Swedish Institute for Social Research (SOFI)

Stockholm University

WORKING PAPER 9/2014

CHILDHOOD LEAD EXPOSURE AND CRIMINAL BEHAVIOR: LESSONS FROM THE SWEDISH PHASE-OUT OF LEADED GASOLINE

by

Hans Grönqvist, J Peter Nilsson and Per-Olof Robling

Childhood Lead Exposure and Criminal Behavior: Lessons from the Swedish Phase-Out of Leaded Gasoline[◊]

Hans Grönqvist[*], J Peter Nilsson[°] and Per-Olof Robling[•]

October 16, 2014

Abstract
This paper examines the effect of childhood lead exposure on crime using population based register data. We follow all children in Sweden for more than twenty years and observe criminal behavior both before and after the peak of the age-crime profile. By exploiting the variation in childhood lead exposure induced by the phase-out of leaded gasoline, we show that the sharp drop in lead exposure reduced crime by between 7 and 14 percent on average. At the relatively low levels of exposure considered, the analysis reveals a nonlinear relationship, indicating the existence of a threshold below which further reductions early childhood lead exposure no longer affect crime. The impact is moreover largest among children in low-income families

JEL: I18, K42, Q53
Key words: Environmental policy, Lead, Crime

[◊] We are grateful to comments by Anders Björklund, Markus Jäntti and seminar participants at SOFI.
[*] Department of Economics, Uppsala University; hans.gronqvist@nek.uu.se
[°] Institute for International Economics (IIES), Stockholm University; peter.nilsson@iies.su.se
[•] Institute for Social Research (SOFI), Stockholm University; robling@sofi.su.se

1. Introduction

Lead exposure is a major public concern because of its well-known adverse health effects. One of the most important policy efforts to reduce lead exposure was undertaken between the 1970s and early 80s when many developed countries phased out leaded gasoline. In Sweden, the main reduction in gasoline-lead levels occurred between 1973 and 1981 when the maximum allowed lead level per liter of gasoline was cut by close to 80 percent. Since, in Sweden, leaded gasoline was the main source of lead exposure in the general population blood-lead levels (B-Pb) fell sharply until the mid-1990s when leaded gasoline was banned.

In other countries, despite similar efforts, lead exposure remains a public health concern. A large fraction of the population is still exposed to lead on a daily basis from paint, batteries, water pipes, and children's toys. For example, the U.S. Department of Housing and Urban Development estimates that 38 million homes constructed before 1978 still contain lead-based paint. At the global level, WHO estimates that about 49 percent of all children have blood lead levels above 5 micrograms/dl blood.[1] In South East Asia, this number is close to 74 percent.

In this paper we examine the impact on crime of childhood lead exposure in the context of the Swedish phase-out of leaded gasoline. We exploit the fact that the phase-out implied large variation in the reduction of lead exposure across localities over time. The effect of the phase-out of leaded gasoline on adult outcomes has previously been studied by Nilsson (2009a), who takes advantage of cross-regional and cross-cohort variation in lead exposure induced by the Swedish phase out. Nilsson shows that reduced lead exposure early in life improves school performance, cognitive ability, and labor market outcomes.

[1] C.f. http://apps.who.int/gho/data/node.main.142 . Until recently, in the US, children were identified as having a blood lead "level of concern" if the test result is 10 or more micrograms per deciliter of lead in blood.

A growing body of research has linked lead exposure to decreased cognitive ability and behavioral problems. Lead exposure is believed to be especially harmful early in life when higher brain functions develop. Infants and toddlers are particularly vulnerable due to their high exposure from hand-to-mouth activities and inhalation of lead-laced dust, high absorption rate, increased penetration of the blood–brain barrier and a developing nervous system (Etzel 2003). The rate of absorption is as high as 40 percent in children compared to 10 percent among adults. Cellular and animal studies have confirmed the hypothesis that lead exposure during critical stages of development disrupts the formation of neuron networks and the process of neurotransmission in ways that increases the risk of these types of behavioral problems (Weiss and Elsner 1996). In a seminal paper, Herbert H. Needleman and co-authors showed that high blood levels among 3,329 first and second graders in Massachusetts strongly predicted lower scores on intelligence tests and various neuropsychological deficiencies (e.g. Needleman et al. 1979). These results have been corroborated by subsequent studies that in addition to cognitive deficiencies also find that lead exposure during childhood is associated with substantially higher risks of attention problem, impulsivity, and aggressive behavior (e.g. Canfield et al. 2003; Banks et al. 1997). The detrimental effects of lead exposure on cognitive skills have also been documented in recent studies that use various quasi-experimental research designs (Rau, Reyes and Urzua 2012, Reyes 2011; Nilsson 2009a).

Since low cognitive skills, attention deficiencies, impulsiveness and aggression are all well-known predictors of criminal behavior, lead exposure has been hypothesized to be an important determinant of crime. Recent psychological and neurotoxical research has showed strong association between childhood lead exposure and criminal behavior (e.g. Needleman et al. 1996: Dietrich et al. 2001; Wright et al. 2008). Yet, the results have been questioned on the basis of the use of small and unrepresentative samples, the inability to follow the subjects over a longer time-period[2], and failure to employ methods to control for the influence of confounders. The only studies that employ a designed-based identification strategy to study the link between childhood lead exposure and crime are Reyes (2007, 2014). Reyes (2007) shows that the state level reductions in childhood lead exposure in the United States which occurred in the late 1970s and early 1980s account for as much as 56 percent of the decline in violent crime observed in the last decades. Reyes made a novel contribution to the literature when attempting to control for the influence of confounders by using a state level panel dataset with information on the removal of lead from gasoline in the late 1970s under the Clean Air Act (CAA). The CAA provides a plausibly exogenous source of variation in childhood lead exposure.[3] Reyes (2014) uses a similar research design, but instead of state level data she studies a sample of about 8,000 children included in the National Longitudinal Survey of Youth (NLSY). She finds a strong positive effect of childhood lead exposure on self-reported crime as well as arrests and criminal convictions for children followed up to age 17.

Our paper adds to the previous literature in several ways. First and foremost, our study contributes by combining a policy-induced source of variation in childhood lead exposure with exposure levels much lower than what is typically used as a benchmark for intervention. While Reyes (2007, 2014) also exploits a quasi-experimental research design, she uses state level averages of the amount of lead in gasoline to measure childhood lead exposure. In contrast, we follow Nilsson (2009a) and measure lead exposure using moss (bryophyte) samples collected by the Swedish Environmental Protection Agency covering the whole of Sweden. Mosses are particularly useful as air pollution biomonitors since they lack roots and

[2] Needleman et al. (1996) study 301 first graders in Pittsburgh, Pennsylvania. Needleman et al. (2002) examine 194 children aged 12-18 also living in Pittsburgh, Pennsylvania. Dietrich et al (2001) examine 195 inner city adolescents from Cincinnati, Ohio. The study by Wright et al. (2008) involves 376 children also from Cincinnati, Ohio.

[3] Stretesky and Lynch (2004) also use aggregated data but at the county level. This study does not however control for county specific fixed effects. Nevin (2000) uses time-series data and document strong correlations.

therefore solely absorb heavy metal depositions from the air.[4] The measure of lead exposure we use has the advantage that it is provides a more localized measure (in about 1,000 locations throughout Sweden) and is therefore likely to provide a good proxy for actual exposure. Nilsson et al. (2009b) verifies that this measure of lead exposure is a strong predictor of blood lead levels in children.

