested reader is referred to that study for further details on the data and execution.

The moss samples Nilsson et al. (2009) use was collected at 55 sites in the municipality of Landskrona in 1983, 1995 and 2006, following the same principles as in the national bio-monitoring program. These data where then matched to the blood lead measurements from about 420 children aged between 7 and 10 collected by Strömberg et al. (1995, 2003) in the year prior to that during which the mosses where sampled. Using the coordinates of the children home address each child is assigned an average lead exposure level using the 5 nearest moss sampling sites.[42] The raw correlation between this lead exposure measure and children's blood lead level is 0.75, which compares very well with findings in previous studies linking ambient air pollution to actual population exposure.

Table A1 report the estimated elasticity between lead in moss and children's blood lead levels using six different versions of the following specification,

$$\ln(blood\_lead)_{it} = \alpha + \gamma \ln(exposure)_{it} + X'\beta + \theta_c + \theta_t + \varepsilon_{it} \qquad \text{(A1)}$$

In the first column of Table A1 the elasticity between B-Pb and M-Pb using the full sample is shown without any additional control variables added to the model. The estimated coefficient suggest that for an 10% increase in the lead level in moss the blood lead level increases with on average 3%. In column (2)-(5), individual characteristics, fixed community, year of sampling and finally year*community fixed effects is stepwise introduced. The year fixed effects seem to be the only control which influence on the estimated elasticity.

[42] Following Currie and Neidell (2003), in order to assess the accuracy of the air pollution measure Nilsson et al., compare the actual level of pollution at each moss sample site with the level of pollution that they would have assigned using the implemented method (i.e. using the five closest measuring sites), if the actual moss sample was not in fact available. The correlation of the actual and estimated level is high for Pb (r=.88), suggesting that it is an accurate measure for the air pollution exposure for the children's home address. Also note that as long as the measurement errors in assigned and actual exposure are not systematic, the relationship between the children's blood-lead levels and our air pollution measure will be biased towards zero.

Table A1 The relationship between blood lead and moss lead levels

Dependent variable:	ln (B-Pb)	ln (B-Pb)	ln (B-Pb)	ln (B-Pb)	Ln (B-Pb)	ln (B-Pb)
Specification:	(1)	(2)	(3)	(4)	(5)	(6)
Time period:	ALL	ALL	ALL	ALL	ALL	Before 1995
(ln) Lead exposure	.303***	.333***	.383***	.250**	.287***	.440***
	(.034)	(.035)	(.037)	(.095)	(.099)	(.111)
Individual controls	no	yes	yes	yes	yes	yes
Community F.E.	no	no	yes	yes	yes	yes
Year F.E.	no	no	no	yes	yes	yes
Year*community FE	no	no	no	no	yes	yes
R-squared	0.55	0.69	0.74	0.92	0.92	0.92
Nr of children	410	410	410	410	410	249

Notes: The table reports regression results from of OLS estimations of equation (A1). All in all there are 410 children in 50 cells (249 children and 30 cells in column (6)). The dependent variable is the average blood-lead level at each monitoring point and is weighted with the number of children in each cell. The blood lead is measure in µg/L blood. The lead exposure is µg/kg of moss. The controls are gender, whether the child's practicing any lead exposing hobbies, and ln(hemoglobin) level. The data has been trimmed so to leave out children with blood lead values below the 1st and above the 99th percentiles in each year (7 children in total). Standard errors are reported in parenthesis and are robust with respect to heteroscedasticity. */**/*** reflects significance at the 10/5/1 percent levels respectively. Source: Nilsson, Skerfving, Stroh and Strömberg (2009)

The first five columns report the estimated elasticity using the full sample. However, from 1995 lead in gasoline was banned. Hence as the relative contribution of air lead for total body burden decreases, the predictive power of the moss samples is likely to decrease as mosses only take up lead from the air. This is mirrored in Figure 1 which show that while the moss in lead continued to decrease throughout the observation period, the children blood-lead levels leveled off at around 2 µg/dL after the ban on leaded gasoline. This is pattern is clearly in line with a shift away from air-borne sources as the major source of lead exposure in children after lead was phased out of gasoline. In column (6) we test this notion by estimating the full model, only on the two cohorts sampled before the ban on leaded gasoline, i.e. in 1984 and 1994. When using this restricted sample the estimated elasticity increases to 0.44 while the standard errors increase only marginally. This result suggests that the relative importance of air-lead exposure indeed was stronger in the period prior to the ban than after, as expected. The pre-ban period is also the one focused on in this paper. The final result also provides suggestive evidence on the validity of using mosses as monitors of *air* pollution.