We are able to study effects at relatively low levels of exposure. In 2012, the US Centre for Disease Control and Prevention (CDC) lowered their "limit of concern" from 10 μg/dL blood to 5 μg/dL.[5] In Sweden when average blood lead peaked in the early 1970s, average blood lead levels was already below 10 μg/dL blood, and in 1995, when lead was banned, the average blood lead level was around 2 μg/dL (Nilsson 2009a, Skerfving et al 2001). Although several recent psychological studies argue that even low to moderate blood lead levels may cause deficits in cognitive development (Canfield et al., 2003; Lanphear et al., 2000), no safe blood lead level in children has been identified, and there is no knowledge about the effect of childhood lead exposure on criminal behavior at the levels studied in this paper.[6]

Other contributions are linked to the unique individual data at hand. Our data contain information on every criminal conviction in Sweden since 1985 along with a broad set of individual and family characteristics. We study children in the 1972-74, 1977-79 and 1982-84 birth cohorts for whom we are able to link municipality of birth to moss lead levels. Our data allows for large representative samples and we are able to follow the 360,857 children up to age 24, essentially without any attrition. This is crucial as the age-crime profile peaks in the late teens and early twenties. Ignoring the period in life when crime is most frequent would

---

[4] The use of moss as biomonitors for ambient heavy metal air pollution is well established and the methodology is used today in many European countries.
[5] Approximately 450,000 children in the United States have blood lead levels above this reference value (CDC 2010).
[6] The cohorts studied in Reyes (2007) were estimated to have a blood lead level between 10 and 20 μg/dL. The average predicted childhood blood level in Reyes (2014) is about 7.6 μg/dL.

risk understating the total social costs (if any) of lead exposure. Data restrictions have until today prevented this type of analysis. Last, most previous studies originate from the United States. The Swedish context is particularly interesting to examine because it sheds light on the consequences of lead exposure in a setting that abstracts from differences in access to public safety nets which could exacerbate the implications of early initial health insults.

Our results show that childhood lead exposure is strongly associated with crime. The reduction in lead induced by the Swedish phase-out of leaded gasoline on average implied a significant 4.5 percent reduction in overall crime for males. Scaling this number with our estimated link between moss lead levels and children's blood lead levels (.57), i.e. the "first-stage", imply a 7.4 percent reduction in crime. This is by all measures a sizable effect. For instance, Meghir, Palme and Schnabel (2013) provide causal evidence that sons of fathers who were exposed to the Swedish compulsory schooling reform which prolonged school by about one year were 2.5 percent less likely to be convicted for a crime. The effect is larger both for property crime (10.5 percent) and for violent crime (14.3 percent). Our results also show large differences in the response by parental socioeconomic background. Children from poor socioeconomic conditions exposed to lead are substantially more likely to engage in crime compared to children from more affluent backgrounds.

In an auxiliary analysis we attempt to disentangle the potential channels through which childhood lead exposure is likely to matter for the decision to commit crime. We find that about two thirds of the effect size remains after controlling for compulsory school GPA. To the extent that compulsory school GPA provides a better proxy for cognitive skills rather than non-cognitive skills, these results may be interpreted as suggestive evidence that childhood lead exposure matters primarily because it affects non-cognitive skills. Various robustness checks confirm that the results are not likely to be driven by confounding factors such as exposure to other pollutants or parental sorting.

Finally, semi parametric-regressions provide clear evidence of non-linear effects in the relationship between childhood lead exposure and crime. Above a municipality average blood lead of about 5 micrograms/dl blood there is a clear positive effect of lead exposure on crime, while we find no evidence of a relationship below this level. This non-linearity has clear implications for the optimal design of policy initiatives to reduce lead exposure.

The rest of this paper is structured as follows. Section 2 discusses issues related to measuring lead exposure as well as describes our data. Section 3 presents our research design and the empirical results. Section 4 concludes.

2. Data

2.1 Measuring lead exposure in childhood[7]

To measure local lead exposure levels, we use data from the Swedish Environmental Protection Agency which since 1975 has monitored heavy metal air pollution every five years 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. On a national scale, the use of moss as air pollution monitors expanded to Norway and Finland in 1985. 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.

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

[7] This section draws on Nilsson (2009a).

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 allow 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 meters away from bigger roads and closed residential areas, or at least 100 meters from smaller roads and single houses. At each site 5 to 10 subsamples are collected in an area of approximately 100 square meters. 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 liter of gasoline decreased particularly sharply. The changes in maximum allowed gasoline lead levels are depicted in Figure 1. We can see that the maximum allowed lead level in gasoline was lowered in 1972 from 7 to 4 g/dl. This change applied to all types of gasoline. In 1980 (1981) there was an additional cut in allowed lead levels for regular (premium) gasoline from 4 to 1.8 g/dl. In 1986 (1995), lead was finally completely banned for regular (premium) gasoline. We can also see from Figure 1 that the total amount of lead in gasoline fell sharply throughout this period. Second, since the main outcomes focused on criminal behavior, it is necessary to restrict the sample to those cohorts that have reached the peak of their criminal-age profile 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. Hence, in order to construct a measure of municipality lead exposure we follow a similar approach as Neidell (2004) and Neidell and Currie (2004): first we calculate the centroid of each municipality. Then we measure the distance between the sampling site and the center of the municipality. Finally, we 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 displays the lead concentrations in the municipalities in 1975 and 1985 using this definition of exposure. We can see that there is a great deal of variation in local lead levels between municipalities. It is also clear that lead concentrations fell sharply between 1975 and 1985. This is also evident in Figure 3 which shows Box-Whisker plots of the distribution of municipality moss lead levels by year. It is clear that the entire distribution of municipality lead levels shifted dramatically and became more compressed. Another way to illustrate this is by means of a visual inspection of the distribution of the average changes in municipality moss lead levels that occurred between 1975 and 1985, as illustrated in Figure 4. We can see that most municipalities experienced decreases in moss lead levels by between 15 and 45 μg/Kg, with an average reduction between these years of 30 μg/Kg. A variance decomposition reveals that most of the reduction in lead exposure is due to within municipality rather than between municipality differences.[8] These sharp within municipality

[8] The overall standard deviation for our measure of lead exposure is .172 while the between and within standard deviation is .096 and .142, respectively.

differences in the reduction of early childhood lead exposure across the cohorts is a key feature of our research design.

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, we 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). Secondly, to get an idea on how accurate the five nearest sample approach is in predicting the actual exposure level, we 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, we estimate the air lead level at a given sampling point based on the air lead levels at the five nearest sampling points. We 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 indicates that the pollution assignment method employed provides reasonably accurate predictions of actual air lead levels.

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 biannually from about 120 primary school children (age 8-11) per year. The results from these studies on the trends in childhood lead exposure are described in detail in 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.

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. Figure 5 shows the evolution of tons of lead added to gasoline and mean blood-lead levels among primary school children in Sweden 1976-1996. Even a simple visual inspection of this graph shows that both measures are strongly correlated. Nilsson et al. (2009b) use regression-based methods to 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 characteristics, time and locality fixed effects they estimate 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 suggests 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.

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

2.2 *Other data sources*

To investigate the effect of childhood lead exposure on crime we use data from administrative registers collected and maintained by Statistics Sweden and the National Council for Crime Prevention. The data span the entire Swedish population aged 15 and above and include information on a wide range of labor market, educational and demographic characteristics of the individuals for the period 1985 to 2007. There is also information on all convictions in Swedish district courts during the period. This includes information on the type of crime as well as the exact date of the offense. A conviction may include several crimes and all crimes are recorded in the data. Speeding tickets, and other minor crimes not severe enough to warrant a trial are not covered. The analysis follows the established convention in the literature and focuses on the most common types of crimes: violent crime and property crime. Violent crime represents the full spectrum of assaults from minor assault to murder according to the penal code BRB Chapter 3. Property crime represents the full spectrum of thefts from shop-lifting to burglary and robbery according to the penal code BRB Chapter 8. We also study overall crime as measured by having any type of recorded criminal conviction.

Our sample consists of 360,857 individuals born in Sweden three years prior to the year the moss samples were collected: i.e. all individuals are those born in 1972-1974, 1977-1979 and 1982-1984. The analysis includes a range of standard individual background characteristics, such as gender, month of birth, number of siblings, parental educational attainment, parental age at birth, parental earnings, along with a host of municipality level characteristics: average birth weight, average gestation length, average maternal age at birth, population size, the share welfare recipients, the share single parents, the share convicted criminals, average disposable income, the unemployment rate, population size, and the share high educated individuals.[9] Importantly, the data allow us to identify each individual's municipality of birth. This is an important advantage compared to previous studies using

[9] Some of these variables are measured at age 15.

aggregated state level data which suffer from attrition due to (possibly selective) inter-state migration between the year of birth and the year when the crime rate is measured. There is also information on compulsory school grades in all subjects for individuals who finished school during the period 1988 to 2007.[10] We use this information in an attempt to unpack the mechanisms through which childhood lead exposure may affect crime.

3. Results

We start by describing our research design and then provide the main estimation results along with robustness tests. We continue by presenting evidence of potential non-linear effects. Then we show results from supplemental analyses and further robustness tests. This section ends with presenting results from estimations where we stratify the sample by parental socioeconomic background.

3.1 Main results

To identify the effect of childhood lead exposure on crime we use variations of the following baseline regression model

$$Crime_{imc}^{\leq 24} = \alpha + \beta Lead_{mc} + \gamma X_i + \delta Z_{mc} + \theta_m + \rho_c + \varepsilon_{imc}$$

where $Crime \leq_{imc}^{24}$ is a dummy set to unity if individual i, born in cohort c and municipality m, was convicted for a crime up to age 24, zero otherwise.[11] $Lead_{mc}$ is moss lead levels measured in terms of micrograms per kilogram moss. This measure varies for individuals born in different years and in different municipalities. X_i and Z_{mc} represent vectors of

[10] To account for changes in the grading system over time as well as potential grade inflation, grades are computed as the percentile rank by year of graduation.

[11] Since 15 is the age of criminal majority in Sweden we are not able to study criminal convictions prior to that age.

individual and municipality level covariates described earlier. θ_m denote a set of municipality (of birth) fixed effects. ρ_c represent year of birth fixed effects. This model amounts to a standard differences-in-differences estimator where $\hat{\beta}^{OLS}$ should be interpreted as the effect of childhood lead exposure on adolescent and adult crime. As described earlier, the variation in lead exposure largely stems from the phase-out of leaded gasoline. We cluster the standard errors at the municipality level to account for arbitrary serial correlation and heteroscedasticity.

Table 1 presents the main results. Each table entry corresponds to a separate regression. We show results by type of crime and successively add more covariates to the model. In the first row we only control for individual level characteristics. We can see that all estimates are significant at conventional levels. Evaluating the estimates with the decrease in moss lead that occurred between 1975 and 1985 after the phase-out (30 μg/Kg) shows that the risk of crime decreases by about 4.25 percent ((.000336*30)/0.237)). For property and violent crime the corresponding numbers are 6.1 and 8.2 percent respectively. It is important however to remember that the air measures of municipality lead concentrations used in this study does not perfectly predict blood lead levels. This means that we cannot directly interpret the estimates as representing the effect of actual childhood lead exposure on crime. Fortunately we can use the estimated Blood-moss lead elasticity provided by Nilsson (2009b) to assess the effect of actual childhood lead exposure on crime. In this exercise it is also important to take into account the fact that the 'first-stage' blood-moss relationship has been estimated to be about 30 percent stronger among children age 0-6 (Wolpaw Reyes 2009). It turns out that scaling our estimated effect for overall crime with the Blood-moss lead elasticity in Nilsson (2009b) (.44) and also taking into consideration the fact that the relationship between moss lead level and blood level is higher among children aged 0-6 suggest a 7.4 ((.0425/(.44*1.3)) percent decrease in overall crime. The analogous figures for property and violent crimes are larger: a 10.5 and an 14.3 percent decrease, respectively.

There are three main threats to our identification strategy. First, it is possible that municipality moss lead levels simply proxy for unobserved municipality characteristics not captured by the municipality fixed effects. This could for instance be changes in the local business cycle or various difficult-to-measure local amenities that are associated with both neighborhood lead levels and with crime (e.g. Bellinger, 2004). To investigate the likelihood that such omitted variables are driving our results we add to our baseline model a wide set of municipality characteristics. If the estimates substantially changes when including potential important covariates such as the unemployment rate, average income or population size then caution is warranted when interpreting the estimates. It is reassuring that the estimates are virtually unchanged by this exercise, see Table 1.

Another potential threat is that municipality lead levels may be correlated with other pollutants that also increase the risk of criminal involvement. 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 other common heavy metal pollutants it is easy to do an initial assessment of the potential severity of this problem. Of all the observable heavy metal pollutants in the data, only cadmium (Cd) displays even nearly as large and widespread changes during the observation period as lead does. Cd has previously been found to be associated with adverse health outcomes (kidney damage, bone disease). Early exposure to Cd has been shown to be able to produce neurotoxic effects in laboratory experiments (Anderson et al., 1997; Peterson et al., 2004), and in a recent study Cd air releases are shown to affect infant health in humans (Currie and Schmeider, 2009). Moreover, air Cd and air lead concentrations display a fairly high correlation at the municipality level. 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 Cd exposure rather than local air lead exposure. This is so since unlike lead, the primary exposure route of Cd is dietary rather than respiratory (WHO, 1972; IPCS, 1992; Moon et al., 2003; Ohlsson et al., 2005). The data also includes information on levels of Copper (Cu) and Zinc (Zn). Cu and Zn have been suggested to be used as marker elements for motor vehicle emissions in previous studies (e.g. Huang 1994), and are therefore useful to include in the analysis to control for traffic density.[12]

For overall crime and for property crime, the size of the coefficient in Table 1 is almost unchanged when adding controls for Cd, Cu and Zn, but the estimate for violent crime falls just below the 10 percent significance level. It therefore seems as if a small part of the effect of childhood lead exposure on violent crime operates through other traffic related pollutants. Overall, however, essentially the entire lead-crime relationship survives this exercise and the results still suggest that childhood lead exposure substantially increases the risk of crime.

The third potential concern for our identification strategy is the risk of selective migration. If parents sort into residential location based on factors associated with lead pollution levels and if such factors also are linked to the likelihood of criminal behavior among their children then our estimates could reflect the influence of parental background. Since local lead levels are not likely to be known by the parents we find it implausible that this type selective sorting took place. In the case such Tiebout sorting actually does occur, the most important predictor of residential location is probably socioeconomic status. Parental socioeconomic status has also been shown to be closely related to crime among their children (e.g. Meghir, Palme, and Schnabel 2013).

---

[12] Brake linings are the major source of road traffic emitted Cu. Zn concentrations are high in tiers.

We assess the risk of parental sorting by running regressions where pre-determined variables reflecting parental socioeconomic status are used as dependent variables and regressed on childhood lead exposure. We do this both with and without municipality fixed effects. The results are displayed in Table 2. We can see that, in the cross-section, parental socioeconomic status is strongly associated with childhood lead exposure. In fact, all estimates are strongly statistically significant. Controlling for municipality fixed effects, however, reduces the magnitude of all coefficients substantially. Seven out of eight estimates become statistically insignificant.[13] We believe that this results shows: (1) the importance of controlling for municipality fixed effects; (2) that parental sorting is not likely to pose a serious threat to our identification strategy, at least not after the removal of unobserved heterogeneity across municipalities.

3.2 Non-linear effects

After having shown that childhood lead exposure is strongly related to subsequent criminal behavior and that these results passes various sensitivity checks, we next ask whether the relationship is linear or not. Remember that the estimates presented above should be interpreted as the average effect of changes in childhood lead exposure. One possibility is however that the effect is weaker below some level of lead exposure. 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. Reyes (2007) find no or only weak nonlinearities in the lead exposure-violent crime relationship, but the average blood lead levels in her sample were considerably higher than in the present study. The fact that we find large effect sizes at low doses suggests

[13] Note also that the sign on the coefficient switches for father's high school.

that further lowering the governmental safety levels for lead exposure is warranted. Still, knowledge of whether there exists some threshold level under which the effect size weakens is important for optimally designing environmental policy.

To investigate whether the relationship between childhood lead exposure and crime is non-linear we employ the semi parametric estimator proposed by Robinson (1988).[14] The results are shown graphically in Figure 6. For any crime the effect is close to linear in childhood lead exposure. When estimating the model separately for property crime, however, there is a clear kink around 60 µg/Kg. A similar but less pronounced pattern is also shown for violent crime.[15] These results suggest that reductions in lead exposure levels below 60 µg/Kg have no noticeable effects on crime, whereas reductions above this threshold level has a clear impact. How does this moss-lead threshold level translate into children's blood lead levels? Nilsson et al. (2009) present empirical evidence on the regression adjusted link between moss lead levels and blood lead concentrations. It turns out that plugging in our estimated threshold value of 60 µg/Kg into the model and evaluating the estimates at sample means produces a predicted blood level threshold of 5.45 µg/dL blood.[16] As already mentioned, close to half of all children worldwide have blood lead concentrations above this threshold and this fraction is even larger in developing countries. Moreover, the CDC recently estimated that around 450,000 children in the United States today have blood lead levels that exceed this threshold (CDC 2010).