Appendix B: Tables

Table B1: Descriptive statistics

Outcome variables	Definitions	Mean	Std. dev.
GPA	Grade point average (percentile ranked)	50.09	28.8
Low GPA	=1 if GPA in bottom 25%, 0 otherwise	.25	.43
High GPA	=1 if GPA in top 25%, 0 otherwise	.25	.43
IQ	IQ test score (percentile ranked)	50.0	28.5
Low IQ	=1 if IQ in top 25%, 0 otherwise	.25	.43
High IQ	=1 if IQ in bottom 25%, 0 otherwise	.25	.43
Schooling	Year of schooling (imputed)	12.7	1.9
High School	=1 if completed high school, 0 otherwise	.89	.31
University	=1 if ever attended Higher education, 0 otherwise	.33	.47
Earnings	Natural log Labor market earnings	7.2	1.14
Welfare	=1 if receiving welfare, 0 otherwise	.04	.19
Parental characteristics			
% with at least one parent graduated from High school		60	41
% with at least one parent graduated from College		32	47
Sum of parent earnings: SEK 100 (measured in 1990)		2584	1379
Family size		1.5	0.6
Mothers year of birth		1950	0.6
Municipality of birth characteristics:			
Lead exposure (µg/kg)		35	16
Cadmium exposure (µg/kg)		.54	.19
% in childcare average (age 0-6) share of cohort in daycare		13.5	6.9

Table B2: Cross-sectional estimates

Specification	(1)	(2)	(3)	(4)
Sample:	1975	1980	1985	All years
OUTCOMES	N=291,539	N=255,587	N=250,763	N=797,889
GPA	-.0094	-.0298	-.0591	-.0168
	(.0122)	(.0235)	(.0402)	(.0144)
Low GPA	.00023	.00055*	.00042	.0003*
	(.00016)	(.0003)	(.00047)	(.00018)
High GPA	.00006	-.00015	-.0009*	-.0001
	(.00015)	(.00027)	(.0005)	(.00017)
IQ (Men)	-.0258**	-.0395	-	-.0297*
	(.0131)	(.0257)		(.0153)
Low IQ (Men)	.00025	.00035	-	.00029*
	(.00016)	(.00025)		(.00017)
High IQ (Men)	-.00024	-.00043	-	-.00029
	(.00016)	(.00034)		(.00019)
High School	-.00034***	.00009	-.00004	-.00017
	(.0001)	(.00028)	(.00025)	(.00011)
University	-.0002	-.00035	-.0001	-.00024
	(.0002)	(.00045)	(.0004)	(.00023)
Years of schooling	-.0017	-.00106	-.00059	-.00136
	(.0011)	(.0022)	(.0015)	(.0009)
Welfare	-.00006	-.00006	-.0008	-.00012
	(.00004)	(.00014)	(.00019)	(.00008)
Earnings	.00024	.0014***	.00321	.00105
	(.0002)	(.00041)	(.00102)	(.0002)
Teenage mother	-.000016	-.0001	-.00004	-.00005
	(.00007)	(.0001)	(.00012)	(.00006)
Individual characteristics	Yes	Yes	Yes	Yes
Parental characteristics	Yes	Yes	Yes	Yes
Year of birth fixed effects	Yes	Yes	Yes	Yes
Municipality of birth F.E.	No	No	No	No
Mean lead level (μg/Kg)	49.41	30.81	22.77	35.08

Notes: Each row and column represent a separate regression. The reported estimates is the marginal effect of a (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two time-periods and hence adjust standard errors at the timperiod-municipality level). Parent's characteristics include maternal education (7-levels) and indicators for quintile of total parental earnings in 1990. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects.

Table B3 Baseline estimates for municipalities above 25%-tile initial (1975) lead exposure: Grade point averages and cognitive test scores.