---

[14] The approach is a two-step method where the first step estimates $E(Crime|X,Z,\theta,\rho)$ and $E(Crime|Lead)$ and then secondly allows nonparametric estimation of the impact of the latter on the former. The coefficient β can then be obtained from a regression of the residuals $\zeta_1 = Crime - \delta_1 X - \delta_2 Z - \delta_m - \delta_c$ on $\zeta_2 = Lead - \rho_1 X - \rho_2 Z - \rho_m - \rho_c$. Our preferred specification here, as well as in the remainder of this study, is the one on row 3 in Table 1that includes other pollutants as well as municipality and individual level covariates.

[15] The fact that there is no clear threshold for any crime is perhaps not so surprising given that the bulk of crime that falls into this category are minor infractions and crimes that are not directly related to violence or financially motivated crimes. For instance, traffic offenses is by far the most common type of crime in the data. Other crimes that falls into this category include violations against the hunting law and various tax crimes. Since these crimes are not likely to be affected by childhood lead exposure they will not contribute to identifying threshold effects.

[16] See Nilsson et al. (2009b) for more details.

3.3 Extensions and further sensitivity checks

We next show results from some auxiliary analyses. First, it is possible that childhood lead exposure has different implications for the decision to commit crime at the intensive margin rather than at the extensive margin. When evaluating the estimates presented in Table 3 with the decrease in moss lead that occurred between 1975 and 1985 and scaling them as earlier we find that the estimates are in fact stronger when we study crime at the intensive margin. For any crime, the estimate implies a 9.6 percent decrease (not statistically significant), for property crime a 19.1 percent decrease, and for violent crime a 13 percent decrease. Note that the decrease in violent crime which was statistically insignificant at the extensive margin is now significant at the five percent level. Recall that Reyes (2007) finds remarkably large effects of childhood lead exposure on crime using state level crime data. The fact that we find that the effect of childhood lead exposure on crime is larger at the intensive than at the extensive margin suggests that childhood lead exposure matters more for the aggregate crime rate because it induces relatively few individuals to commit substantially more crime rather than increasing the overall number of criminals. Still, it is important to remember that the effect sizes we find for crime at the extensive margin are by no means trivial.

We also provide brother fixed effects estimates. This is possible since the data include a complete linkage between all biological parents and their children. Since siblings in most cases grow up in the same municipality this estimator absorbs most municipality level confounders. Identification therefore mostly stems from the fact that lead levels have changed in the municipality of residence from the time the first child was born until the birth of his brother.

Column (3) in Table 3 shows that, if anything, the brother fixed effect estimates are larger compared to baseline for any type of crime and for property crime. One interpretation consistent with this pattern could be that parents reinforce initial disadvantages by reallocating investments to the less exposed sibling (e.g. Behrman, Rosenzweig and Taubman 1994). Another possibility is that going from a within-municipality to a within-family type of analysis changes the way errors in our measure of lead exposure influences our estimates since altering estimator implies changing the source of variation used for identification. A third explanation, which we find less likely, is that the sibling fixed effects estimators picks up unobserved characteristics related to parental sorting since some siblings will grow up in different municipalities.

The two main mechanisms that through which childhood lead exposure might matter is by affecting cognitive skills or by influencing non-cognitive skills such as attention, aggression, impulsiveness etc. In an attempt to tease out the most likely channel we exploit the fact that our data contain information on compulsory school grades. Our idea is that compulsory school grades are likely to provide a better proxy for cognitive skills than for non-cognitive skills. Controlling for compulsory school grades in the regressions therefore provides a crude way to assess the relative importance of these different mechanisms. We can see that the estimates fall for all types of crime when conditioning on compulsory school GPA in the regressions. The magnitude of the decrease is about one third of its original size. Under the assumption that compulsory school GPA is a better proxy for cognitive rather than non-cognitive skills this suuggests that at least part of the effect is likely to operate via cognitive skills. Yet, the estimates are still statistically significant and large in magnitude. It therefore seems as if the most important channel through which lead exposure matters is by influencing non-cognitive skill.

So far we have followed the convention in the literature to focus the analysis on males. This is plausible since males account for the bulk of all crimes committed. It is however relevant also to ask how childhood lead exposure affects crime among females. These results can be found in column (5). As can be seen, all estimates are close to zero and statistically insignificant. This result is consistent with Reyes (2014) who finds that separating boys and girls reveals that for the most part the results are driven by the boys.[17] It has been speculated that males are more sensitive to lead exposure than females (Barett 2009). Another potential explanation for this finding is that female risky behavior may manifest itself in other ways than criminal acts, e.g., through risky sexual behavior.

We also note that our results do not seem sensitive to our choice of a linear probability model. As can be seen, the average marginal effects from using a Probit model closely resembles the OLS estimates. We have also estimated models where the data were collapsed at the municipality-cohort level to alleviate concerns relating to statistical inference due to the fact that our lead exposure measure varies at the municipality-cohort level while we use individual level outcome data (note though that the standard errors are clustered at the municipality level). Reassuringly our baseline estimates are highly similar and statistically significant when aggregating the data to the municipality level

3.4 Differential effects across socio-economic groups

It is possible that children of parents with low socioeconomic status may be differentially affected from a given level of exposure compared to children of parents from more affluent backgrounds. Socioeconomically disadvantaged parents may for instance have fewer resources to compensate for health insults. In Table 4 we separate between parents' incomes and highest completed level of education. Low (high) family income refers to families with no (at least one) parent having above median annual earnings in the year the child turns 15. Low (high) educated parents refer to having no (at least one) parent with university education. To

[17] For the cognitive skills outcomes the evidence is mixed. Several correlational studies document that females seem to respond less than males for a given exposure level (e.g. Barrett 2009). However, Nilsson (2009a) using the same source of variation in lead exposure as in this study find no clear gender differences in the impact on cognitive skills, or educational outcomes.

the best of our knowledge, this is the first study of the effect of childhood lead exposure on crime where the data allow for a separate analysis by parental socioeconomic background.

We can see that the coefficients are substantially larger for children of low income parents. The estimate for violent crime is not statistically significant, but is more than twice as large as the one for children of high income parents. A similar picture appears also when stratifying the sample by parental education. The estimates are larger for children of low educated parents when looking at any type of crime as well as when looking at violent crime, the latter estimate being statistically significant. In fact, the entire lead-violent crime relationship appears to be driven by children of low educated parents. There is however no statistically significant effect for property crime. Taken together, it is clear that children from more socioeconomically disadvantaged conditions respond stronger to childhood lead exposure compared to children from more advantaged backgrounds.

4. Concluding remarks

This paper examines the effect of childhood lead exposure on criminal behavior. We take advantage of repeated exact measures of childhood lead exposure from moss samples for the whole of Sweden and rich population based administrative data allowing us to trace the subjects up to 24 years post exposure. The analysis combines these unique data with policy induced variation in lead exposure at levels lower than what is normally used to call for intervention. Taken together, these features of our research design allows us to shed light on several new aspects of the link between childhood lead exposure and crime.

We find that childhood lead exposure is strongly associated with crime. Our estimates suggest that the reduction in lead induced by the Swedish phase-out of leaded gasoline imply reductions in crime by between 7.4 percent reduction in crime and 14.3 percent, although there is no evidence that childhood lead exposure affects criminal behavior among females.

We also find striking differences in the response by parental socioeconomic background. For a given level of exposure, children from poor socioeconomic conditions are more likely to engage in crime compared to children from more affluent. We also provide tentative evidence that the effect of childhood exposure on crime to a larger extent seems to be driven by non-cognitive skills rather than cognitive skills.

We provide evidence of non-linear effects in lead exposure on property crime and violent crime. Below an estimated municipality level average blood lead level of around 5.5 microgram/dl blood there is no relationship between lead exposure and crime. Since WHO estimate that around 50% of the children globally have blood lead levels above this threshold our results suggests that reducing lead exposure in small children could have a non-negligible impact on crime-rates. This may be especially important in developing countries where the level of lead exposure among children is even larger. It is however important to stress that the estimated level corresponds to municipality level averages. Hence, while suggestive, this threshold needs to be confirmed in future studies with access to individual blood lead level data in early childhood, criminal behavior later in life *and* a credible empirical strategy.

References

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.

Banks, E.C., L.E. Ferretti and D.W. Shucar. 1997. "Effects of Low Level Lead Exposure on Cognitive Function in Children: A Review of Behavioral, Neuropsychological, and Biological Evidence." *Neurotoxicology*, 18(1), 237-81.

Barret, J. (2009), “Children’s Health: Sex-Specific Cognitive Effects of Lead”, *Environmental Health Perspectives*. 117(9): pp. A393.

Behrman, J., M. Rosenzweig, and P. Taubman (1994), “Endowments and the allocation of schooling in the family and in the marriage market: the twins experiment,” *Journal of Political Economy*, pp. 1131–1174.

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

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.

Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentrations below 10 micrograms per deciliter. N Engl J Med. 2003;348:1517–1526

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.

CDC (2010), Centres for Disease Control and Prevention, “*Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention*”

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), pp 1003-1030, August.

Currie, J., J. Schmieder (2009) “Fetal Exposure to Toxic Releases and Infant Health,” (2009), *American Economic Review: Papers and Proceedings, 99(2): 177-183*.

Dietrich, Kim N., Ris M. Douglas, Paul A. Succop, et al. 2001. "Early Exposure to Lead and Juvenile Delinquency." *Neurotoxicol Teratol*, 23(6), 511-18.

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

Etzel R.A. (2003). American Academy of Pediatrics Committee on Environmental Health.Pediatric Environmental Health, 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics, 2003.

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.

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.

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

National Research Council1993. Measuring Lead Exposure in Infants, Children and Other Sensitive Populations. Washington, DC:National Academy of Sciences.

Needleman, Herbert. L. 1979. "Lead levels and children's psychologic performance." *New England Journal of Medicine*, 301(3), 163.

Needleman, Herbert L., J. A. Riess, M. J. Tobin, et al. 1996. "Bone Lead Levels and Delinquent Behavior." *Journal of the American Medical Association*, 275(5), 363-9.

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.

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

Nilsson, J. P. (2009a), “ The Long-term Effects of Early Childhood Lead Exposure: Evidence from the Phase-out of Leaded Gasoline”, Chapter included in doctoral thesis, Department of Economics, Uppsala University

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

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

Matte, T. (1999), ”Reducing Blood Lead Levels: Benefits and Strategies”, *Journal of American Medical Association*, Vol 281, No. 24.
IPCS (1992), International Programme on Chemical Safety *Environmental health criteria 134. Cadmium*., Geneva: World Health Organization.

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.

Meghir, C., Palme, M. and M. Schnabel (2012), “The Effect of Education Policy on Crime: An Intergenerational Perspective”, NBER Working Paper 18145.

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.

Rau, Tomas, Loreto Reyes and Sergio Urzua. 2012. "Early Lead Exposure and Its Effects on Academic Achievement and Earnings: Evidence from an Environmental Negligence." NBER Working paper No.18915

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

Wolpaw Reyes, J. (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

Wolpaw Reyes, J. (2011), "Childhood Lead and Academic Performance in Massachusetts." *New England Public Policy Center Working Paper*, 11(3).

Wolpaw Reyes, J. (2014),” Lead Exposure and Behavior: Effects on Antisocial and Risky Behavior among Children and Adolescents”, NBER Working Paper No. 20366

Robinson P.M. 1988. Root-N-consistent semiparametric regression. Econometrica 56: 931-954.

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.

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. 52

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.

Wright, John Paul, Kim N. Dietrich, M. Douglas Ris, et al. 2008. Association of Prenatal and Childhood Blood Lead Concentrations with Criminal Arrests in Early Adulthood." *PLoS Med*, 5(5), e101.

Weiss, B., Elsner, J., 1996. The intersection of risk assessment and neurobehavioral toxicity. *Environ. Health Perspect*.104 (Suppl 2), 173 – 177.

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

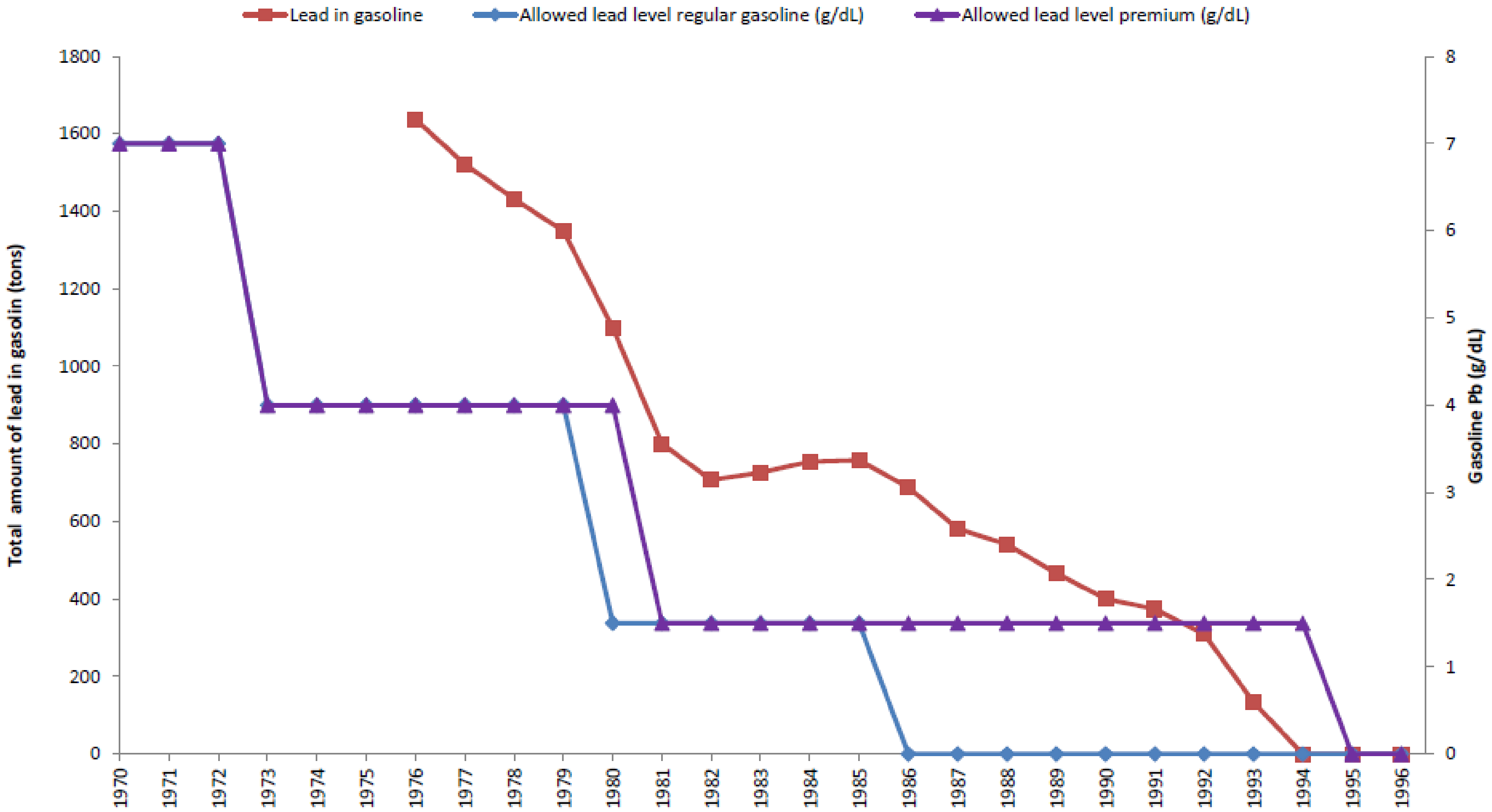

Figure 1. Changes in maximum allowed lead levels in Sweden by type of gasoline and the total amount of lead added to gasoline 1970-1996.