Specification	(1)	(2)	(3)	(4)	(5)	(6)
Outcomes:	GPA	Low GPA	High GPA	IQ	Low IQ	High IQ
Sample	ALL	ALL	ALL	Males	Males	Males
Lead exp. (µg/Kg)	-.0356**	.0006***	-.0003	-.0336***	.0003**	-.00026**
	(.0143)	(.0002)	(.0002)	(.0128)	(.00015)	(.00012)
R-squared	0.22	0.12	0.14	0.17	0.09	0.1
Mean of dep. var.	50	025	0.25	49.8	022	0..26
Individ. controls	Yes	Yes	Yes	Yes	Yes	Yes
Y. of birth F.E.	Yes	Yes	Yes	Yes	Yes	Yes
Muni. F.E.	Yes	Yes	Yes	Yes	Yes	Yes
# Obs.	668,909	668,909	668,909	220,324	220,324	220,324

Notes: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

Table B4 Baseline estimates for municipalities above 25%-tile initial (1975) lead exposure: Educational attainments and other long-term outcomes

Specification Outcomes:	(1) High School	(2) Ever in College	(3) Yrs. in School	(4) Log earnings	(5) Welfare	(6) Teen mother
Sample	ALL	ALL	ALL	ALL	ALL	ALL
Lead exp. (µg/Kg)	-.00033* (.00017)	-.0002 (.0002)	-.0022* (.0012)	-.0009** (.0005)	.00014 (.00009)	.0001 (.0001)
R-squared	0.06	0.18	0.20	0.1301	0.03	0.03
Mean of dep. var.	0.89	0.32	12.7	177,283	0.037	0.042
Individ. charact.	yes	yes	yes	yes	yes	yes
Year of birth	yes	yes	yes	yes	yes	yes
FE muni.	yes	yes	yes	yes	yes	yes
# Obs.	696,690	696,690	696,690	601,774	696,690	325,010

Notes: The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. Standard errors are clustered at the municipality level. */**/*** indicate significance at the 10/5/1 percent levels.

Table B5 Alternative sample restrictions

Specification *Sample:*	(1) Exclude <25%-tile *initial* lead exposure	(2) Exclude <25%-tile *change* in lead exposure	(3) Exclude >75%-tile *initial* lead exposure	(4) Exclude >75%-tile *change* in lead exposure
OUTCOMES	N=696,690	N=665,116	N=572,019	N=565,758
GPA	-.0356** (.0143)	-.0326** (.0143)	.0120 (.0171)	.0121 (.0157)
Low GPA	.0006*** (.0002)	.00052*** (.0002)	-.0003 (.0002)	-.0003 (.0002)
High GPA	-.0003 (.0002)	-.00025 (.0002)	-.0001 (.0002)	-.00016 (.0002)
IQ (Men)	-.0336*** (.0128)	-.0322** (.0129)	.0265 (.0190)	.0248 (.0186)
Low IQ (Men)	.0003** (.00015)	.0003 (.0002)	-.0002 (.0003)	-.0001 (.0003)
High IQ (Men)	-.00026** (.00012)	-.0003 (.0002)	.0004 (.0003)	.0004 (.0003)
High School	-.00033* (.00017)	-.00025* (.00015)	-.0001 (.0002)	-.0001 (.0002)
University	-.0002 (.0002)	-.00028 (.00024)	.00005 (.00046)	-.0001 (.0004)
Yrs. of schooling	-.0022* (.0012)	-.0019* (.0010)	.0011 (.0020)	.0002 (.0018)
Welfare	.00012 (.0001)	.0001 (.0001)	.0001 (.0001)	.0001 (.0001)
Earnings	-.0009** (.00045)	-.0009*** (.0005)	.0023*** (.0007)	.0021*** (.0007)
Teenage mother	.0001 (.0001)	.0001 (.0001)	-.0003*** (.00013)	-.00014 (.0001)
Individ. char.	Yes	Yes	Yes	Yes
Parental & muni. characteristics	Yes	Yes	Yes	Yes
Y. of birth F.E.	Yes	Yes	Yes	Yes
Mun. of birth F.E.	Yes	Yes	Yes	Yes

Notes: Each row and column represent a separate regression. The reported estimates is the marginal effect of a (1) unit (1 μg/Kg) increase in municipality of birth lead exposure during early childhood. The IQ and GPA variables are percentile ranked for each graduation/ enlistment cohort. */**/*** indicate significance at the 10/5/1 percent levels. Standard errors are clustered at the municipality level (except for the IQ outcomes regressions that only uses two timeperiods and hence adjust standard errors at the timeperiod-municipality level). Parent's characteristics include maternal education (7-levels) and indicators for quintile of total parental earnings in 1990. The IQ (GPA) outcomes also control for year of enlistment (Graduation) specific effects.