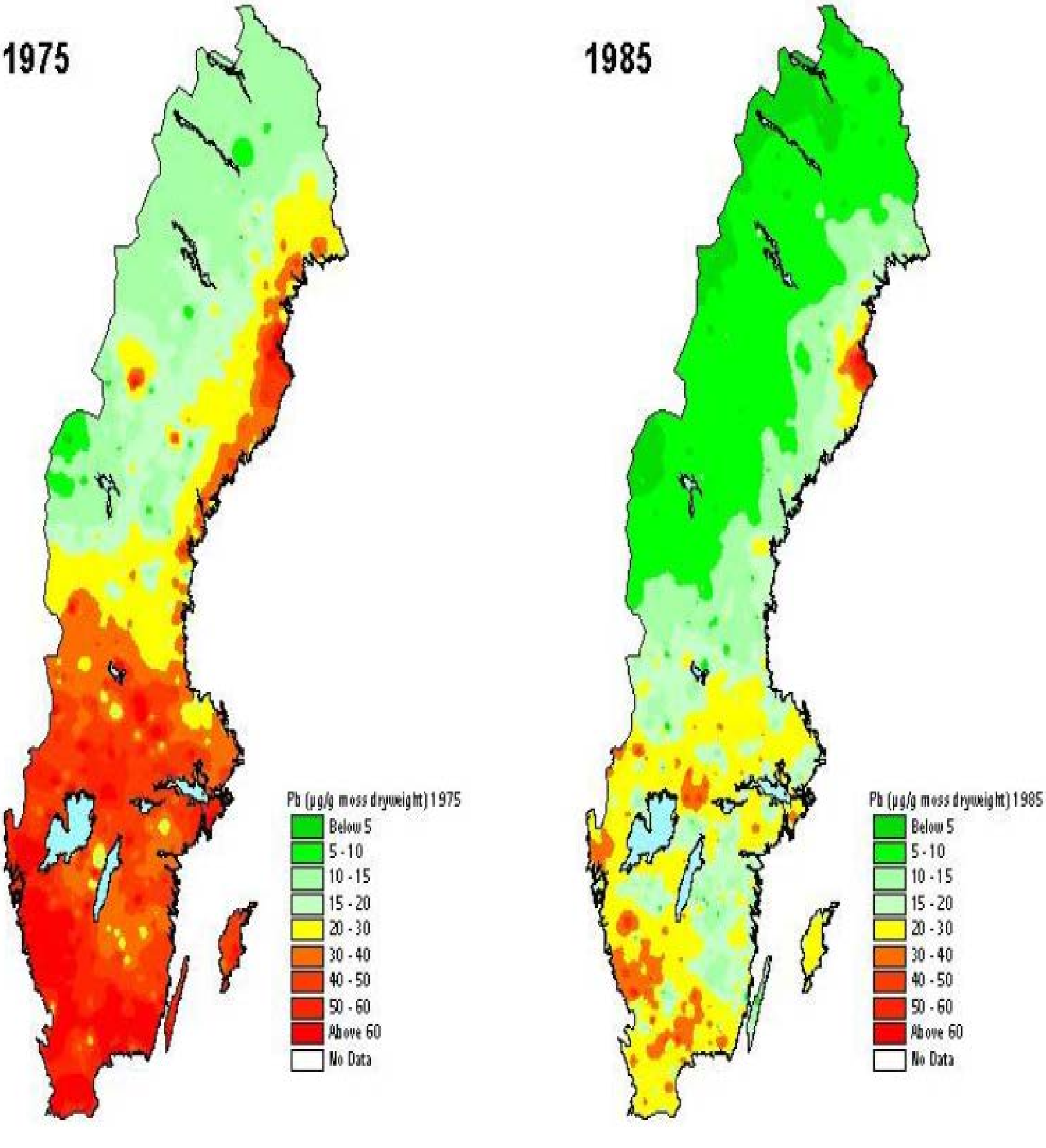

Figure 2 Moss lead levels (µg/Kg) in Swedish municipalities in 1975 (top map) and 1985 (bottom map). Source: Swedish Environmental Research Institute (IVL).

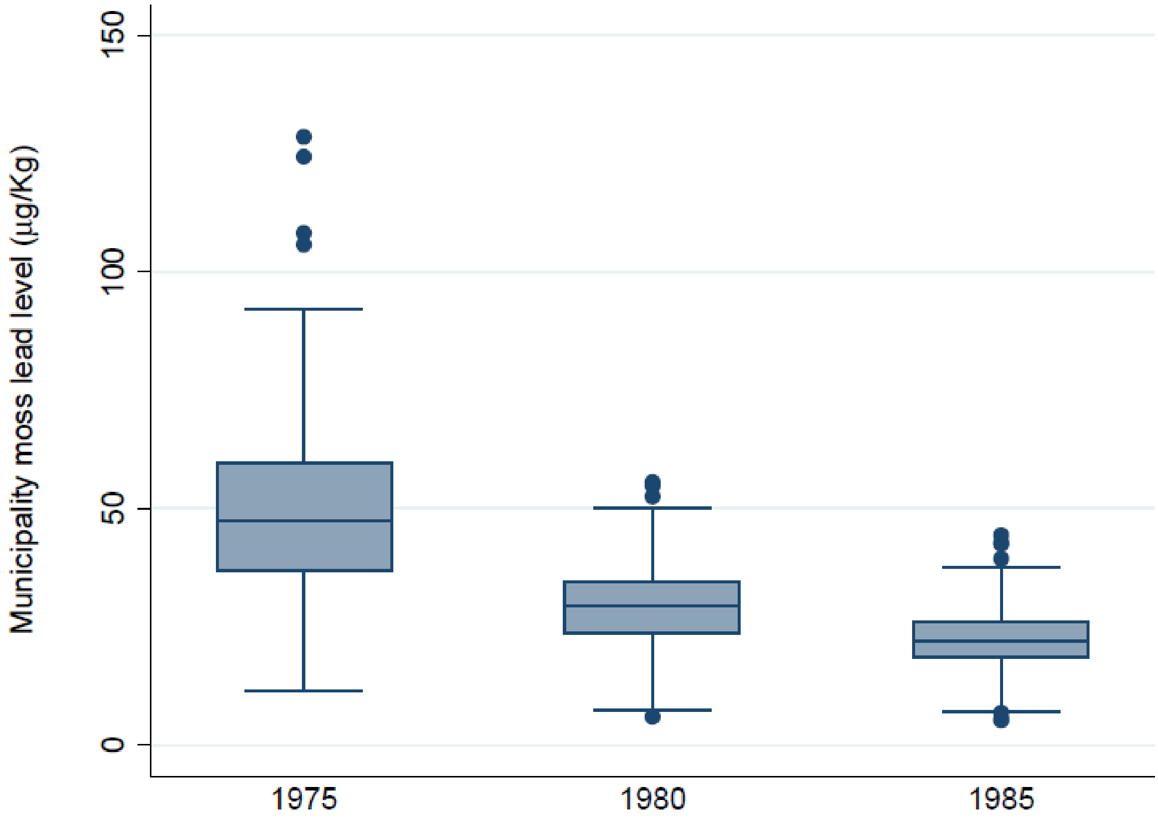

Figure 3. Box-Whisker plots of the distribution of municipality moss lead levels by year

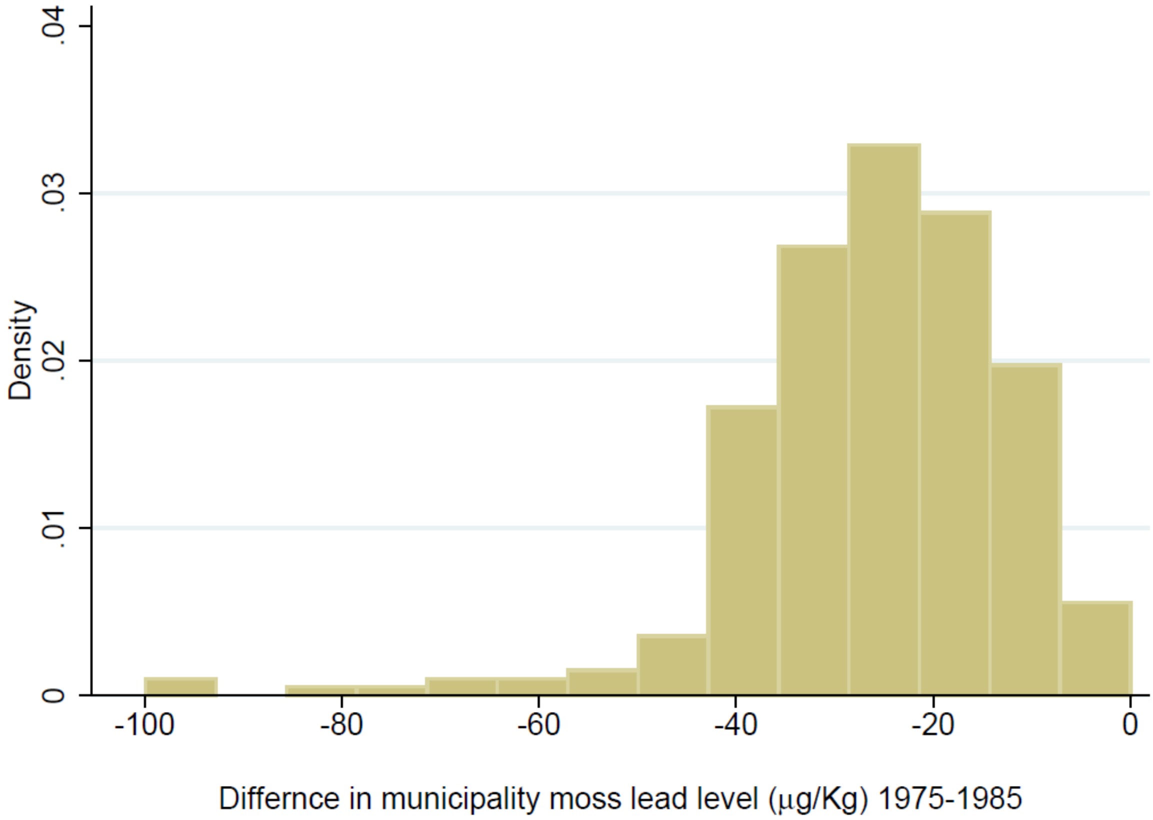

Figure 4. Distribution of the average changes in municipality moss lead levels between 1975 and 1985.

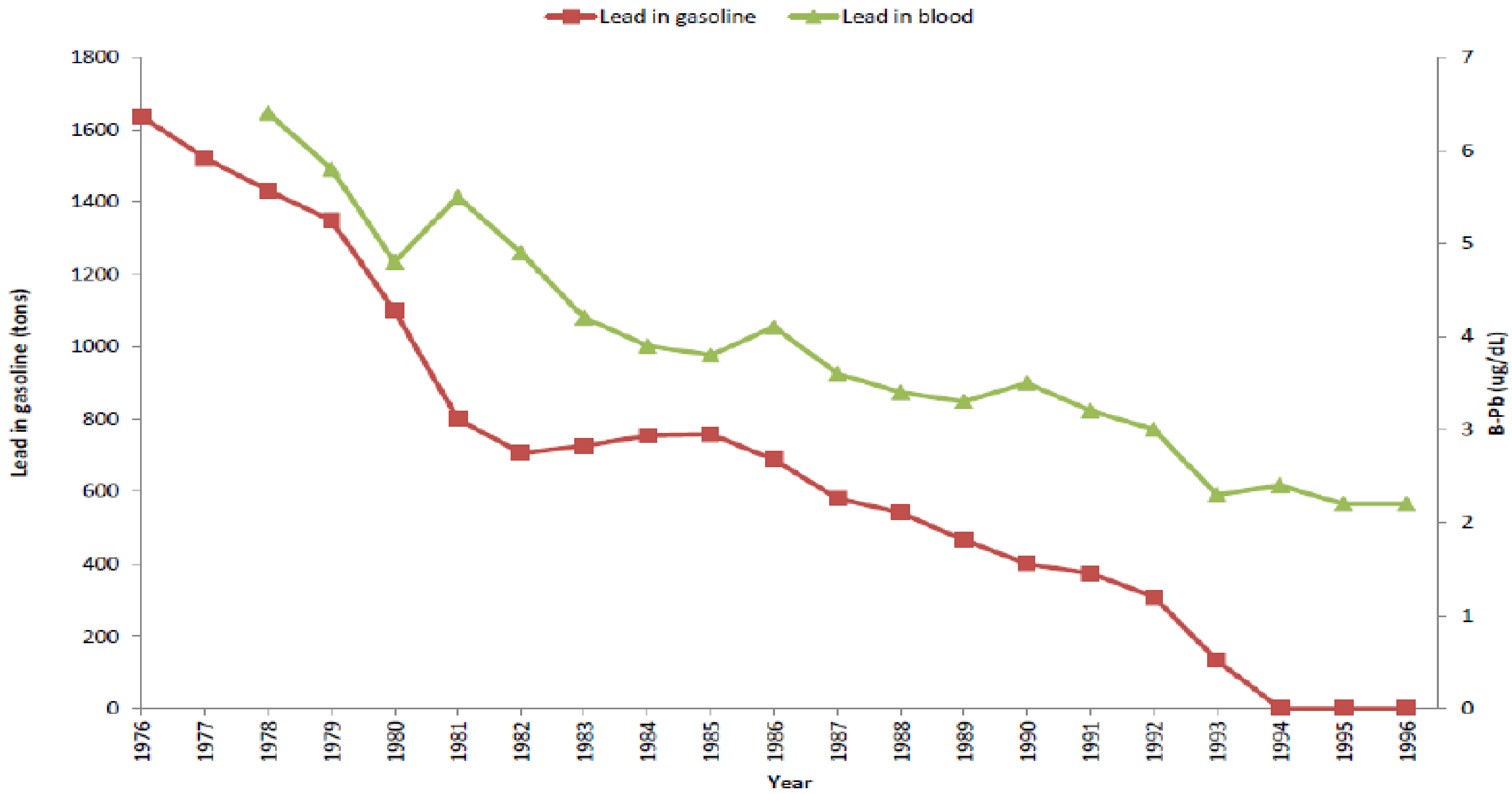

Figure 5. Evolution of tons of lead added to gasoline and mean blood-lead levels among primary school children in Sweden 1976-1996. Source: Stromberg et al. (1995) and Stromberg et al. (2003)

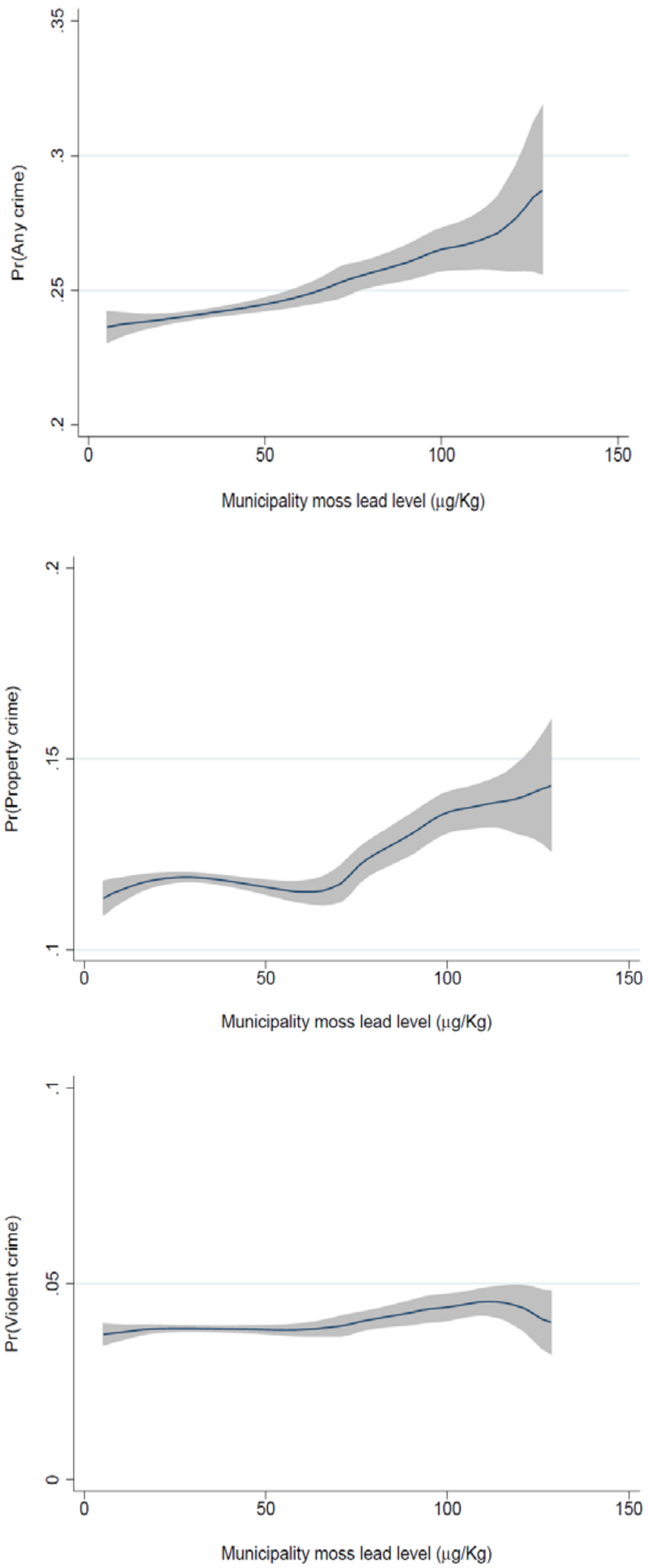

Figure 6. Semiparametric regression of the link between crime and municipality moss lead levels using the partially linear semiparametric regression estimator proposed by Robinson (1988)

Table 1. Estimates of the effect of childhood lead exposure on crime

	Any crime [23.7%] (1)	Property crime [11.9%] (2)	Violent crime [4.1%] (3)
Baseline	.0336***	.0241**	.0112**
	(.0085)	(.0031)	(.0050)
+ Controlling for municipality characteristics	.0354***	.0223**	.0100**
	(.0093)	(.0091)	(.0046)
+ Controlling for Cadmium, Zink and Copper	.0317**	.0220**	.0070
	(.0105)	(.0095)	(.0046)
Municipality FEs	Yes	Yes	Yes
Year of birth FEs	Yes	Yes	Yes

Notes: All coefficients are from separate OLS regressions. All numbers are scaled by a factor 100. The dependent variable is a dummy for having been convicted for a crime at least once between age 15 and 24. The sample consists of males born in 1972-1974, 1977-1979 and 1982-1984. Childhood lead exposure is measured as an average over age 1-3. Sample means expressed in percent are shown in brackets. The baseline regressions control for age, parental education, parental age, and parental disposable income. Cluster robust standard errors (at the municipality level) are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % .

Table 2. The correlation between pre-determined parental characteristics and their children's lead exposure

	(log) Parental income (1)	Parents high school (2)	Parents College (3)	Mother's College (4)	Mother's High school (5)	Father's College (6)	Father's High school (7)	Teen mom (8)
Coefficient on lead exposure *without* muni. FEs	0.0258	-0.111**	-0.0426*	-0.0570*	-0.111***	-0.0528*	-0.0763**	-0.0206**
	(0.0324)	(0.0353)	(0.0252)	(0.0321)	(0.0264)	(0.0305)	(0.0313)	(0.0086)
Coefficient on lead exposure *with* muni. FEs	0.0093	0.0040	-0.00810	0.00800	0.00177	-0.000639	0.0247*	-0.00347
	(0.0170)	(0.0148)	(0.0150)	(0.0174)	(0.0140)	(0.0167)	(0.0144)	(0.0078)
Cohort FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Muni. char.	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Cd, Cu, Zn	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: All coefficients are from separate OLS regressions. The sample consists of males born in 1972-1974, 1977-1979 and 1982-1984. Childhood lead exposure is measured as an average over age 1-3.All regressions control the child's cohort size and birth year fixed effects. Cluster robust standard errors are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % .

Table 3. Robustness and some extensions

	Baseline	Crime at the intensive margin	Sibling FE	Controlling for comp. school GPA	Girls	Probit
	(1)	(2)	(3)	(4)	(5)	(6)
Any crime	.0317**	.1370	.0517**	.0225**	.0114	.0316***
	(.0105)	(.0093)	(.0262)	(.0101)	(.0083)	(.0100)
	[23.7%]	[0.750]	[23.3%]	[23.7%]	[7.9%]	[23.7%]
Property crime	.0220**	.0960*	.0492**	.0160*	.0079	.0202**
	(.0095)	(.0038)	(.0223)	(.0084)	(.0057)	(.0081)
	[11.9%]	[0.263]	[11.9%]	[12.1%]	[5.1%]	[11.9%]
Violent crime	.0070	.0144**	-.0030	.0045	-.0007	.0077*
	(.0046)	(.0071)	(.0121)	(.0043)	(.0016)	(.0047)
	[4.1%]	[0.058]	[4.1%]	[4.1%]	[0.6%]	[4.1%]
Municipality FEs	Yes	Yes	Yes	Yes	Yes	Yes
Year of birth FEs	Yes	Yes	Yes	Yes	Yes	Yes
Municipality controls	Yes	Yes	Yes	Yes	Yes	Yes
Cd, Cu and Zn controls	Yes	Yes	Yes	Yes	Yes	Yes

Notes: All coefficients are from separate OLS regressions. All numbers are scaled by a factor 100The dependent variable is a dummy for having been convicted for a crime at least once between age 15 and 24. The sample consists of males born in 1972-1974, 1977-1979 and 1982-1984. Childhood lead exposure is measured as an average over age 1-3. Sample means expressed in percent are shown in brackets. The baseline regressions control for age, parental education, parental age, and parental disposable income. Sample means expressed in percent are shown in brackets. Cluster robust standard errors (at the municipality level) are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % .

Table 4. Differential effects of childhood lead exposure on crime by parental education

	Baseline (1)	Low family income (2)	High family income (3)	Low educated parents (4)	High educated parents (5)
Any crime	.0317**	.0458**	.0189	.0311**	.0284*
	(.0105)	(.0147)	(.0130)	(.0145)	(.0153)
	[23.7%]	[28.0%]	[19.5%]	[26.9%]	[17.1%]
Property crime	.0220**	.0353**	.0091	.0205	.0261**
	(.0095)	(.0153)	(.0085)	(.0128)	(.0101)
	[11.9%]	[15.3%]	[9.0%]	[13.9%]	[8.1%]
Violent crime	.0070	.0099	.0040	.0128**	-.0013
	(.0046)	(.0077)	(.0047)	(.0059)	(.0050)
	[4.1%]	[5.5%]	[2.7%]	[5.0%]	[2.1%]
Municipality FEs	Yes	Yes	Yes	Yes	Yes
Year of birth FEs	Yes	Yes	Yes	Yes	Yes
Municipality controls	Yes	Yes	Yes	Yes	Yes
Cd, Cu, Zn controls	Yes	Yes	Yes	Yes	Yes

Notes: All coefficients are from separate OLS regressions. All numbers are scaled by a factor 100. The dependent variable is a dummy for having been convicted for a crime at least once between age 15 and 24. The sample consists of males born in 1972-1974, 1977-1979 and 1982-1984. Low (high) educated parents refer to having no (at least one) parent with short university education. Low (high) family income refers to families with no (at least one) parent having above median annual earnings. Childhood lead exposure is measured as an average over age 1-3. Sample means expressed in percent are shown in brackets. The baseline regressions control for age, parental education, parental age, and parental disposable income. Cluster robust standard errors (at the municipality level) are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % .