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Early lead exposure and outcomes in adulthood

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Early lead exposure and outcomes in adulthood[1]

by

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Abstract

We exploit the phase-out of leaded gasoline to isolate the impact of early childhood lead exposure on outcomes in adulthood. By combining administrative data on school performance, high school graduation, crime, earnings, and cognitive and non-cognitive skills with a novel measure of lead exposure, we follow 800,000 children from birth into adulthood. We find that reduced lead exposure improves the adult outcomes, particularly among boys. Below certain thresholds, the relationship becomes much weaker. Non-cognitive traits (externalizing behavior, conscientiousness, and neuroticism) follow a similar non-linear dose-response pattern and seem to be the key mediators between early lead exposure and adult outcomes.

Keywords: Environmental policy, human capital; crime, non-cognitive skills.
JEL-codes: I18, K42, Q53.

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1 Introduction

Lead (Pb) is extremely toxic and high levels of exposure are well documented to cause adverse and often irreversible health effects and even death. Today, exposure causing clinical symptoms is relatively rare, primarily due to policies first limiting and finally banning lead from gasoline. However, the general population is still exposed to lower diffuse doses of lead from a variety of sources including food, air, toys, contaminated soils, lead-based paint, and water.[5] Recently, in Flint, Michigan, exposure due to deteriorating lead water pipes was made salient when an estimated 6,000 to 12,000 children experienced increased lead exposure, and the share of children aged below 6 that tested positive for elevated blood lead levels (EBLL) more than doubled (Hannah-Attisha et al. 2016). Globally, EBLL is still common: WHO estimates that in 49 percent of all children the blood-lead (B-Pb) level exceeds five micrograms per deciliter blood (5 µg/dL), i.e., the reference value for elevated blood lead established by the Centers for Disease Control and Prevention (CDC) in 2012.

Lead exposure is believed to be particularly harmful to infants and toddlers because of their rapidly developing central nervous system, increased exposure due to hand-to-mouth activities and, conditional on exposure, a higher absorption rate (Etzel 2003). But, understanding whether, and at what level of exposure, lead is negative for long-run human capital outcomes has proven challenging for several reasons. One problem is data availability. The ideal dataset would be a long and representative panel with measures of lead exposure in early childhood, relevant adult outcomes such as high-school completion, criminal behavior, and labor market outcomes, as well as direct measures of the mediating inputs (cognitive and non-cognitive skills) in the production of human capital leading up to outcomes in adulthood. In most countries, however, the required data does not exist. Another major challenge is the threat to identification from correlated unobservables. While it is known that children experiencing high levels of lead exposure have lower cognitive and non-cognitive skills and worse adult outcomes, these correlations could be driven by unobserved characteristics as opposed to early lead exposure.[6]

[5] For example, the Department of Housing and Urban Development estimates that 38 million homes constructed before 1978 still contain lead based paint.

[6] Given that lead levels are not randomly distributed across neighborhoods, any study estimating the effects of lead exposure needs to address the concern of confounding factors. For example, parents with higher incomes or preferences for cleaner air are likely to sort into areas with better air quality. Failing to account for residential sorting

Due to these challenges, evidence on the causal effects of low levels of lead exposure on adult outcomes is scarce. In the 1970s and 1980s, several cross-sectional studies documented strong associations between lead exposure and cognitive and behavioral outcomes among children without clinical symptoms. But, by today's standards, even these children experienced extreme levels of exposure (e.g. Landrigan et al. 1975, Needleman et al. 1979).[7] More recent epidemiological studies have detected cognitive and behavioral deficits at ever lower levels of exposure (e.g. Canfield et al. 2003; Banks et al. 1997). The evidence on effects of low lead exposure is nonetheless still debated (Lanphear et al. 2000). Not only is it unclear at what level damage actually occurs, but the results have been questioned for the use of small and unrepresentative samples,[8] measurement error in blood-lead and outcomes, a focus on short-run effects, and the failure to properly control for confounding factors (e.g. Ernhart 1995; Kaufman 2001a, 2001b; Heben 2001; Needleman and Bellinger 2001). Bellinger (2004) suggests that developing and controlling for "more differentiated measures […] of social class", consistency of findings from multiple cross-sectional studies, and taking the evidence from animal experiments into account, could be valuable for drawing causal inference. Arguably, each of these solutions has limitations.[9]

Our study overcomes both the data and the identification issues by approaching this question from a different angle. First, by merging data on local air lead levels with comprehensive population micro data, we are able to measure lead exposure during the first three years of life for about 800,000 children and document their outcomes up to two decades later. The data include multiple markers of development and behavior: compulsory school performance, educational attainment, criminal convictions as well as direct measures of cognitive and non-cognitive skills in adulthood. We use a novel measure of lead exposure based on moss (bryophyte) samples collected all across

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of this kind can result in an upwardly biased estimate of the effect of lead exposure on children's subsequent outcomes. However, pollution also tends to be higher in densely populated areas and metropolitan areas attract highly educated parents with more resources, have better access to good child care, health care etc. which could understate the role of lead exposure if not properly taken into account.

[7] In Landrigan et al. (1975), mean B-Pb was 48µg/dl in the "high exposure" and 27 µg/dl in the "low exposure" group.

[8] Needleman et al. (1996) study 301 first graders 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.

[9] It is not at all clear that relying on consistent results from multiple cross-sectional studies solves the identification problem since similar confounders are likely to be prevalent in most settings, nor that adding more and more observables will be likely to satisfy the conditional independence assumption. Finally, while evidence from animal studies holds information on the toxicity of lead, it is, for example, unclear how such findings map back to cost-benefit analyses of particular policy interventions with respect to effects of human exposure on adult outcomes.

Sweden by the Environmental Protection Agency. Mosses are particularly useful as air pollution biomonitors since they lack roots and therefore solely absorb air-lead depositions.[10] Blood-lead, which is the most commonly used measure of lead exposure in the literature, provides measures of current exposure (half-life of 2 to 3 months), while the moss samples provide a measure of long-term exposure (average over three preceding years). However, in a smaller sample, we do verify the link between changes in local moss lead levels and changes in individual blood.

Second, we address the threats to identification by focusing on children born from the early 1970s until the mid-1980s. The reason is that during the 1970s, Sweden initiated a phase-out of leaded gasoline in order to protect the environment and public health.[11] In Sweden, the main reduction in gasoline-lead levels occurred between 1973 and 1981 when the maximum allowed lead level per liter of gasoline dropped by 80%. Since gasoline lead was the main source of lead exposure in the general population,[12] there was a sharp decrease in children's blood lead levels from the 1970s until the mid-1990s, when leaded gasoline was banned. Due to large differences in the initial lead levels, the phase-out of leaded gasoline induced a substantial variation across localities in the reduction of lead exposure. Our main estimation strategy exploits the differential within-neighborhood changes in early childhood lead exposure that occurred for the cohorts born between 1972 and 1984. By exploiting these sharp changes in exposure across birth cohorts within neighborhoods, we are not only able to account for observable background characteristics, but importantly also for neighborhood-specific unobservables. In addition, exploiting the timing of the changes in maximum allowed lead content following the major reforms in 1980/81, we trace-out how the reform effect evolves across cohorts.

Our study provides comprehensive evidence on the effects of low-levels of early lead exposure on adult outcomes. Our baseline linear specification shows that reducing early lead exposure improves compulsory school grade point averages, increases the likelihood of high school completion, and reduces the risk of being convicted before the age of 25. We also document stronger effects for boys, and for children from low

[10] The use of moss as biomonitors for heavy metal air pollution is well established today and used in many countries.
[11] Other parts of the developing world lagged far behind. For example, a continent-wide ban on leaded gasoline in Africa was not reached until 2006 (with the exception of Algeria).
[12] 80% of the air lead levels in the late 1980s, i.e. after the bulk of the reforms, were still due to traffic (MOENR 1994). Lead-based paint was banned in the 1920s in Sweden (in the end of the 1970s in the US).

income households. The results are robust to a wide range of specification checks, such as controlling for exposure to other pollutants and local economic conditions as well as testing for endogenous parental sorting.

The identification of thresholds, below which the effects of reductions in exposure have little or no effects on child outcomes, is of key economic policy relevance. Previous studies have failed to document such thresholds for non-clinical outcomes. However, earlier studies either use cross-sectional variation and/or examine effects at *much* higher levels of exposure. The Swedish setting is particularly compelling in search for a threshold, since the blood lead levels were much lower than in most other countries, including the US, already before the phase-out of leaded gasoline. Using a semi–parametric estimator, we show that below an estimated blood-lead level between 3 to 5 µg/dL (depending on the specification), the link between lead exposure and adult outcomes becomes much weaker. To put our estimates into perspective, reducing blood lead from 10 to 5 µg/dL, which corresponds to CDC's recently changed definition of elevated blood lead levels, taking the non-linear relationship into account implies a 10 percent increase in the probability of high-school completion and a 3 percentage point decrease in property crime convictions.

The analysis of the 1980/81 reform shows that: (*i*) the timing of the changes in outcomes across birth cohorts corresponds well with the sharp reduction in lead consumption following the reform, but (*ii*) children in neighborhoods *below the exposure threshold* are not affected by the reforms, whereas (*iii*) the outcomes of children in neighborhoods *above the exposure threshold* improve significantly. These results provide support for the validity of the main results.

Animal studies suggest that lead interferes with development, yet we know little about the pathways to later human capital outcomes, or if different types of skills and behavior respond more strongly to a given level of exposure. The specific channels are of obvious importance for efficient remediating investments. We provide direct evidence on two key channels highlighted in previous work: cognitive and non-cognitive skills. Interestingly, in this low-exposure setting, we find no significant or consistent pattern with respect to *cognitive skills*. However, *non-cognitive skills* follow the same non-linear dose-response pattern as human capital and crime outcomes. When examining the effects on the non-cognitive ("Big-Five" personality trait) skill sub-

components, we find that lead primarily influences Impulsiveness, Emotional stability (Neuroticism) and Conscientiousness.

Our study contributes to a large literature examining the effects of childhood lead exposure on human capital accumulation. Much of this literature focuses on the effects on grades, and cognitive or behavioral test scores in (primary) school using cross-sectional research designs. There is less evidence on effects on adult outcomes and very few previous studies report estimates that are interpretable as causal. However, a few recent studies address the identification problems. Reyes (2007) uses state-level panel-data and shows that the gasoline lead content reductions 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 during the 1990s. Reyes (2014) uses a similar research design for a sample of about 8,000 children in the National Longitudinal Survey of Youth (NLSY), finding a strong positive effect of childhood lead exposure on measures of delinquency for children followed up to age 17. Billings and Schnepel (2015) exploit the substantial measurement error in capillary blood lead tests to study a sample of 120 children that was treated in a lead remediation program in Charlotte, NC, and find that behavioral and educational deficits previously associated with high levels of early-life exposure can largely be reversed by intervention. Aizer et al. (2016) use an individual-level dataset of children with low lead exposure in Rhode Island and show that policies aimed at reducing lead hazards in homes increased reading test scores at age 8. Gazze (2016) exploits similar state mandates and finds that the share of kids attending special education decreases. A series of recent studies have exploited historical data from the late 19th and early 20th century to look at the importance of lead in water pipes for infant mortality, cognitive ability, and homicide (Clay, Troesken and Hains 2014; Troesken 2008; Ferrie, Rolf and Troesken 2012; Fiegenbaum and Muller 2016).

Our most important contribution is that we examine the effects of low levels of exposure on adult outcomes, while the earlier work examines short-run (elementary school) outcomes or effects at *much* higher levels of exposure. [13] The thresholds that

[13] Reyes (2007) report that average childhood blood lead levels in the adult US population will have decreased from 10 µg/dL in 2002 to less than 3 µg/dL in 2018. Billings and Schnepel (2015) examines of the intervention that occurs at 10 and 20 µg/dL 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. Gazze (2016) examines the impact on the share of children with special education following reductions in the share of children with B-

we document (~ 3 to 5 µg/dL) are informative about the effects of early childhood lead exposure on adult outcomes at levels that are still common in the US and in many other countries today. In addition, previous work tends to examine the outcomes separately, while we provide comprehensive results for a broad set of adult outcomes using the same approach and population-wide administrative data.

Of key importance for policy makers (and parents) choosing between remediating investments following early lead exposure, we also present evidence shedding some light on the impact on the inputs through which low levels of lead exposure affects adult outcomes. We find that low levels of early lead exposure do primarily seem to influence Externalizing behavior, Neuroticism, and Conscientiousness, rather than Cognitive skills. This is an important finding since grades and achievement test scores is widely used as measures of leads effect on *cognition* (e.g. Aizer et al. 2016), but achievement tests have been shown to be substantially explained by personality traits (Borghans, Golsteyn, Heckman, Humphries et al. 2015, 2011). Moreover, Almlund et al. (2011) show that Neuroticism and Conscientiousness are particularly strongly related to Externalizing behavior and Heckman et al. (2013) find that Externalizing behavior, in turn, is the key explanatory factor behind the crime reducing effects of the Perry Pre-school program. Since non-cognitive skills are more malleable to intervention than cognitive skills (Francesconi and Heckman 2016), our results suggest that policies that particularly target non-cognitive behavioral traits may be more successful in compensating for the negative effects associated with low levels of early exposure to lead.

Our study is also related to the large literature on the effects of air pollution on health, human capital, and mortality (e.g. Chay and Greenstone 2003; Currie and Neidell 2005; Lüchinger 2009; Jans et al., 2014). Most air pollution studies have not been able to assess the effects of environmental policies on adult outcomes. Isen, Rossin-Slater, and Walker (*forthcoming*) is a notable exception, who assess the effects of early childhood exposure to Total Suspended Particulate matter (TSP) by exploiting the 1967 Clean Air Act on earnings. We contribute, first of all, by examining the effects of exposure not only on earnings but also on academic outcomes, and cognitive- and non-cognitive skills. In addition, TSP is a mix of numerous components, including

Pb>10µg/dL and 20µg/dL. Aizer et al. (2016) study effects of low exposure (<10 µg/dL) but their analysis is limited to 3rd grade reading and math test scores.

heavy metals such as lead. It is still uncertain, but highly policy relevant, what properties of particulate matter that are responsible for the toxic effects on health (Harrison and Yin 2000). We contribute by isolating the effects on adult outcomes of regulating a specific pollutant at low exposure levels that are still common on a global scale. This specificity should help policy makers find cost-effective solutions irrespective of the exact source of lead exposure.

The rest of this paper is structured as follows. Section 2 presents a conceptual framework on how to think about the consequences of lead exposure for child development. Section 3 discusses the measurement of lead exposure and describes our data. Section 4 presents the empirical results and Section 5 concludes the paper.

2 Conceptual framework

Lead is highly toxic to humans, irrespective of how it enters the body, and affects brain-development and organ functioning. Appendix B provides a review of the evidence on the specific mechanisms that affect development documented in animal studies. Below, we highlight some findings that are particularly important for our empirical strategy and the interpretation of our results and then use the model of human capital skill formation technology developed by Cuhna and Heckman (2007) to formalize how even low early lead exposure may affect adult outcomes.

First, early lead exposure poses the greatest risk because of a higher absorption rate into the blood among children and also because conditional on absorption, lead is more likely to affect the developing nervous system than the mature brain (Lidsky and Schneider 2003).

Second, blood-lead levels follow an inverted v-shaped pattern between the ages of 6 to 60 months, reaching its peak at the age of 24 months due to the intense hand-mouth activity at these ages (Needleman and Bellinger 2001; Canfield et al. 2003).

Third, early childhood lead exposure/lead take-up does not differ by gender,[14] yet as we show empirically, lead exposure seems to affect boys more than girls. Several recent studies have similarly documented similar higher vulnerability of males to adverse prenatal, childhood, family, and neighborhood conditions.[15] However, the direct

[14] Studies that have investigated gender differences in blood lead levels have found that up until around the age of 10, the blood lead levels in boys and girls are highly similar (c.f. Strömberg et al. 1995).
[15] Nilsson (2008); Chetty et al. (2016); Author et al. (2015); Grönqvist, Niknami and Robling (2016).

evidence on gender differences in the effects of early childhood lead exposure is mixed. Burns et al. (1999) find no gender differences in the impact on cognition, while Cecil et al. (2008) document that early childhood lead exposure is correlated with a lower brain volume using MRI scans at ages 19–24 years, particularly among boys, and particularly in the prefrontal cortex. Damage to the prefrontal cortex has been linked to the inability to plan and see the future consequences of one's actions, impulsive behavior, lack of self-control and a lower ability to delay gratification. Such non-cognitive traits have previously been linked to, for instance, a higher risk of convictions (Åkerlund et al. 2016). However, as for the more common measures of development, it is unclear whether the gender differences detected by the MRI scans are due to biological differences in the sensitivity to lead or simply reflect correlated unobservables. Moreover, both studies focus on children with high levels of exposure (>10 µg/dL).

To better understand how early life lead exposure may influence individuals' life trajectories, it is useful to follow the framework proposed by Cunha and Heckman (2007, 2009) and characterize an individual's skill set as $\theta_t = (\theta_t^C, \theta_t^N)$, where θ_t^C is a vector of cognitive capabilities and θ_t^N is a vector of non-cognitive capabilities at age *t*. Adult outcomes is produced by a combination of the different skills at the end of childhood, with different weights placed on different outcomes. As suggested by Cunha and Heckman, the stock of skills in the next time period can be seen as a multi-stage developmental process where the stock of skills at age *t*+1 (θ_{t+1}) depends on the level of skills in the previous period *t*, investments (I_t) and parental environment (θ_t^P):

$$\theta_{t+1} = f_t(\theta_t, I_t, \theta_t^P).$$

The technology f_t is assumed to be increasing in each argument, twice differentiable, and concave in I_t. A key prediction of the model is that of "self-productivity", which arises when higher stocks of skills in one period create higher stocks of skills in the next period, i.e. $\partial f_t / \partial \theta_t > 0$. Secondly, the model assumes that the stocks of skills acquired by the end of the period $t-1$ make investments in period t more productive, i.e. $\partial^2 f_t / \partial \theta_t \partial I_t > 0$.

Following Cunha and Heckman (2007), we assume that the technology can be depicted as a CES and that there are two childhood periods and one adult period in an

individual's life-cycle: early childhood (*t*=1) and late childhood/adolescence (*t*=2). An individual's adult outcome Y can then be written as

$$Y = f_2(\theta_1^C, \theta_1^N, \theta_P, \left[\gamma \tilde{I}_1^{\phi} + (1-\gamma) I_2^{\phi}\right]^{\frac{1}{\phi}})$$

where $\in [0,1]$, $\phi \in (-\infty, 1]$. The share parameter (γ) is here a skill multiplier, and $\tilde{I}_1 = I_1 + \mu$, where μ is an exogenous shock occurring in period 1 (i.e. lead exposure). The elasticity of substitution, $1/(1-\phi)$, is a measure of the substitutability of $\tilde{I}_1$ and I_2 . ϕ represents the degree of complementarity/substitutability and determines how easy it is to compensate for low levels of investments (or a negative μ) in early childhood and in adolescence. When ϕ is small, it is difficult to compensate for low early investments ($\tilde{I}_1$) later in childhood (I_2). When $\phi = 1$, i.e. when $\tilde{I}_1$ and I_2 are perfect substitutes, the timing of investments is irrelevant. In the other extreme case, $\phi \to -\infty$, it is impossible to compensate for low early childhood investments later in childhood.

Early childhood is a sensitive period and the "neurological and behavioral effects of lead are believed to be irreversible" (WHO, 2016). Hence, $\phi < 0$ is likely to be the empirically relevant case. If so, even small early shocks may generate large negative effects on adult outcomes. Following Nilsson (forthcoming), to allow for differential effects of lead exposure across gender in (2) we allow ϕ to vary by gender, with $\phi_{boys} < \phi_{girls}$. Under this parameterization, the elasticity of substitution across periods is lower for boys than for girls, implying that it is more difficult to remedy an early shock for boys than for girls. Early lead exposure could therefore result in larger effects for males than for females.

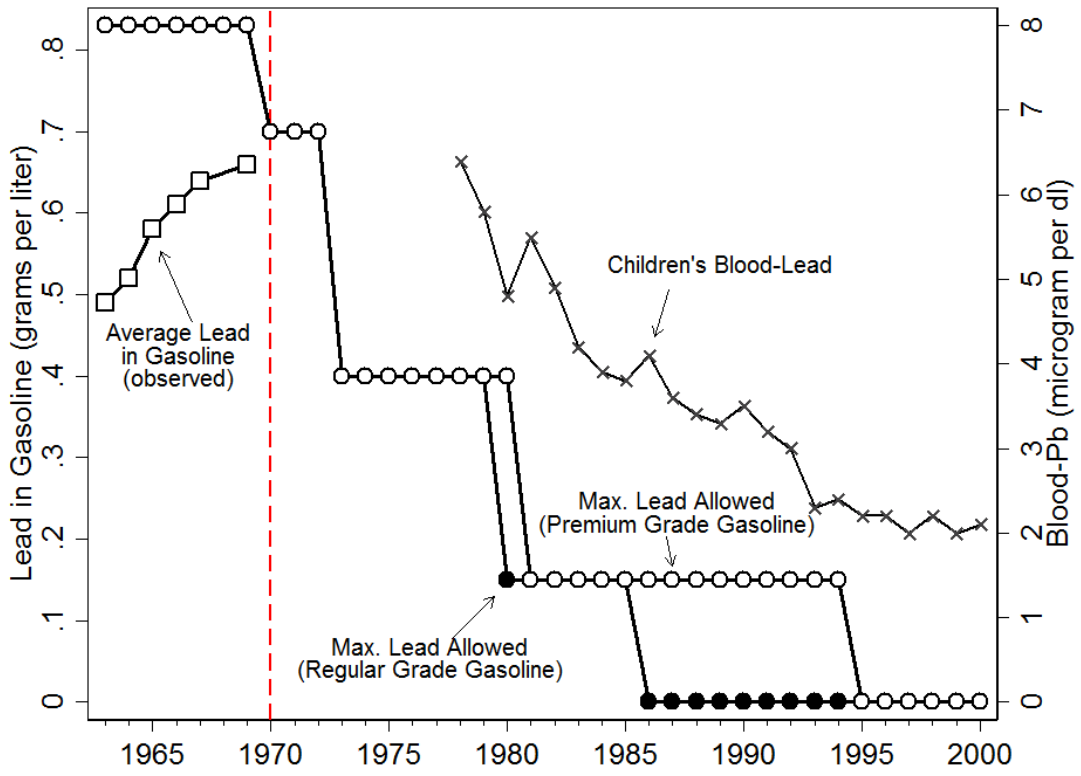
3 Background

3.1 Measuring lead exposure

Lead started to be added to gasoline in Sweden in 1946 (Danielson, 1970). In response to the increasing trend in lead emissions from motor vehicles (see Figure 1), Sweden initiated a gradual phase-out of leaded gasoline in order to protect the environment and public health in 1970. While the 1970 reform prevented further increases in gasoline lead content, the main reductions in gasoline-lead levels occurred between 1973 and

1981 when the maximum amount dropped by 79% (see Figure 1). Gasoline lead was the main source of lead exposure in the general population and, as shown in Figure 1, there was a sharp decrease in children's blood-lead levels (B-Pb) from the 1970s until the mid-1990s when leaded gasoline was banned.[16]

Figure 1. Changes in lead levels in Sweden by type of gasoline 1970–2000 and mean blood lead levels among elementary school children in Sweden 1978–2000



Notes: From the left to the right, the figure shows the observed average lead levels in gasoline for the period 1963–1967, and 1969 (hollow squares) (c.f. Danielson 1970), the timing of the first reform (vertical line), the maximum allowed lead content in gasoline for premium grade (hollow circles) and regular grade gasoline (solid circles), before 1980 the regulation was the same for all grades (data from The SPI). The right-hand side y-axis shows the blood-lead level of elementary school children (µg/dL) (c.f. Stromberg et al. (1995) and Stromberg et al. (2003).

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 fifth year using a nationwide grid of moss (bryophytes) samples. The method of using mosses as biomonitors of heavy metal air pollution was developed in Sweden in the 1960s in pioneering work by Rühling and Tyler (1968, 1969) and is by now well

[16] In Sweden, lead water pipes were never an issue and lead based paint was banned in the early 1920s. Even after the lead reforms in the 1980s, 80% of the air lead levels were still due to traffic (MOENR 1994).

established.[17] Moss is particularly suitable for biomonitoring of air pollution levels for several reasons. The lack of roots implies that moss solely depends on surface absorption of pollution through precipitation and dry deposition of airborne particles. Mosses can be found in abundance in nearly all environments. The absorption and retention of metals is high, and it is possible to distinguish temporal patterns in pollution levels.[18]

Biomonitors have some 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 changes in local pollution levels, the samples are collected all over Sweden using a systematic procedure.[19] The growth segments over the last three years are pooled and analyzed and provide a measure of the local air lead levels during the three preceding years.

We use the samples collected in 1975, 1980 and 1985, which reflect 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 sharply. As shown in Figure 1, the maximum allowed lead level in gasoline was reduced from 0.7 to 0.4 g/L (2.65 g/gal to 1.51g/gal) in 1973. 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 0.4 to 0.15 g/L (0.56 g/gal). In 1986 (1995), lead was banned for regular (premium) gasoline. Second, the sample is restricted to cohorts who have reached adulthood and for whom the exposure in early childhood is observed.

The correct location of the specific sampling sites and the collection procedure are strictly defined. However, the sampling locations are not always identical across years. Hence, to construct a measure lead exposure for each period and neighborhood, we calculate the inverse distance weighted average air lead level for each parish using the

[17] 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 survey designed to study regional differences and time trends in heavy metal deposition throughout Europe.
[18] The annual growth segments are easily distinguishable and the transportation of metal across segments is minimal.
[19] The sampling sites are chosen with care; 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 sampling site, 5 to 10 subsamples are collected in an area of 100 m^2.

population weighted parish centroids and the 10 closest sampling sites. The average parish in our data have about 3,000 inhabitants, i.e. it is slightly larger than US Census Block Groups.

As we show in Online Appendix A, there is a great deal of variation in the local lead levels between neighborhoods. Figure A1 and Figure A2 presents maps for the lead concentrations in the neighborhoods in 1975 and 1985, using our definition of exposure. From these, it is clear that the moss lead concentrations fell sharply between 1975 and 1985. Moreover, Figure A3. shows that the entire distribution of lead levels shifted dramatically between 1975 and 1985 and became more compressed. Figure A4. shows the distribution of within-neighborhood changes in lead exposure between 1975 and 1985. We can see that most neighborhoods experienced decreases in moss lead levels by between 15 and 45 mg/kg, with an average reduction of 30 mg/kg. A variance decomposition reveals that most of the variation in lead exposure is due to within neighborhood rather than between neighborhood differences.[20] These sharp within-neighborhood differences in the reduction of early childhood lead exposure across cohorts constitute 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 question concerns the arbitrary choice of using the 10 nearest sampling sites to define the neighborhood of birth lead exposure. To test the sensitivity of the analysis to this assumption, we have also used the five nearest sample points instead. The differences between these definitions are small and they are highly correlated (ρ = .99). Second, to cross-validate our approach, we reestimate the level of lead at each sampling point, as opposed to neighborhood, after excluding the sampling point of interest. We do this for all sample sites, and then correlate the actual and the predicted air lead levels. The correlation is high (ρ = .82), indicating that the interpolation method provides reasonably accurate predictions of local air lead levels.

Finally, as in any study using data on local pollution levels rather than individual exposure, an important question is how well the lead levels in moss predicts the actual B-Pb levels in children. Unfortunately, there exist no data that monitors the trends in B-Pb levels among young children or the population in general in Sweden during this time

[20] The overall standard deviation for our measure of lead exposure is 17.93 while the between and within standard deviations are 10.78 and 14.35, respectively.

period. However, since 1978 venous blood samples have been collected from about 120 primary school children (age 8–10) per year in two municipalities in southern Sweden.[21] One of these municipalities (Landskrona) has also collected around 50 moss samples using the same procedure as in the national survey at three time points (1984, 1995 and 2006). 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 from Landskrona provide a unique opportunity to assess the strength of the relationship between measures of local air lead exposure and measures of children's lead exposure. Figure 1 shows the mean B-Pb levels among primary school children in 1976–2000. Nilsson et al. (2009) link the average lead level of the nearest moss samples to the children using their home addresses. Controlling for important individual characteristics, time and neighborhood fixed effects, they estimate a Blood-Moss-lead elasticity for the pre-ban period (i.e. before 1995) of 0.44. This implies that a 10% reduction in Moss-Pb corresponds to a 4.4% decrease in primary school children's B-Pb, and suggests that the drop in air-lead exposure between 1982 and 1994 can account for as much as 50% of the change in children's B-Pb levels. Appendix B provides more details on the data and estimations.

However, it is important to remember, that the relationship between environmental lead exposure and young children's blood lead levels is significantly higher than among older children due higher exposure and higher absorption. For example, Reyes (2007) finds that the elasticity between lead in gasoline and B-Pb in children aged 0–6 is around 30% higher than among children aged 6–12. In this analysis, we therefore adjust the estimated elasticity accordingly when estimating the relationship between adult outcomes and early childhood B-Pb levels.

3.2 Micro data

Our data originate from three distinct databases at the Institute for Labor Market Policy Evaluation (IFAU), the Department of Economics at Uppsala University and the Institute for International Economic Studies (IIES) at Stockholm University. All databases draw from the same population based registers maintained by Statistics Sweden (SCB), which contain the information necessary to identify our main sample as well as a range of standard background characteristics such as demographics and parental socio-

[21] The methods and results for the trends in childhood lead exposure are described in detail in Strömberg et al. (2003).

economic status. However, the databases differ in terms of the outcome measures included.

In the main analysis, we include all individuals born in Sweden in the three years prior to the year in which the moss samples were collected; that is all those born in 1972–1974, 1977–1979 and 1982–1984. The reason for the 1972 and 1984 constraint is that many of the individuals born after 1985 are less likely to have finished schooling at the time we measure their outcomes 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 at ages 1–3 years, which corresponds to a particularly sensitive period in human development and a period with high lead uptake rates. We limit our sample to children who have completed compulsory schooling (9 years of education) and who were born in Sweden, so that their neighborhood of birth (and hence, childhood lead exposure) is known. Hence, in our main analysis, we focus on outcomes for which we are able to measure at the same age for all cohorts.[22] Human capital is measured using compulsory school grade point average (GPA) (age 16) and the probability of graduating from high school (age 19). We percentile rank the GPA to account for changes in the grading system over cohorts. We observe both these outcomes for all individuals until 2013.

We measure criminal behavior using administrative data on criminal convictions between 1985 and 2008 provided by the National Institute for Crime Prevention (BRÅ). As is typical with administrative crime data, we cannot directly observe criminality, but rather use convictions as a proxy for criminal activity. The data includes full records on the type of crime as well as the exact date of the offense for all individuals up to the age of 24.[23] A conviction may include several crimes and all crimes are recorded. Speeding tickets, and other minor crimes not severe enough to warrant a trial, are not covered. We study crime as the probability of being convicted at least once (for any crime) and the more specific and most common crimes categories: violent crime (representing the full spectrum of assaults from minor assault to murder) and property crime (representing the full spectrum of thefts from shop-lifting to robbery).

[22] Sweden is one of the countries in the OECD with the highest age when obtaining the first university-level degree (average age 29) and more than 40 percent of each birth cohort also enrolls in college (OECD 2014).
[23] The age of criminal majority in Sweden is 15, meaning that we cannot observe criminal convictions before the age of 15.

We shed some light on the mediating factors affecting the adult outcomes by also studying the effects on cognitive and non-cognitive skills as measured for men at military enlistment. All men were obliged by law to go through the military draft. However, due to reforms in the military enrollment procedures affecting those born during the 1980s, the enlistment outcomes are only used for those born before 1980. Among these cohorts, about 90 % of all men went through the draft at the age of 18 or 19.[24]

The cognitive test scores are Stanine (Standard Nine) test scores which are 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. The test score is standardized within each cohort of draftees to account for any minor changes in the tests over time.[25]

The non-cognitive score is based on a 20– to 25–minute interview with a psychologist resulting in four different scales, all ranging from 1 to 5, here displayed with their respective sub-categories and the Big-Five traits of Personality[26] (in italics) to which they are related: (i) Social maturity (extraversion (*E*), having friends (*E*), taking responsibility (*C*), independence (*O*)), (ii) Psychological energy (perseverance (*C*), ability to fulfill plans (*C*), to remain focused (*C*)), (iii) Intensity (the capacity to activate oneself without external pressure (*C*), the intensity and frequency of free-time activities (O)), and (iv) Emotional stability (ability to control and channel nervousness (*N*), tolerance of stress (*N*), and disposition to anxiety (*N*)). The non-cognitive sub-score tests are combined into a Stanine scale which we use as our main measure of non-cognitive ability.[27] Table A1. in the online appendix provides summary statistics for all outcomes.

4 Results

Empirically, we exploit the variation in lead exposure induced by the phase-out of leaded gasoline in the following three ways. Using the full data, we first provide (1)

[24] In principle, only the physically and mentally handicapped were exempt from the enlistment tests.
[25] The test has been subject to evaluation by psychologists and appears to be a good measure of general IQ (Carlstedt 2000).
[26] (*O*)peness to Experience, (*C*)onscientiousness, (*E*)xtraversion, (*A*)greeableness, (*N*)euroticism.
[27] Appendix D in Nilsson (2016) provides an account of how the military non-cognitive sub-scores (c.f. Bihagen et al., 2012) relate to the Big-Five traits of personality. Lindqvist and Vestman (2011) relate the cognitive and non-cognitive skills to labor market outcomes.

linear fixed effect estimates, and then provide results from (2) a semi-parametric fixed effects model that allows for a fully flexible exposure-response function. Finally, (3) we home in on the cohorts born between 1976–1984 and provide a detailed analysis of the effects of the large 1980/81 reforms through which the maximum lead content dropped from 0.4 g/l to 0.15 g/l.

4.1 Baseline results and robustness

To identify the effect of lead exposure on life trajectories, we use variations of the following linear regression model regressed on data aggregated to the cohort-neighborhood level:

$$Y_{nc} = \alpha + \beta Lead_{nc} + \theta X_{nc} + \delta Z_{mc} + \lambda_n + \lambda_c + \varepsilon_{nc} \quad (3)$$

where the Y_{nc} is compulsory school GPA (percentile rank), the probability of completing high school, or the probability of having been convicted for a crime up to the age of 24. $Lead_{nc}$ is the local air lead levels measured in terms of milligram per kilogram (mg/kg) moss. This measure varies for individuals born in different years (*c*) and in different neighborhoods (*n*). X_{nc} and Z_{mc} represent vectors of parental and individual characteristics aggregated to the neighborhood level (*n*) (parental income and education, maternal age at birth, and gender, and municipality (*m*) level covariates (employment rate and population size). We include neighborhood fixed effects (λ_n) that account for persistent differences across neighborhoods over time, and birth cohort fixed effects (λ_c) which take cohort-specific effects into account. ε_{nc} is the error term. Throughout, we cluster the standard errors at the commuting zone (CZ) level to account for arbitrary serial correlation and heteroscedasticity. Clustering at the CZ level also accounts for spatial correlation in the errors between neighborhoods within the 74 CZs.[28]

The parameter estimate of interest, $\widehat{\beta}^{OLS}$, should be interpreted as the effect of an additional unit of lead exposure (1 mg/kg) in early childhood on adult outcomes. Before proceeding to the main results, we discuss three potentially important threats to consistent estimates of β in equation (3).

[28] There are 2,586 parishes in the data. Statistics Sweden divided these into 74 commuting zones. For a map of the 74 commuting zones, see: *http://www.scb.se/Statistik/AM/AM0207/_dokument/LA2009_KARTA.pdf*.

The first concern is that parents might sort into residential location based on factors associated with lead pollution levels. This could overstate the effects of lead exposure if parental preferences for clean air are also associated with higher parental investments in children. 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 the adult outcomes of the children. Such local amenities could, in turn, for instance, via gentrification, result in an underestimated role of childhood lead exposure if not properly taken into account.

When Tiebout sorting occurs, the most important predictor of both residential location and child outcomes is probably socioeconomic background. To better understand the importance, direction, and severity of this bias, we start by regressing parental educational attainments and income on their children's lead exposure.[29] This gives a hint of whether omitted variables associated with parents' characteristics, or migration that changes the composition of families within neighborhoods, are correlated with changes in lead exposure.

The correlation between lead exposure and parental characteristics are displayed in Table A2. Panel A shows the cross-sectional correlation and Panel B shows the same results after adding neighborhood fixed effects to the specification. Overall, the coefficients in Panel A suggest that higher lead exposure of the child is associated with lower parental education, lower pre-birth maternal income, and lower maternal age at birth. This suggests that cross-section regressions likely overstate the effects of lead exposure on children's outcomes. After taking into account fixed differences across neighborhoods, the coefficients generally drop substantially and often change sign.

[29] These parental characteristics can be considered to be predetermined in the present context since the lion's share of the parents (>95 percent) were born before 1960, i.e. before environmental lead exposure became a serious environmental problem in Sweden. 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 was restricted to two distinct periods: a first increase towards the end of the nineteenth century, and a second increase during the 1960s (80-90 mg/kg in 1968). Before this, the average lead level in Skåne (the southernmost region in Sweden with the highest lead level in the data used in this study) was around 40µg/kg moss. They conclude that the first rise is probably due to industrial pollution, possibly due to the increased use of coal, and that the second rise is more than likely caused by the rapidly increased use of lead gasoline. Exactly the same pattern is found in a study by Rehnberg et al. (2000). They 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 during the parents' childhood are not expected to be able to influence the children's adult outcomes to any large extent.

However, note that neither the coefficients in Panel A nor in Panel B are statistically distinguishable from zero, and that parents' socioeconomic characteristics show no consistent association with their children's lead exposure.

A second concern is that although there is a consensus that the overall drop in lead exposure is driven by gasoline lead policies (see Thomas et al. 1999), local lead exposure could also proxy for unobserved neighborhood characteristics that are not captured by the neighborhood fixed effects. Perhaps the two most obvious candidates include changes in the local business cycle and population density, which can both be hypothesized to be linked via emissions to local lead levels and to outcomes. To the extent that they are correlated with changes in parental income and education, the analysis in Table A2. suggests that they are not a concern.

However, from the bi-decennial Censuses, we also gathered municipality level data on population density and employment covering not only the parents. Figure A5. and Figure A6. show that changes in local lead exposure between 1975 and 1980 and between 1980 and 1985 appear to be unrelated to changes in population density or local employment. Put differently, in terms of labor market conditions and migration patterns, places with high lead declines seem to have been relatively similar to places with low lead declines. Accordingly, as we see below, controlling for these variables in the regressions does not change the magnitude of the estimates in any meaningful way.

Similarly, local lead levels may be correlated with other pollutants that also affect child outcomes. The focus on the changes in air lead levels induced by government regulations that specifically targeted gasoline lead content should mitigate much of this concern. Still, since the moss data also hold information on other common heavy metals, it is easy to make an initial assessment of the potential severity of this problem. Of all the observable heavy metal pollutants in the data, only Cadmium (Cd) displays nearly as large and widespread changes during the observation period as does lead. Cd has previously been found to be associated with adverse health outcomes (kidney damage, bone disease).[30] The data also include information on levels of Copper (Cu)

[30] Early Cd exposure has been shown to be able to produce neurotoxic effects in laboratory experiments (Anderson et al. 1997; Peterson et al. 2004), and in utero exposure to cadmium may affect infant health in humans (Currie and Schmeider 2009). Air Cd and air Pb concentrations display a fairly high correlation at the parish level. Hence, changes in air Cd levels could at least partly explain the estimated relationship between lead and subsequent outcomes. However, in this context, it is not likely that the estimates for Pb are driven by the changes in local air Cd exposure rather than local air lead exposure. This is because 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) Cd accumulates in

and Zinc (Zn). Cu and Zn have been suggested to be used as marker elements for motor vehicle emissions in previous studies and are therefore useful to include in the analysis to control for other traffic related pollutants.[31] As we will see in the next section, the estimates are stable when controlling for these other heavy metal pollutants.

4.1.1 Baseline results

Table 1 presents the main results. Each table entry corresponds to a separate regression. The table provides results for GPA, High school completion, and Criminal convictions by type of crime. In the first row, we only control for cohort and neighborhood fixed effects. Then, we successively add more controls. Our preferred specification is found in the bottom row and provides the neighborhood fixed effect estimates of increasing moss lead by 10 mg/kg, after including the full set of controls (individual, parental, municipal and other traffic-related pollutants).

The pattern of the coefficients is clear. Higher levels of exposure in early childhood lead to lower GPA and high-school completion, and a higher risk of being convicted. Overall, adding the controls hardly changes the estimated coefficients at all. The exception is the effect on violent crime, which is always positive but becomes insignificant in the full specification. The property crime coefficient is positive and stable across the different specifications but is always statistically insignificant in this linear specification. We will return in Section 5 to the question of how to assess the magnitude of these estimates.

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, it is *a priori* not expected that the intertemporal changes in *local* air Cd levels in early childhood are necessarily associated with adverse future outcomes.

[31] Brake linings wear are the major source of road traffic emitted Cu, and Zn concentrations are high in tiers (e.g. Johansson et al. 2009; Hjortenkrans et al. 2007).

Table 1. The effect of childhood lead exposure on life trajectories

Outcomes:	Grade 9 GPA (pct. rank)	P(High school complete)	P(Ever convicted)	P(Property crime)	P(Violent crime)
	(1)	(2)	(3)	(4)	(5)
Baseline	-.1890**	-.0034**	.0014**	.0007	.0004**
	(.0961)	(.0014)	(.0007)	(.0008)	(.0002)
+ individual & family controls	-.2018***	-.0038**	.0017**	.0008	.0005**
	(.0716)	(.0015)	(.0006)	(.0007)	(.0002)
+ municipality characteristics	-.2009**	-.0039**	.0018**	.0008	.0005**
	(.0716)	(.0015)	(.0007)	(.0007)	(.0002)
+ other traffic pollutants	**-.2097****	**-.0031****	**.0018****	**.0009**	**.0002**
	(.0925)	**(.0014)**	**(.0008)**	**(.0009)**	**(.0003)**
Mean of Outcome	[50.72]	[.79]	[.164]	[.070]	[.025]
Neighborhood FEs	Yes	Yes	Yes	Yes	Yes
Year of birth FEs	Yes	Yes	Yes	Yes	Yes

Notes: The table presents the coefficient on early lead exposure from separate OLS regressions. Coefficients and standard errors are scaled by a factor of 10. Crime (in columns 3-5) is defined as having been convicted at least once up to the age of 24. The sample consists of children born in 1972-1974, 1977-1979 and 1982-1984. Childhood lead exposure is measured as an average over ages 1-3. The baseline model only controls for cohort and neighborhood fixed effects. We then add individual (gender, birth month dummies, birth weight) and parental controls (dummies for the unique combination of the parent's highest completed level of education (55 combinations), maternal age dummies in years at birth, dummies for quartile of total parental earnings two years before birth (in 1985 for crime outcomes), birth order dummies. Municipality controls include population size and employment rate from the 1975, 80 and 85 censuses. Other traffic related pollutants include Cadmium, Zinc and Copper. Sample means expressed in percent are shown in brackets. Cluster robust standard errors (at the commuting zone level, 74 cells) are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % .The preferred (full) specification is highlighted in bold.

4.1.2 Estimates by Gender

As discussed in Section 2, several recent studies have documented a greater vulnerability of males to adverse early conditions, but the evidence on gender differences in the impact of lead is mixed. Hence, it is of interest to examine differences across genders in our low lead setting. Panel A of Table 2 repeats the results from the full sample from the main specification in Table 1, while panels B and C provide the same estimates separately for boys and girls. It is striking that the baseline results not only for criminal convictions but also for GPA and high school completion seem to be driven by the impact on boys. Since early childhood lead take-up does not differ across genders, these results suggest that the effects of early lead exposure on adult outcomes are stronger for males than for females.

Table 2. Heterogeneity in the effect of lead exposure by gender and parental income

Outcomes:	Grade 9 GPA (pct. rank) (1)	P(High school completion) (2)	P(Ever convicted) (3)	P(Property crime) (4)	P(Violent crime) (5)
	Panel A: Full sample				
Early lead exposure	-.2097** (.0925)	-.0031** (.0014)	.0018** (.0008)	.0009 (.0009)	.0002 (.0003)
Sample mean	50.72	.79	.164	.070	.025
	Panel B: Girls				
Early lead exposure	-.1452 (.1110)	-.0011 (.0015)	.0004 (.0007)	.0001 (.0006)	.0000 (.0001)
Sample mean	56.69	.808	.079	.045	.006
	Panel C: Boys				
Early lead exposure	-.2773*** (.0989)	-.0051*** (.0019)	.0029** (.0013)	.0017 (.0014)	.0006 (.0005)
Sample mean	44.85	.769	.244	.095	.042
	Panel D: Low Income Parents				
Early lead exposure	-.2640** (.1170)	-.0037* (.0019)	.0017 (.0011)	.0014 (.0011)	.0007** (.0004)
Sample mean	45.39	.739	.191	.087	.033
	Panel E: High Income Parents				
Early lead exposure	-.2120** (.1020)	-.0027** (.0013)	.0015* (.0007)	.0004 (.0007)	-.0002 (.0002)
Sample mean	55.38	.834	.136	.054	.016

Notes: The table presents the coefficient on early lead exposure from separate OLS regressions. Coefficients and standard errors are scaled by a factor of 10. Crime (in columns 3-5) is defined as having been convicted at least once up to the age of 24. The sample consists of children born in 1972–1974, 1977–1979 and 1982–1984. Childhood lead exposure is measured as an average over ages 1–3. Parental income is measured as the sum of each parent's earnings. Low(High) income parents are classified as below(above) median family earnings either two years prior to when lead exposure is measured (for human capital outcomes) or in 1985 (for crime outcomes). Besides neighborhood and birth cohort fixed effects, the model controls for the full set of controls as specified in the note of Table 1. Cluster robust standard errors (at the commuting zone level (74 cells)) are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % .

4.2 The role of nonlinearities

The default dose-response model in toxicology is the threshold model, which posits that the effect of toxins follows a hockey-stick shape across the distribution. Below certain thresholds, further reductions in exposure no longer affect the outcome of interest. Some indicative health thresholds for lead have been established for children:

encephalopathy, seizures and coma (>70–80 µg/dL), renal (kidney) failure (50µg/dL), hemoglobin disruption (25 µg/dL) (Agency for Toxic Substances and Disease Registry 2007). However, no thresholds have been established for more subtle effects on neuro-cognitive development or behavior. To what extent that this is due to the limitations of the cross-sectional research designs, or that previous studies generally test for non-linarites at high levels of exposure, or both is unclear.[32] The literature that attempts to address the identification issues and directly test for non-linearities has not detected any thresholds, but again, it has examined effects at much higher exposure.[33]

To examine the role of non-linearities in our low-exposure setting, we estimate semi-parametric fixed effect models (Baltagi and Li 2002). This estimator takes into account neighborhood fixed effects and the other controls used in the linear specification, but allow the exposure-response function to be estimated fully flexibly:

$$Y_{nc} = \alpha + f(Lead_{nc}) + \theta X_{nc} + \delta Z_{mc} + \lambda_n + \lambda_c + \varepsilon_{nc}. \quad (4)$$

For human capital outcomes, the estimated exposure-response function, $\hat{f}(\cdot)$, for males is presented in Figure 2 together with bootstrapped 95% confidence intervals (1,000 replications).[34] The top panel shows the results for GPA which reveal some indication of non-linearites at the very bottom of the distribution (~ 20 mg/kg), where the relationship flattens out. However, the confidence bands are wide, and it is impossible to reject that the relationship is linear for GPA. The pattern is much clearer for high-school graduation. Here there is a clear threshold at around 30 mg/kg below which further reductions in exposure have limited or no effects on graduation rates.

[32] There is no strong theory that could give any guidance on the exact location of the thresholds for the outcomes we consider (c.f. Needleman 2004). In fact, some cross-sectional studies find that the effects of lead are *increasing* at lower levels of exposure, i.e. changes in exposure at low levels of exposure have stronger effects on test scores than changes at high levels of exposure (see Canfield et al. 2003; Skerfving et al. 2015). The unexpected direction of the "nonlinear dose-response curve is a mystery" (Skerfving et al. 2015, p.118). A potential explanation is that at lower levels of exposure, the composition of children that are exposed to lead is likely to differ from the composition of children at higher levels of exposure. To give a concrete example, before the ban of leaded gasoline, more or less all children were exposed to some extent, with gasoline lead being the key source of exposure. After the phase-out, only a share of the children are exposed, namely children living in low standard housing with deteriorating water pipes, lead paint walls, or living close to heavy industries like smelters. Therefore, it is possible that the bias from unobserved factors is stronger in a low lead setting than in high lead setting, generating a steeper dose-response curve in settings where the sources of exposure are more family specific and hence likely to more closely reflect (unobserved) family characteristics.

[33] Reyes (2011) directly examines non-linarites in the effects of early lead on violent crime, but does not find any threshold effects at average blood lead levels between 10 µg/dl and 20 µg/dl.

[34] To ease the computational burden, we collapse the individual level data at the neighborhood-by-measurement year level and weight the regressions by the number of individuals in each cell. The non-parametric part is estimated using a narrow bandwidth (.3) robust local linear estimator (LOWESS). To further reduce the influence of outliers, we follow standard practice and we exclude the bottom/top 1% of the observations in the exposure distribution.

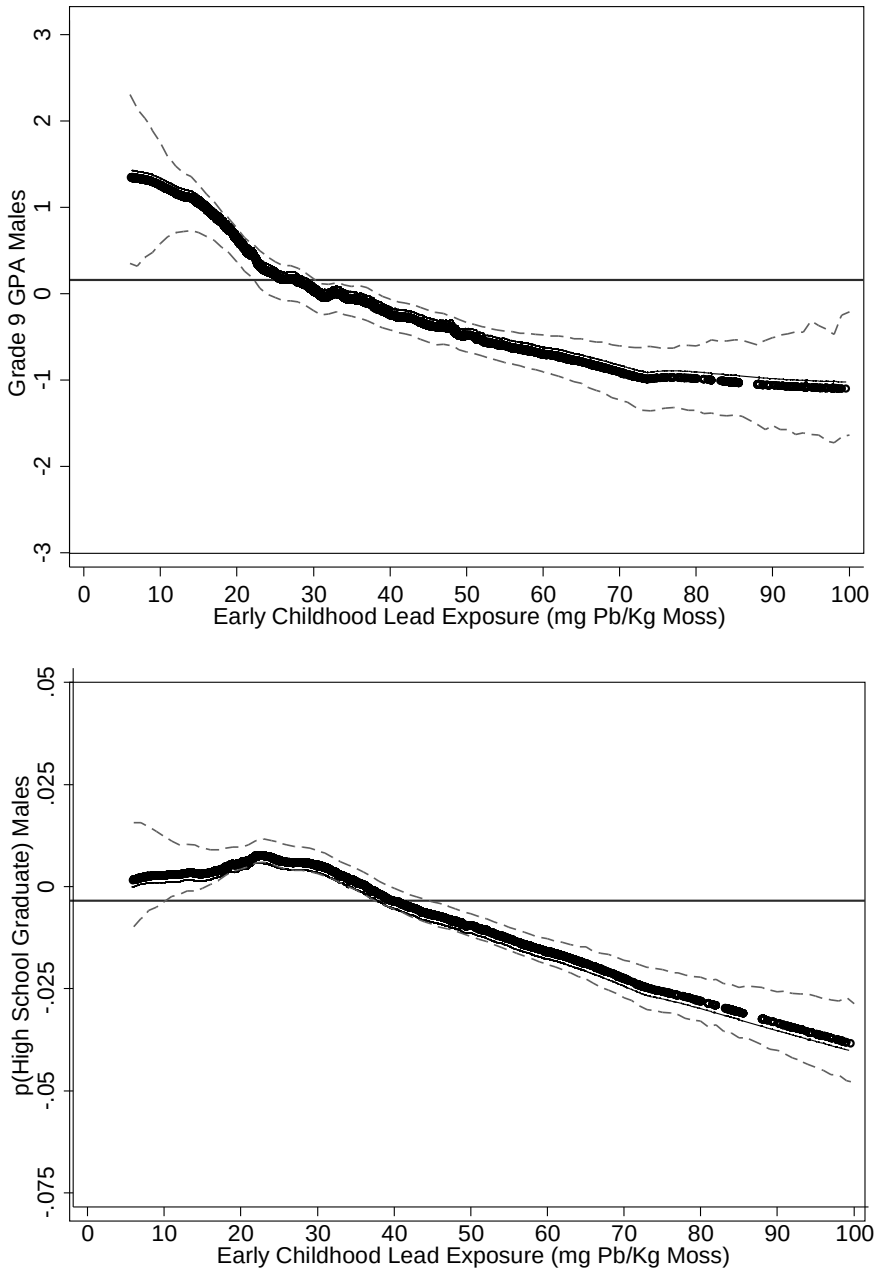
Above 30 mg/kg, there is a clear and statistically precise negative relationship between exposure and the probability of graduating from high school. For completeness, Figure A7. provides the same estimates for females. For females GPA, there is a tendency to a non-linear effect at the very top of the exposure distribution, but overall, as expected from the linear regression results, the pattern is less clear for females than for males.

Figure 3 provides results for Criminal convictions. For Any crime, there is a tendency to the same pattern as for high school completion, but the relationship is imprecisely estimated. For Property crime, the relationship is clearer, above approximately 65mg/kg there is a clear and statistically significant positive relationship between lead exposure and property crime. Below 65mg/kg, the regression line is flat: changes in lead exposure have no impact on Property crime below the threshold. For Violent crime, there is a tendency to a similar pattern as for Property crime, but it is not statistically significant.

The semi-parametric regressions suggest that 65 mg/kg could be a candidate for a threshold for (property) crime. For human capital outcomes, the relevant threshold seems to be located at a lower level (20–30mg/kg). To verify the locations of these thresholds, we follow standard practice and estimate a series of piece-wise linear equations that allows for a change of slope at different *assumed* thresholds (Hansen 1999). Based on these regressions, Hansen's Likelihood Ratio (LR) test statistic provides "no-rejection regions" containing the best estimate of where the *true* threshold is located. Appendix A Figure A8. and Figure A9 show the LR-statistic threshold locations. In general, they verify the location suggested by the more flexible semi-parametric regressions.[35]

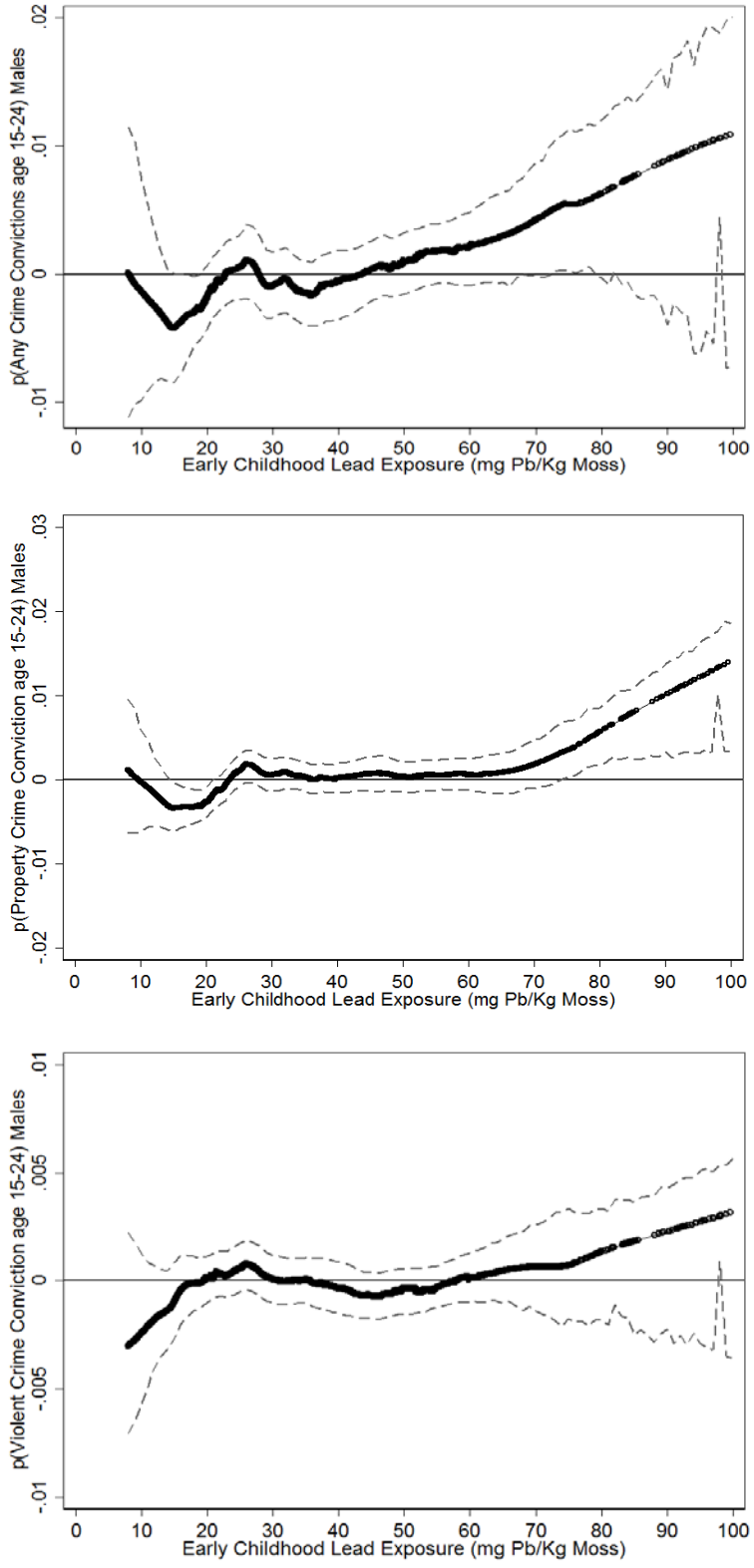
[35] Following Hansen (1999), the criterion to locate the true threshold is the assumed threshold at which the sum of squared residuals in the piecewise linear regression is minimized. Hansen's likelihood ratio (LR) statistic is $nT\frac{SSR_s - SSR_{min}}{SSR_{min}}$ where $SSR_s$is the sum of squared residuals at the assumed threshold *S*, SSR_{min} is the minimum value of this statistic across all thresholds, *n* is the number of observations and *T* is the number of time periods. The advantage of the LR statistic over the *SSR* is that its asymptotic distribution (critical-value) is known.

Figure 2. Semi-parametric estimates for human capital



Notes: The graphs present estimates along with a 95 % confidence interval (bootstrapped with 1000 repetitions) from semiparametric fixed-effects models (see Baltagi and Li 2002). The dependent variable in the top graph is grade 9 GPA and in the bottom graph P(High school completion). The sample consists of males born in 1972-1974, 1977–1979 and 1982–1984. Childhood lead exposure is measured as an average over ages 1–3. See note Table 1 for the full specification set of controls.

Figure 3. Semi parametric estimates for crime



Notes: Semi-parametric fixed effects estimates (see Baltagi and Li 2002) with 95% bootstrapped confidence intervals (1,000 repetitions). The dependent variable in the top graph is P(Ever convicted), in the middle graph P(Property crime) and in the bottom graph P(Violent crime). Additional details on the data are given in the notes to Figure 2.

In summary, the analysis in this section reveals clear signs of nonlinearities in the effect of early childhood lead exposure on adult outcomes. Since the average blood-lead level in the cohorts considered were already initially lower than 10 µg/dL, these results also suggest that, if anything, the threshold of the adverse effects of lead exposure on adult outcomes lies at a level that is significantly lower than a B-Pb level of 10 µg/dL.[36] In Section 5, we use the elasticity provided by Nilsson et al. (2009) to estimate the childhood B-Pb level corresponding to the moss lead level documented in this section.

4.3 Isolating the effects of the 1980/81 reform

It is well documented (c.f. Thomas et al. 1999) that the general dramatic drop in lead exposure that occurred during the observation period is due to the policies mandating reductions of the maximum lead contents of gasoline. However, one may still be concerned that at least a part of the local variation in lead that is used in the panel analyses is driven by correlated unobservables that are not captured by either the neighborhood-of-birth fixed effects or the extensive set of control variables.

To further try to address this concern, below we provide results from an analysis that exploits the timing of the 1980/81 reform.[37] As shown in Figure 1 above, the maximum lead content of regular (premium) gasoline decreased from 0.4 to 0.15 grams per liter gasoline in January 1980(81).[38] Combining this information with yearly municipality data on gasoline sales by grade (available for the period 1976 to 1984), Figure A10. shows the average of the maximum gasoline lead sales (in metric tons) per municipality and year around the time of the reform. The sharp reductions in maximum gasoline lead in 1980/81 (a 63 percent drop) seen in Figure A10. provides an opportunity to more clearly isolate the effect of the reform and how the reform effect evolves across birth cohorts. We estimate the following model for children born between 1976 and 1984:

[36] See Appendix B for evidence on the pre-phase out B-Pb levels.

[37] We focus on this reform since first since there are several years since the previous reform was implemented (1973), and there are several years until the next reform was implemented (1986). This gives us a reasonably long pre-reform period, which we can use to examine pre-reform trends in the outcomes. It also enables us to examine the same outcomes that we focus on in the main analysis. Second, the 1980/81 reforms allows us to use a well-defined pre-reform measure of exposure (1977-1979). The 1973 reform is not as useful since the closest moss lead exposure measure captures a mix of pre and post-reform levels of exposure (1972-1974). The 1986 reform is also less useful both since it targeted only regular grade gasoline which implies that the changes in lead exposure are much more gradual than following the 1980/81 reforms which targeted all grades.

[38] As shown in Figure 1 children's blood lead levels dropped by 30% between 1979 and 1983. In a repeated sample of adults, the blood-lead levels in Stockholm dropped by 30% between 1980 and 1983 and air lead levels between 1979 and 1982 dropped by 50% in Stockholm (Elinder et al. 1986).

$$(5)\ \ Y_{nc} = \alpha + \sum_{c=1976}^{1984} \gamma_c \left(\lambda_c \times \text{Lead}_n^{\text{Pre-Reform}}\right) + \theta X_{nc} + \delta Z_{mc} + \lambda_n + \lambda_c + \varepsilon_{nc}$$

where $Lead_n^{Pre-reform}$ is the local air lead levels just before the reforms (1977–1979), which we interact with the birth cohort dummies (λ_c). The parameters of interest are the cohort-specific coefficients (γ_c). In essence, these coefficients provide estimates of the effect of the reform across areas with different predicted changes in lead exposure generated by the reform. Using the predicted reform-induced variation in exposure, rather than the actual exposure, removes the potential confounding by unobserved factors that may determine actual exposure and affect children's adult outcomes. In this analysis, we are also able to directly control for local gasoline sales. We use the children born in 1978 as the reference cohort since they are aged above 3 at the time of the full reform and have hence passed the critical age (24 months) when lead take up and B-Pb levels start to decrease (Canfield et al. 2003).

However, we do take our earlier findings into account in this analysis. In particular, before the 1980/81 reforms, the median pre-reform exposure level is 29 mg/kg (see Figure A3.). Hence, at least for the human capital outcomes, the estimated threshold documented in Section 4.2 is well within the boundaries of the pre-reform exposure levels. Accordingly, we provide separate estimates of equation (5) for samples above and below the estimated human capital threshold (30 mg/kg). This provides us with four predictions on how the γ_c coefficients should evolve across the birth cohort, depending on the outcome and the pre-reform lead exposure in the neighborhoods of birth. We expect the human capital outcomes for:

i) children in neighborhoods with pre-reform levels above 30 mg/kg to improve after the reforms, and
ii) children in neighborhoods with pre-reform levels below 30 mg/kg to remain unchanged after the reforms.

For Property and Violent crime the estimated threshold (65 mg/kg) is virtually out of range at the time of the 1980/81 reforms and hence, we expect that Property and Violent crime for

iii) and iv) children in neighborhoods above or below 30 mg/kg both should remain unchanged after the reforms.

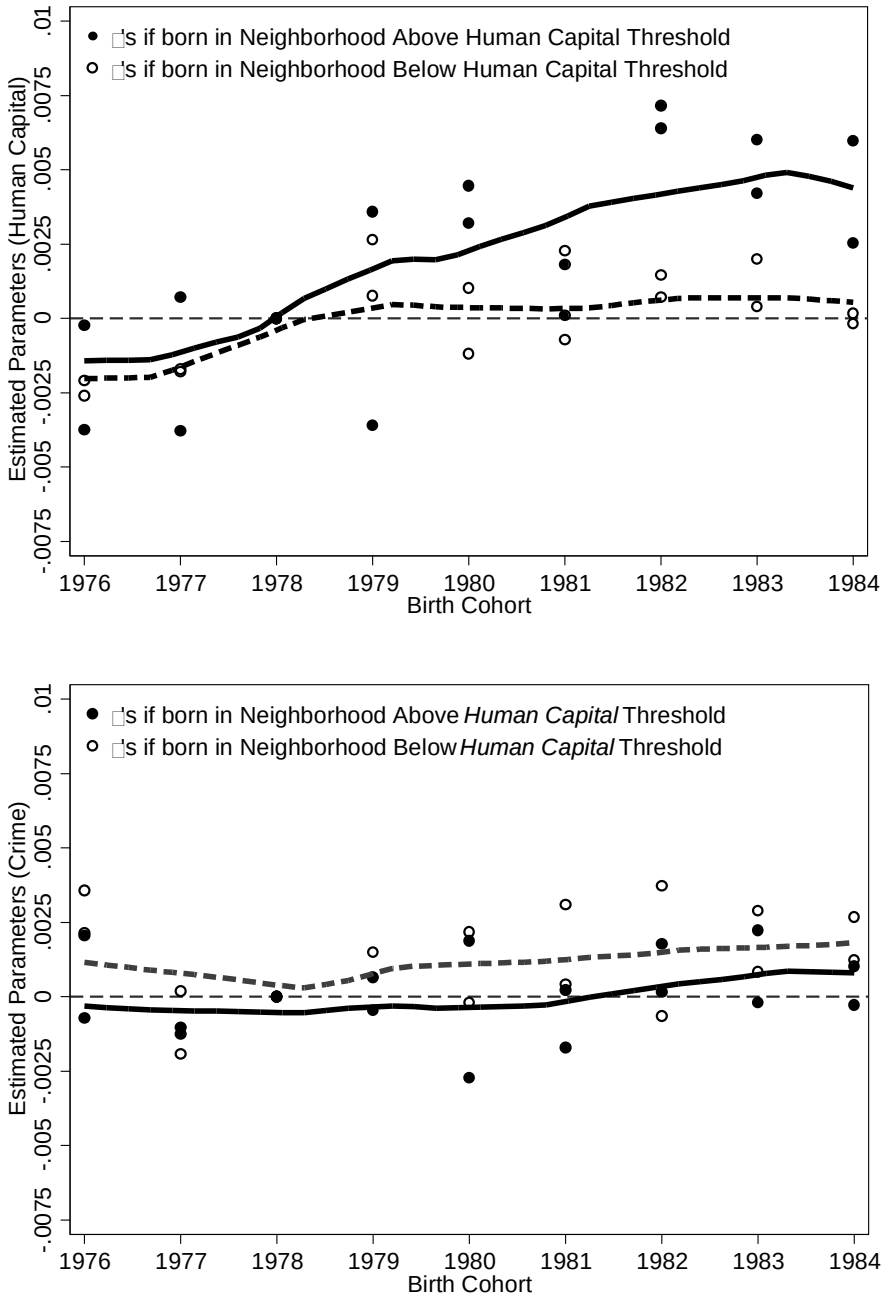
We present the point estimate and point-wise 95%-confidence intervals for the γ_c's (in Equation 5), together with joint *F*-tests of equality of the γ_c coefficients before and after the reform (i.e. standard difference-in-differences) for each outcome in Figure A11 and Figure A12. For brevity, we summarize these results by first standardizing the outcomes and plotting the coefficients from the four separate regressions for human capital outcomes (top) and crime outcomes (bottom) in Figure 4. The black dots represent point estimates of the γ's for children in the above-exposure-threshold neighborhoods and the hollow circles represent the γ's estimates for children in the below-threshold neighborhoods. The sold line is the local average of the coefficients (weighed by the inverse of the standard errors) in the above-threshold neighborhoods and the dashed line is the corresponding averages in the below-threshold neighborhoods.

The pattern for the different outcomes and exposure groups summarized in Figure 4 follows the outlined predictions. The trends in the coefficients for the cohort exposed to the reform at older ages do not differ before the reform between children in above and below threshold neighborhoods and are not statistically distinguishable from zero. For human capital, the coefficients in above and below threshold neighborhoods start to diverge for the 1979 birth cohort (age 1 at the first reform). *F*-tests of the pre vs. post reform coefficients (i.e. 1976–78 vs 1979–84) are statistically distinguishable at the 5%-level in the above threshold neighborhoods, but not statistically distinguishable in the below threshold neighborhoods. For Crime, as expected, the differences before vs. after the reform in the above and below threshold neighborhoods are small and not statistically distinguishable from zero with few exceptions.[39] [40]

[39] Two exceptions are worth mentioning. The first is the estimates for Property Crime for which the *F*-test suggests that there could be a reduction in crime in the low exposure areas, which is contrary to our expectations. For completeness, we also report the cohort-specific estimates for Any crime in Figure A13 despite the uncertainty of the location (if any) of the threshold and the failure to detect non-linearities (c.f. Figure 3 and the Hansen LR statistic for Any Crime in Figure A9). From Figure A13, we can see that after the reform, there is some indication that the probability of ever being convicted decreases following the reform in high-exposure areas but not in the low-exposure areas. This might suggest that for the more noisy Any Crime category, there exists a threshold at similar locations as for human capital outcomes. However, we interpret this finding for Any Crime with some caution given the impreciseness of the estimates in Figure 3 and Figure A9.

[40] For women, the coefficients mirror the main results. There are no clear effects of the 1980/81 reforms on women (see Appendix A Figure A14).

Figure 4. The estimated impact of the 1980/81 reform on human capital and crime



Notes: The figures summarize the result from estimates of equation (5). Here the outcomes are standardized before estimation (mean 0, SD 1) which allows for comparison across outcomes (See in Appendix Figure A11 and Figure A12 for full results). The black dots represent point estimates of Pre-reform lead exposure interacted with the cohort for children in high exposure neighborhoods and the hollow circles the estimates for children in low exposure neighborhoods (see text for details). The sold line is the local average (weighed by the inverse of the standard errors) of the coefficients from high exposure areas and the dashed line is the corresponding averages for children in low exposure areas using a bandwidth of 1 year. The dependent variables are for human capital (upper graph), compulsory school GPA and high-school completion and for crime (lower graph): P(Violent crime) and P(Property crime). The sample consists of males born in 1976-1984. Pre-reform lead exposure is the measurement reflecting exposure in 1977–1979. On top of the full specification controls (see note in Table 1) in this specification we are also able to control for municipality level gasoline sales ($1000m^3$).

In summary, the timing of the changes in outcomes documented in the analysis in this section generally reinforces our beliefs that the main results on adult outcomes reflect changes in childhood lead exposure caused by the reforms. Importantly, if the results in our main analysis were due to unobserved differential trends across high and low exposure neighborhoods, we would not expect to find such a systematic pattern around the timing of the 1980/81 reform. The results provide support for the existence of threshold effects and for the differential impact of low levels of lead exposure across genders.

4.4 The mediating role of cognitive and non-cognitive skills

In this section, we directly examine the relevance of the two mediating channels highlighted in the conceptual framework: cognitive and non-cognitive skills. Distinguishing the role of these two sets of skills is interesting since it could provide additional supporting evidence for the internal validity of the main results, and serve as a comparison to effects of other early interventions (e.g. Heckman et al. 2013; Nilsson forthcoming). But it is also interesting for guiding the design of effective remediating policies following lead exposure: cognitive and non-cognitive skills differ in their malleability to intervention across the child's life-cycle. While cognitive ranks seem to stabilize around the age of 10, non-cognitive skills are malleable at least up to the early 20s (Francesconi and Heckman 2016). Hence, information on whether the effects on adult outcomes are mainly driven by cognitive or non-cognitive skills can provide important information about the appropriate types of intervention and the window of opportunity for remediating investments following early lead exposure.

Figure 5 provides the semi-parametric estimates for cognitive and non-cognitive skills. The top figure shows that there is no clear and consistent exposure-response pattern for cognitive skills. On the contrary, the bottom figure shows that for non-cognitive skills, the exposure-response pattern is similar to the non-linear shape documented for the adult outcomes. Below 30 mg/kg, the slope is not statistically distinguishable from zero, while above this level, it is consistently and significantly downward sloping.

It is also possible to go into even further detail by directly examining the effects on the four sub-scores that make up the overall composite non-cognitive score. Semi-parametric estimates for each of the sub-scores are presented in Figure 6. The estimates in this figure suggest that the overall effects are driven by effects on psychological

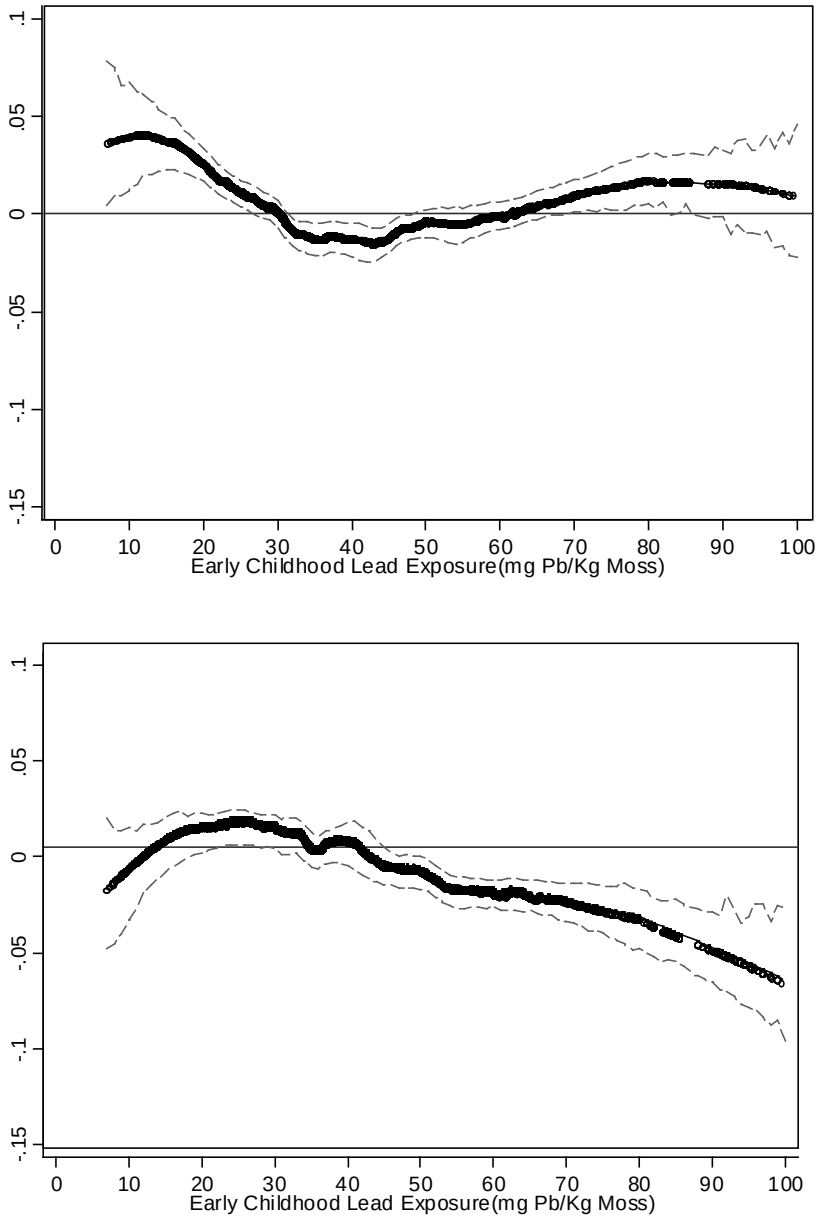
energy (Conscientiousness) and emotional stability (Neuroticism), and social maturity (Extraversion). These personality traits have previously been closely linked to externalizing behavior (e.g. aggression, hyperactivity, antisocial behavior) (Almund et al. 2011) and labor market outcomes, which is the key explanatory factor in the crime reducing effects of the Perry Pre-school program (Heckman et al. 2013).

4.5 Effects on labor market outcomes

Our analysis focuses on the outcomes for which the children in our sample are well-measured at the ages when we are able to observe outcomes for all cohorts. Within OECD, Sweden is one of the countries with the highest age when obtaining the first university-level degree (average age 29) and 40% of each cohort graduate from higher education (OECD 2014). This implies that measuring and examining the effects on labor market outcomes before the mid-30s is problematic, since many are still enrolled in higher education and hence only have a loose connection to the labor market.

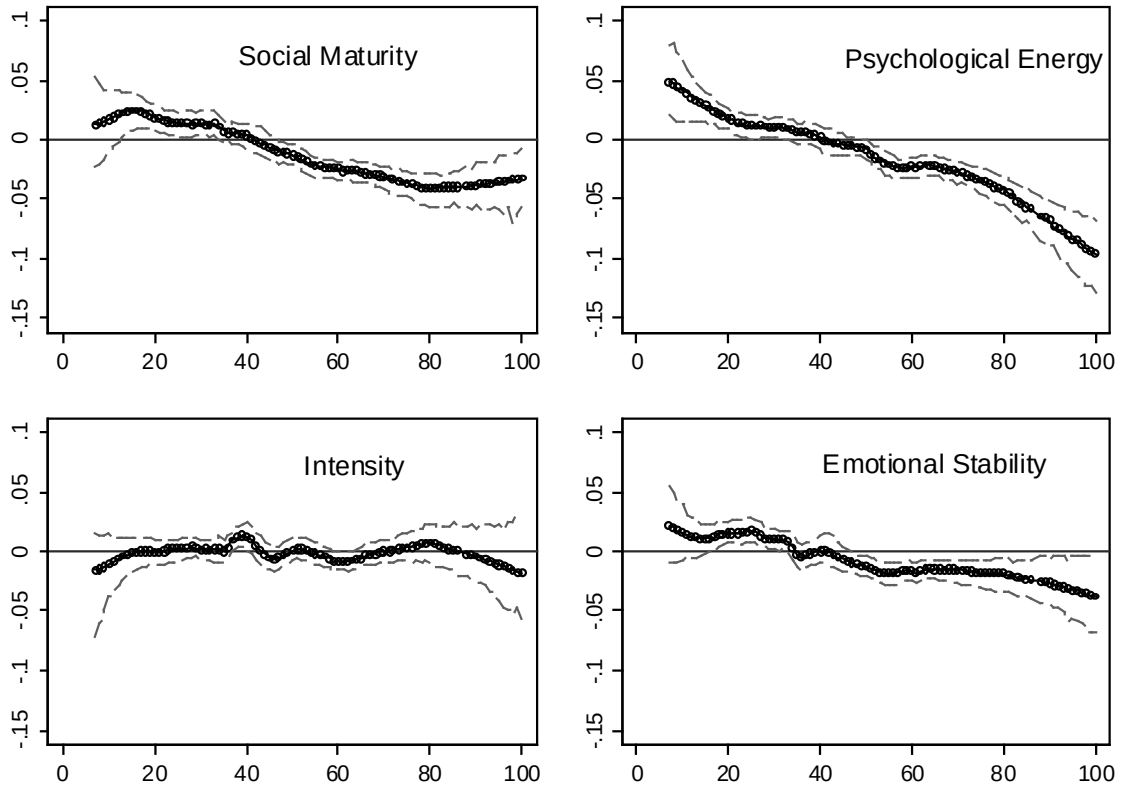
With this caveat in mind, we examine the effects on earnings in Appendix D. In 2013, at ages 29–40, we find little evidence that early lead exposure, at the low-levels of exposure to lead that we observe, has any systematic effect on earnings. Future work could use the same analysis to examine effects on earnings at ages when current earnings better reflect permanent income. Böhlmark and Lindquist (2006) show that to obtain credible measures of permanent earnings require current income to be measured in the early to mid-30s for men and even later for women. To get a sense of the economic magnitudes, in the next section we do instead impute the predicted effects on mid-30s earnings using the estimated impact on high school completion.

Figure 5. Semi parametric estimates for cognitive and non-cognitive skills



Notes: The graphs present estimates along with a 95 % confidence interval (bootstrapped using 1000 repetitions) from semi-parametric fixed effects models (see Baltagi and Li 2002). The dependent variable in the top graph is cognitive skills and in the bottom graph non-cognitive skills. Both outcomes are standardized (mean 0, SD 1). The sample consists of males born in 1972–1974, 1977–1979. Childhood lead exposure is measured as an average over ages 1–3. Besides neighborhood and birth cohort fixed effects, the model controls for the full set of controls as specified in the note of Table 1.

Figure 6. Semi parametric estimates for components of non-cognitive skills



Notes: The graphs present estimates (males only) along with a 95 % confidence interval (bootstrapped using 1,000 repetitions) from semi-parametric fixed effects models (see Baltagi and Li 2002). The non-cognitive score is based on a 20- to 25-minute interview with a psychologist resulting in four different scales, all ranging from 1 to 5, here displayed with their respective sub-categories and the Big-Five traits of Personality ((***O***)peness to Experience, (***C***)onscientiousness, (***E***)xtraversion, (***A***)greeableness, (***N***)euroticism) to which they are related: **(i) Social maturity** (extraversion (*E*), having friends (*E*), taking responsibility (*C*), independence (*O*)), **(ii) Psychological energy** (perseverance (*C*), ability to fulfill plans (*C*), to remain focused (*C*)), **(iii) Intensity** (the capacity to activate oneself without external pressure (*C*), the intensity and frequency of free-time activities (O)), and **(iv) Emotional stability** (ability to control and channel nervousness (*N*), tolerance of stress (*N*), and disposition to anxiety (*N*)) (see Nilsson 2016)). All outcomes are standardized (mean 0, SD 1). The sample consists of males born in 1972-1974, 1977-1979. Childhood lead exposure is measured as an average over ages 1-3. Besides neighborhood and birth cohort fixed effects, the model controls for the full set of controls as specified in the note of Table 2.

5 Discussion and economic significance

The existence and location of a moss-lead threshold value are interesting and policy relevant in themselves since at least 28 European countries measure local lead levels in moss with regularly, and many regions an cities also do so with more irregular intervals.[41] But in order to attain a rough estimate of to which blood lead level the estimated moss -lead threshold value correspond, we use the model estimated in Nilsson et al. (2009) that relates moss lead to children's blood lead for the period preceding the ban of

[41] The city of Portland is a recent example. *http://projects.oregonlive.com/air-pollution/heavy-metals/moss/ http://www.oregonlive.com/environment/index.ssf/2016/06/more_moss_studies_aim_to_uncov.html*

leaded gasoline (see Appendix B for a review of the method). Evaluating this model at the means of the independent variables, after adjusting the blood-lead moss-lead elasticity using the age-specific blood-lead gasoline-lead elasticity estimated in Reyes (2007), and under the additional assumption that the additive separable specification used in the estimation holds for both populations, the relevant blood lead level for property and violent crime (~65mg/kg) corresponds to an early childhood blood-lead level of about 5.7 μg/dL. The corresponding figure for the high school completion threshold is approx. 3.5 μg/dL.

However, it is important to keep in mind that we use the neighborhood lead levels as the exposure measure; hence, the documented threshold effect for crime associated with a *neighborhood* average blood-lead level above 5 μg/dL. In principle, it is possible that the entire effect could be caused by large effects on development among a few children with very high blood lead levels. However, since the standard deviation in childhood blood lead levels is not extremely large this seems less likely. Still, the exactness of this blood lead level "threshold" remains to be confirmed in future research using individual childhood blood lead levels and individual adult outcomes together with a credible empirical identification strategy.

With these caveats in mind, it is interesting to consider the effects on earnings if early childhood blood lead levels would decrease from 10 μg/dL to 5 μg/dL. The Swedish high school graduation premium on earnings at the age of 32 is 17% (Nilsson 2009). By combining this estimate with the estimated effects of lead exposure on high-school graduation rates, the effect of reducing early childhood blood lead levels from 10 to 5 μg/dL implies that the life-time earnings (among males) would increase by 1.7% (17*0.1). Naturally, this estimate does only capture the part of the lead exposure effects on earnings that goes through high school completion.

In the US alone, 530,000 children aged below five have B-Pb levels above 5μg/dL, and 150,000 out of these are above 10μg/dL (CDC 2012). Using the above predictions, it is for example possible to calculate the hypothetical annual gains from reducing the B-Pb from 10 to 5μg among these children. Since general equilibrium effects are most likely not an issue, under the assumption that the earnings effects are directly translatable to the US setting, and given an annual income of USD 30,000, the benefits in terms of increased labor market earnings from reducing the blood lead level in these

children would hence be around USD $76 million annually after the age of 32 (30,000*0.017*150000). This reflects the effect on the average population of children, but since 60% of all children with a B-Pb above 10 μg/dL are eligible for Medicaid (see Currie, 2009), the expected effects on individual earnings could be larger.

6 Concluding remarks

We document the effects of early exposure to low levels of lead on adult outcomes, by exploiting the phase-out of leaded gasoline, a novel measure of lead exposure, and population-wide administrative individual data covering a wide set of key adulthood outcomes in Sweden. Contrary to the unexplained but "common finding" (in cross-sectional studies) that the marginal effects on cognitive and behavioral effects are larger at low lead levels (EPA 2013), we find that below certain thresholds, the relationship for non-cognitive skills, crime, and high school completion becomes much weaker.

An estimated 535,000 children in the United States have blood lead levels (BLLs) at or above the reference value for blood lead established by CDC in 2012 (5 μg/dL). 150,000 of these children's levels are ≥10 μg/dL. Globally, WHO have estimated that more than 40% of the children have blood lead levels above 5 μg/dL. Our results suggest that early lead exposure may have deleterious effects on the academic performance among children with blood lead at least at levels from 3 μg/dL and above, and criminal behavior from 5 μg/dL. The effects sizes are of a clear economic significance. While the exposed cohort is still too young to get a clear estimate of the effects on their labor market outcomes, our back of the envelope calculation suggests that going from 10 to 5 μg/dL implies a 10 percent increase in the probability of high school completion and, taking the non-linear effect into account, a 2.9 percentage point decrease in the probability of being convicted for property crime among males. In terms of earnings, solely via the effects on high school graduation, the same decrease corresponds to an increase by approx. 1.7%. Naturally, if the effects on crime and non-cognitive skills are also taken into account, the effects are likely to be larger.

In the United States, the age of the housing stock together with the neighborhood poverty status constitute a key predictor of elevated blood lead levels in children. Interestingly, we find that boys are more vulnerable to lead exposure, which suggests that early lead exposure could be one contributing explanation for why growing up in

disadvantaged areas is especially harmful for boys (Chetty et al. 2016). There is no evidence that early childhood blood lead levels differ by gender and hence, an important task for future research is to provide evidence on whether the larger effects on boys are explained by greater vulnerability, or whether parents, teachers or others differ in their responsive investments to behavioral changes induced by lead exposure between boys and girls.

In terms of mechanisms, non-cognitive skills seem to play a more important role than cognitive skills. This is an important finding since earlier studies have documented that non-cognitive skills are more malleable than cognitive skills. Hence, our study does not only provide causal evidence on the effects of low levels of lead exposure on adult outcomes, but we also provide key insights for effective parental and public remedial investments following early lead exposure. Our results suggest that interventions that specifically target non-cognitive skills are likely to be more successful than those focusing on cognitive skills.

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Appendix A.I.: Figures and tables

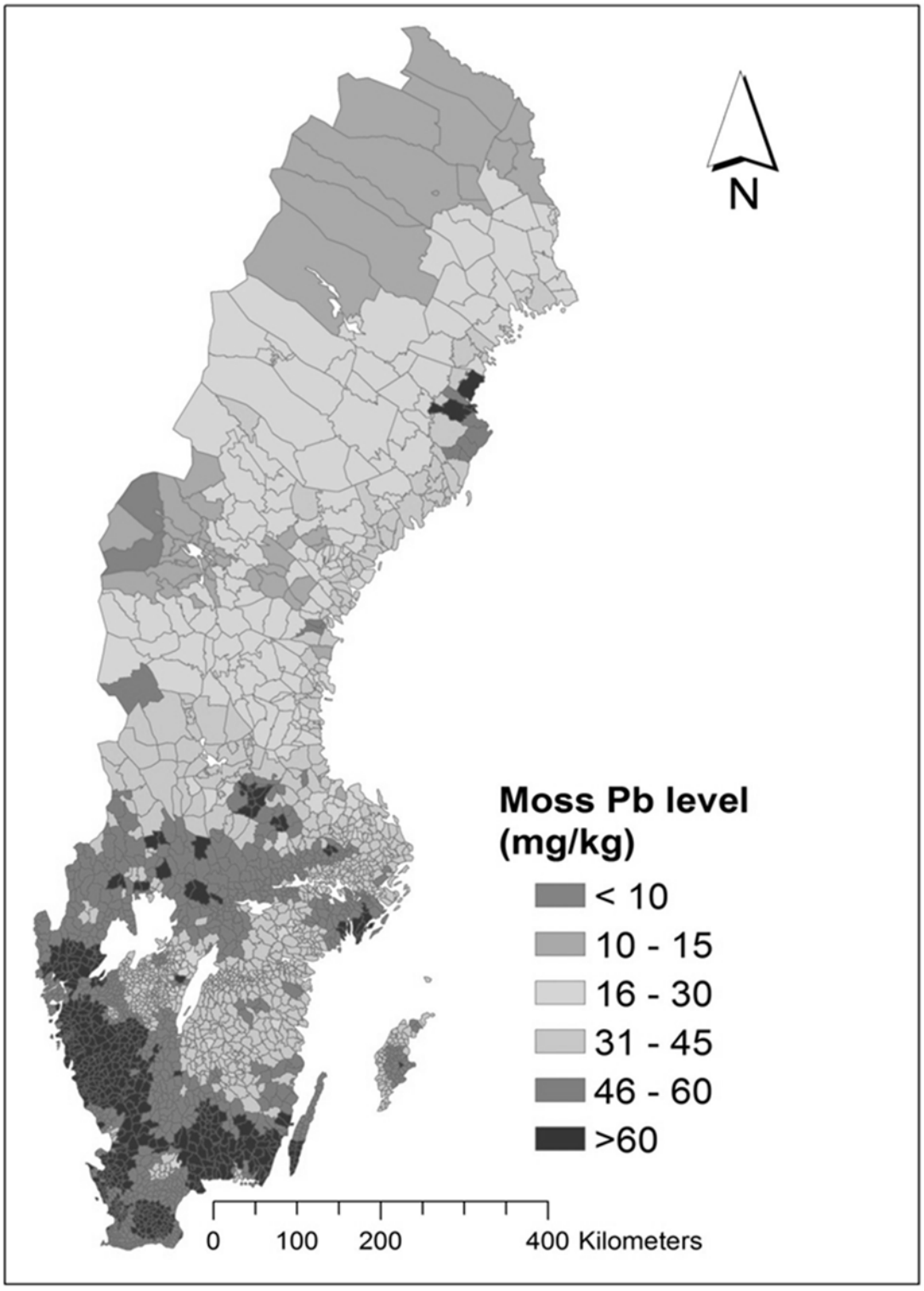

Figure A1. Neighborhood of birth lead exposure levels in 1975

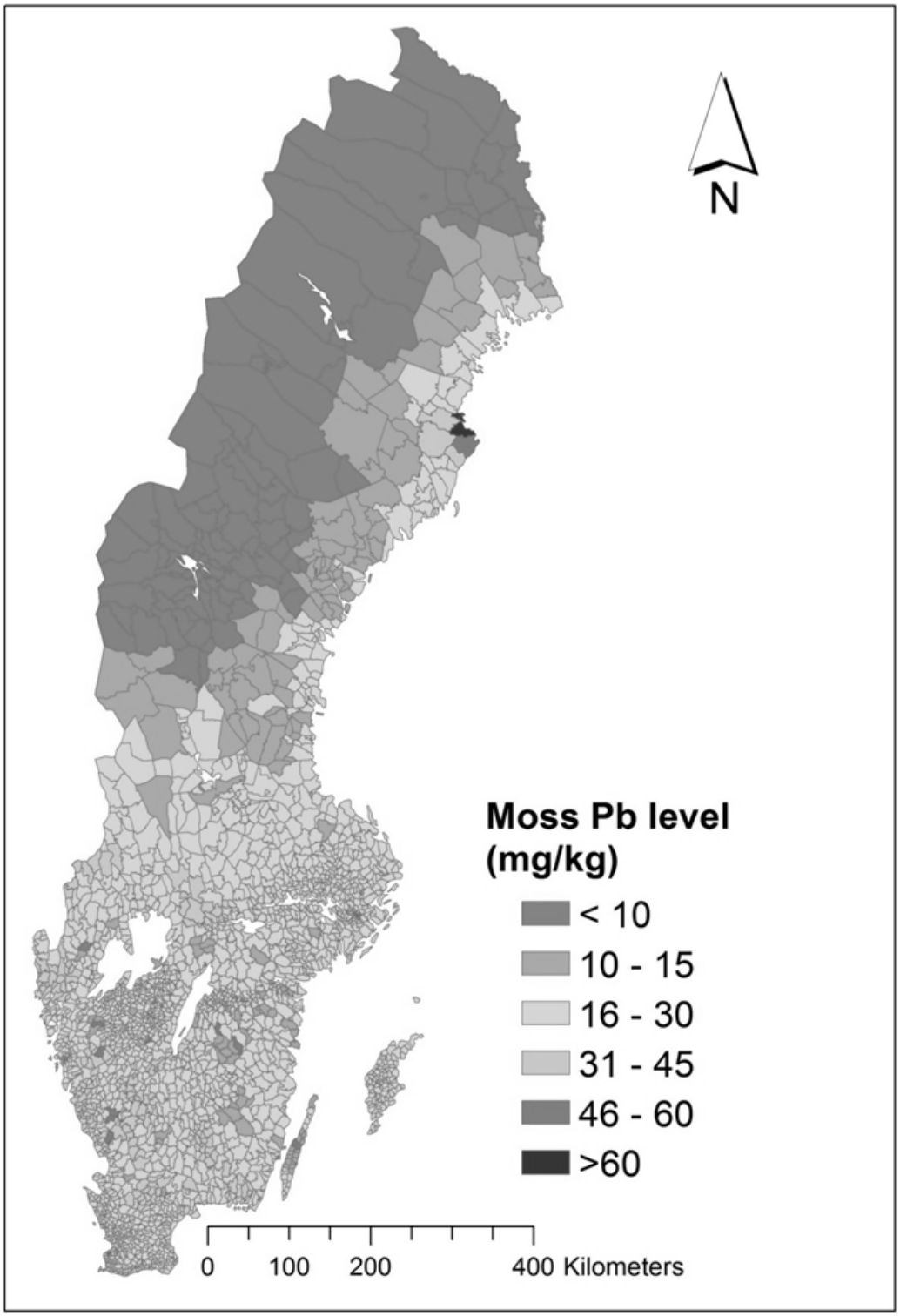

Figure A2. Neighborhood of birth lead exposure levels in 1985

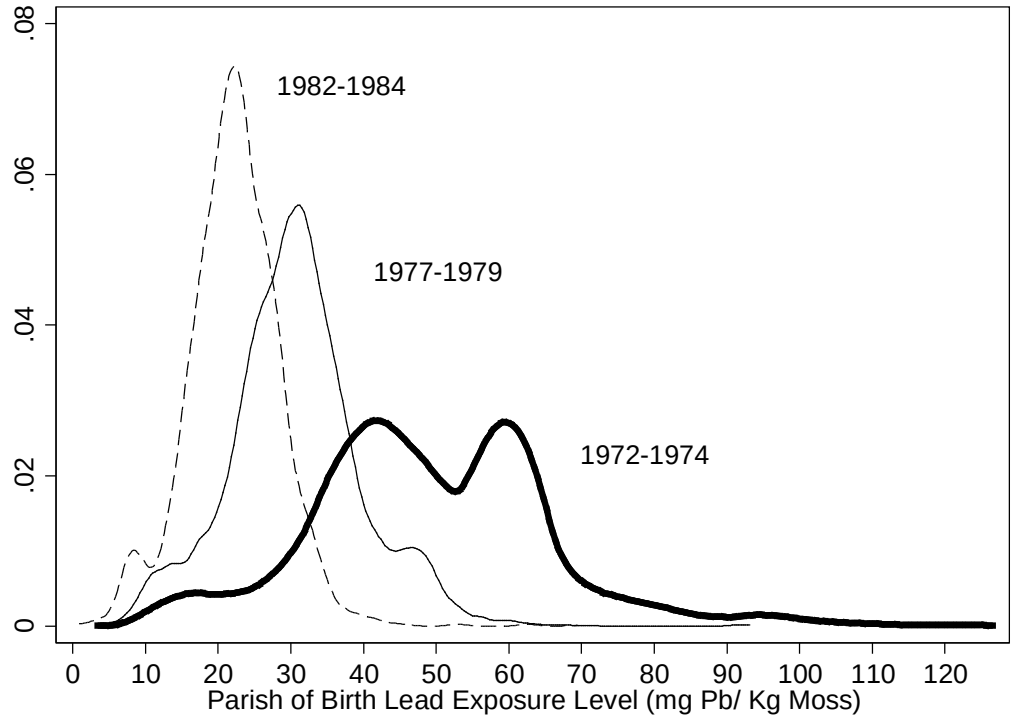

Figure A3. Kernel density distributions of moss lead levels across the neighborhoods

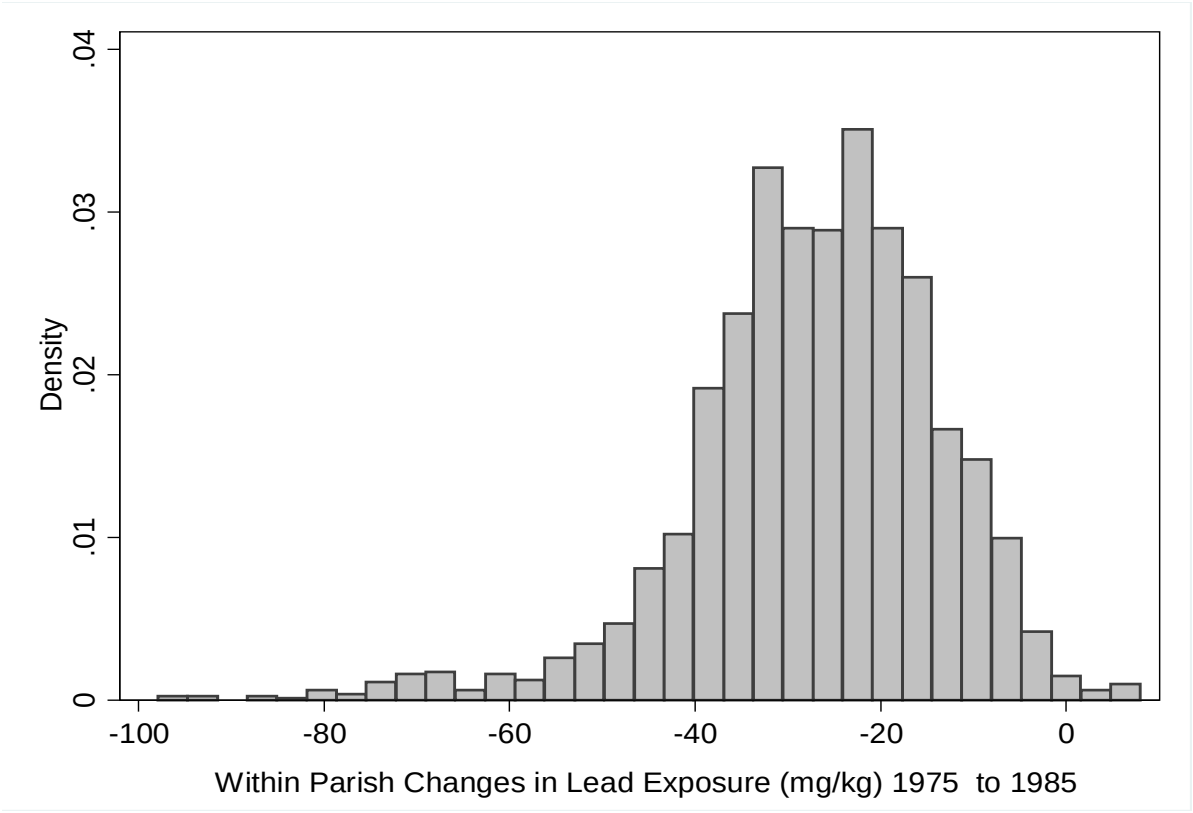

Figure A4. Distribution of changes in neighborhood lead levels

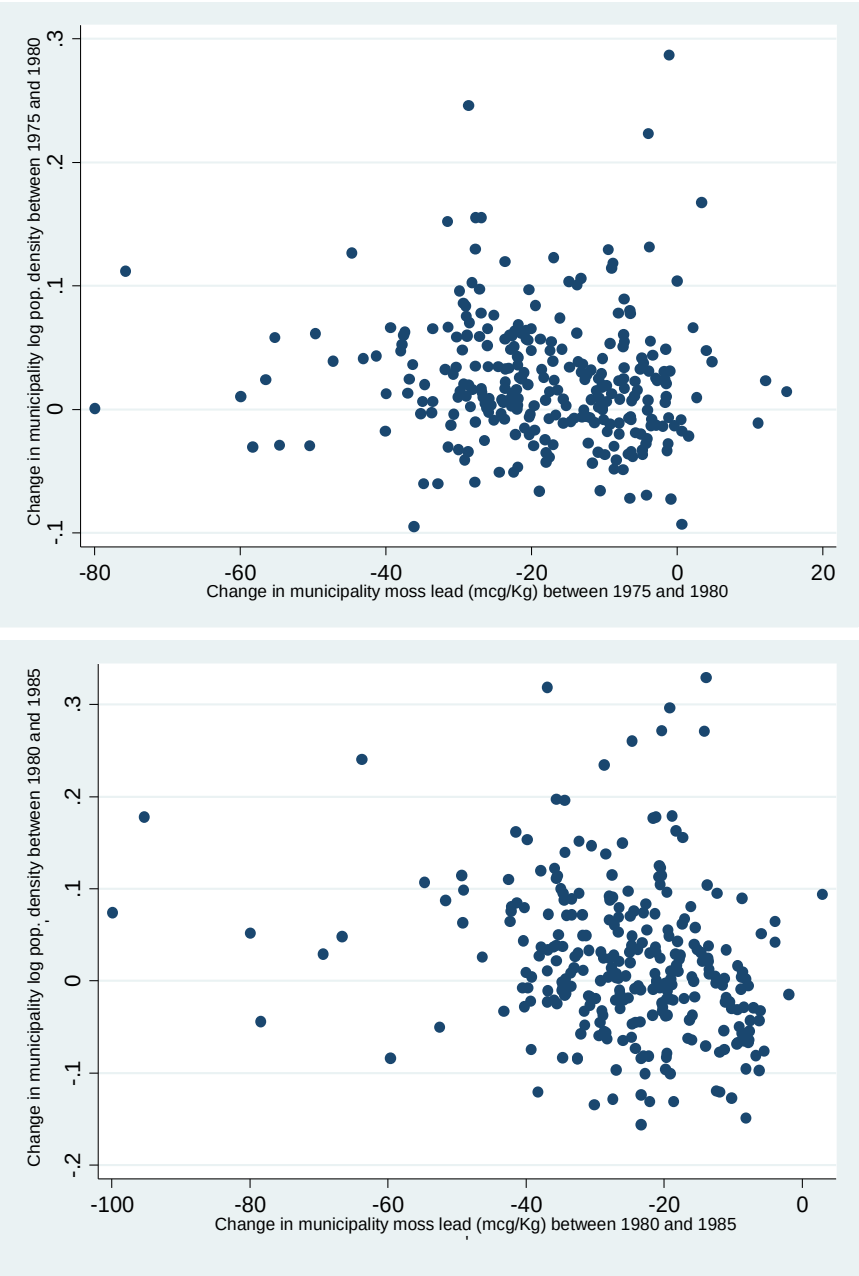

Figure A5. The association between changes in municipality moss lead concentrations and changes in (log) population density

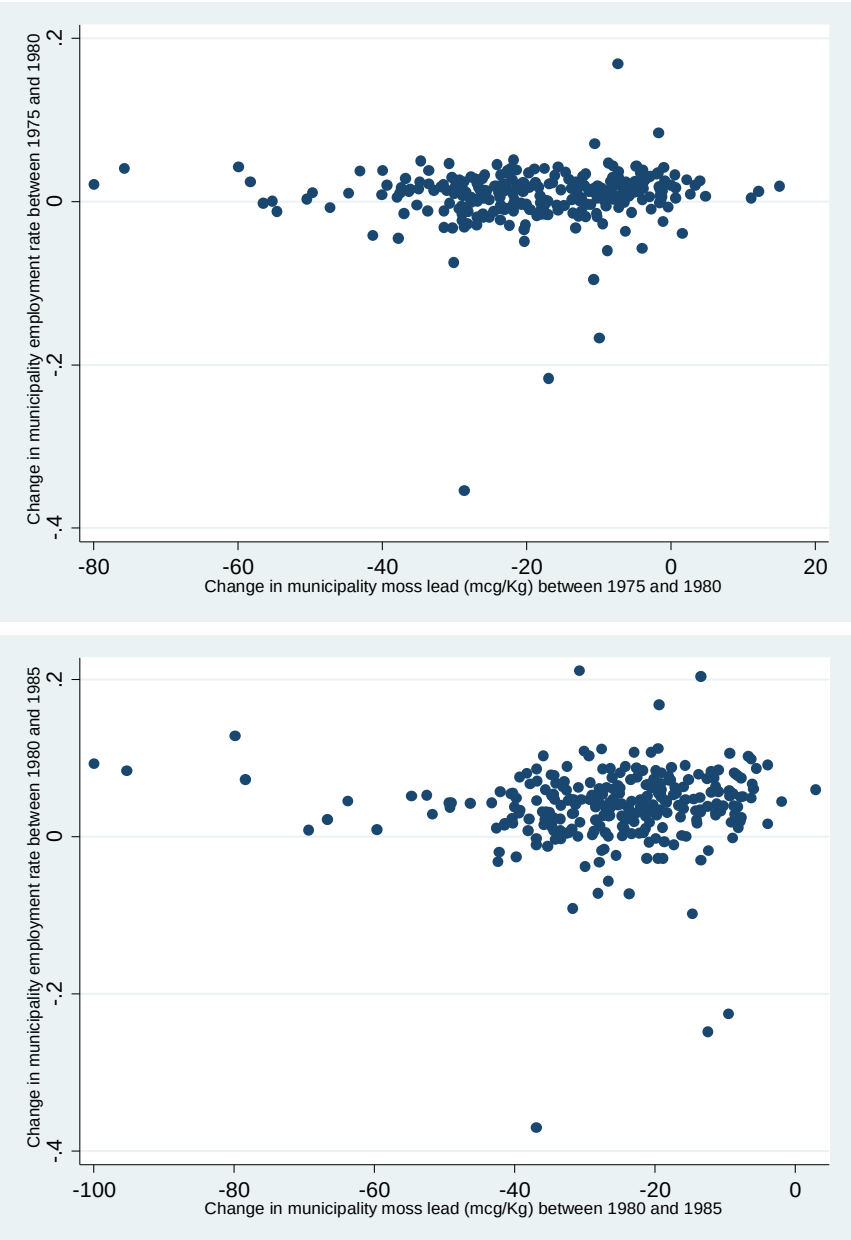

Figure A6. The association between changes in municipality moss lead concentrations and changes in municipality employment rate

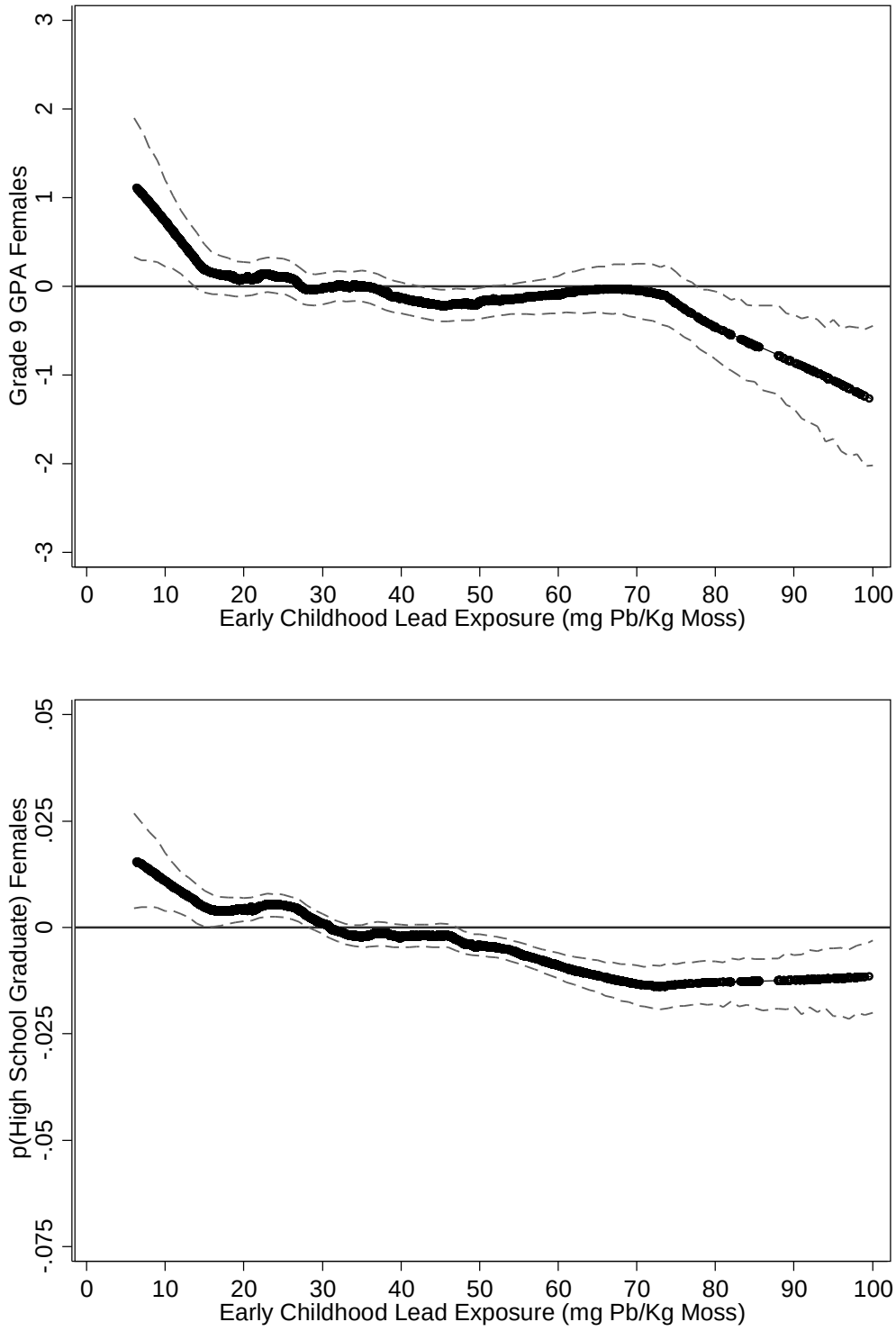

Figure A7. Semi-parametric estimates human capital (WOMEN)

Notes: The graphs present estimates along with a 95 % confidence interval from semi-parametric fixed effects models (see Baltagi and Li 2002). The dependent variable in the top graph is grade 9 GPA and in the bottom graph P(High school completion). The sample consists of males born in 1972–1974, 1977–1979 and 1982–1984. Childhood lead exposure is measured as an average over ages 1–3. See notes of Table 1 for full set of controls. The confidence interval accounts for clustering at the commuting zone level.

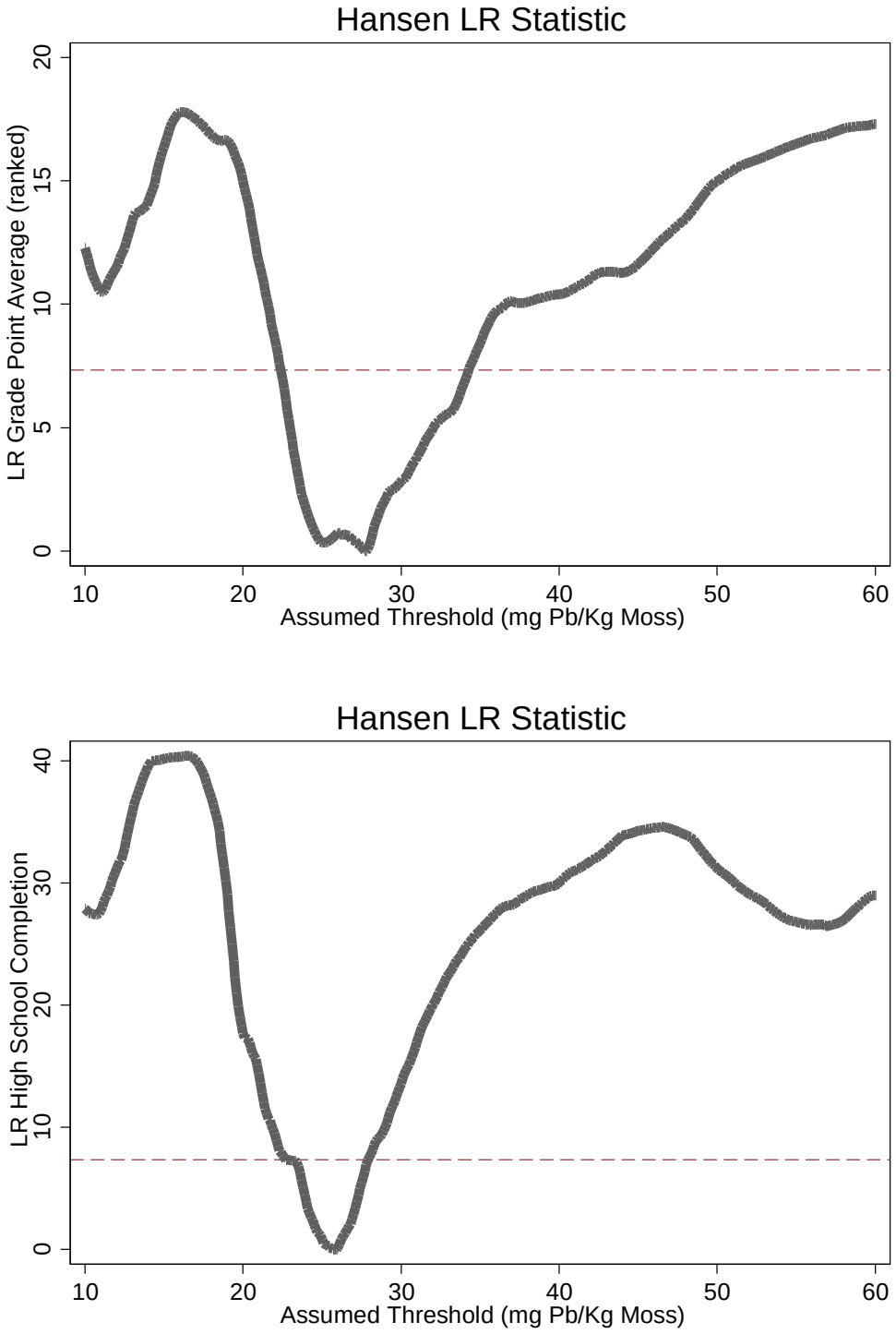

Figure A8. Hansen's LR statistic for human capital outcomes (MALES)

Notes: The graphs present Hansen's (1999) likelihood ratio test to locate the true threshold. The top graph presents results for compulsory school GPA and the bottom graph presents results for P(High school completion). The threshold is the assumed threshold at which the sum of squared residuals in the piecewise linear regression is minimized. The 5% critical value of the likelihood ratio is 7.35.

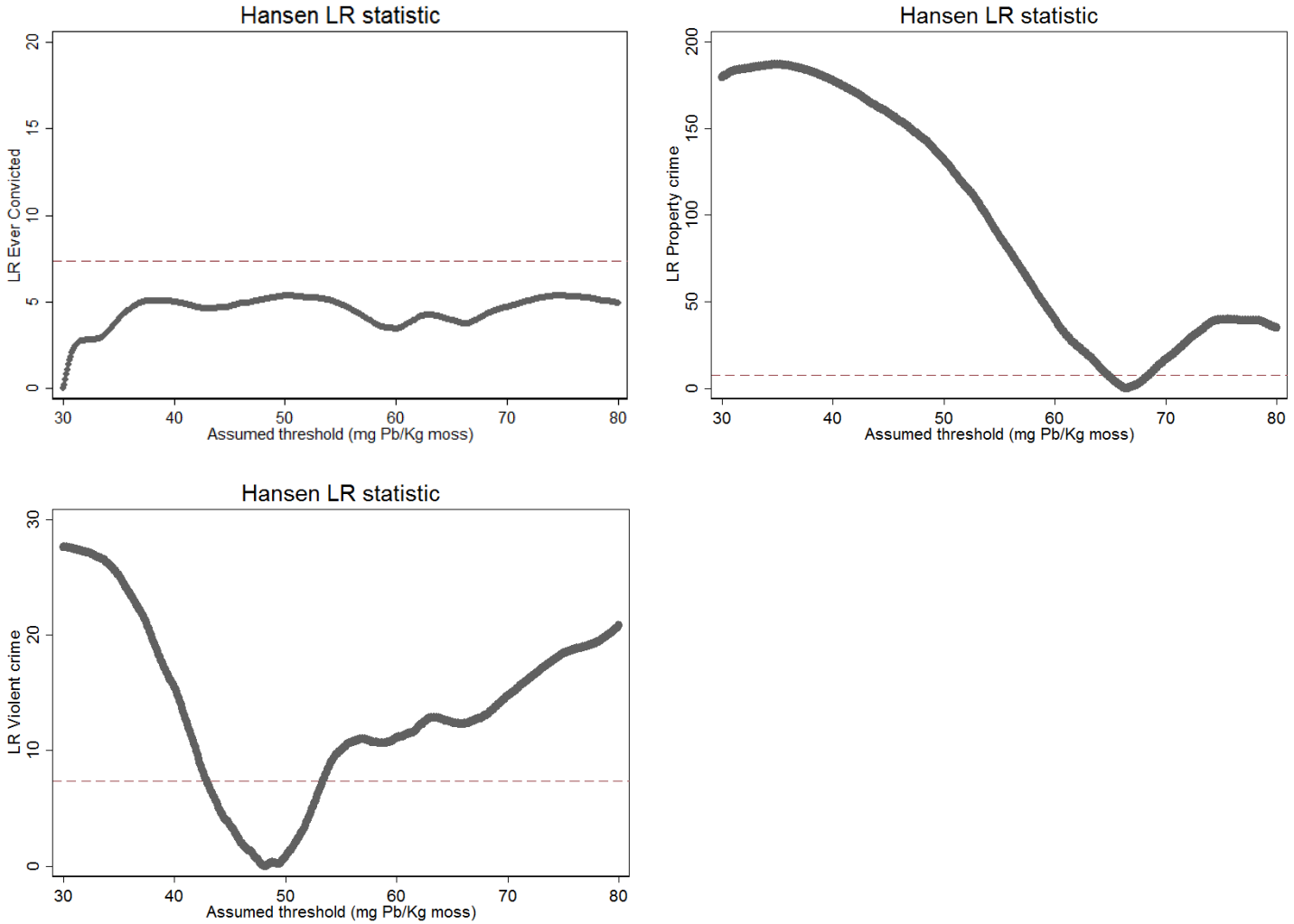

Figure A9. Hansen's LR statistic for crime outcomes. (MALES)

Notes: The graphs present Hansen's (1999) likelihood ratio test to locate the true threshold. The top graph presents results for P(Ever convicted), the middle for P(Violent crime) and the bottom for P(Property crime). The dashed horizontal line is the critical value for the no rejection region (i.e. it is not possible to reject that the true threshold is located at the locations where the LR takes a value lower than the critical value). The threshold is the assumed threshold at which the sum of squared residuals in the piecewise linear regression is minimized. Note that for Violent crime and Property crime we identify thresholds; however, for the ever convicted outcome, there is no location that is significant. The 5% critical value of the likelihood ratio is 7.35.

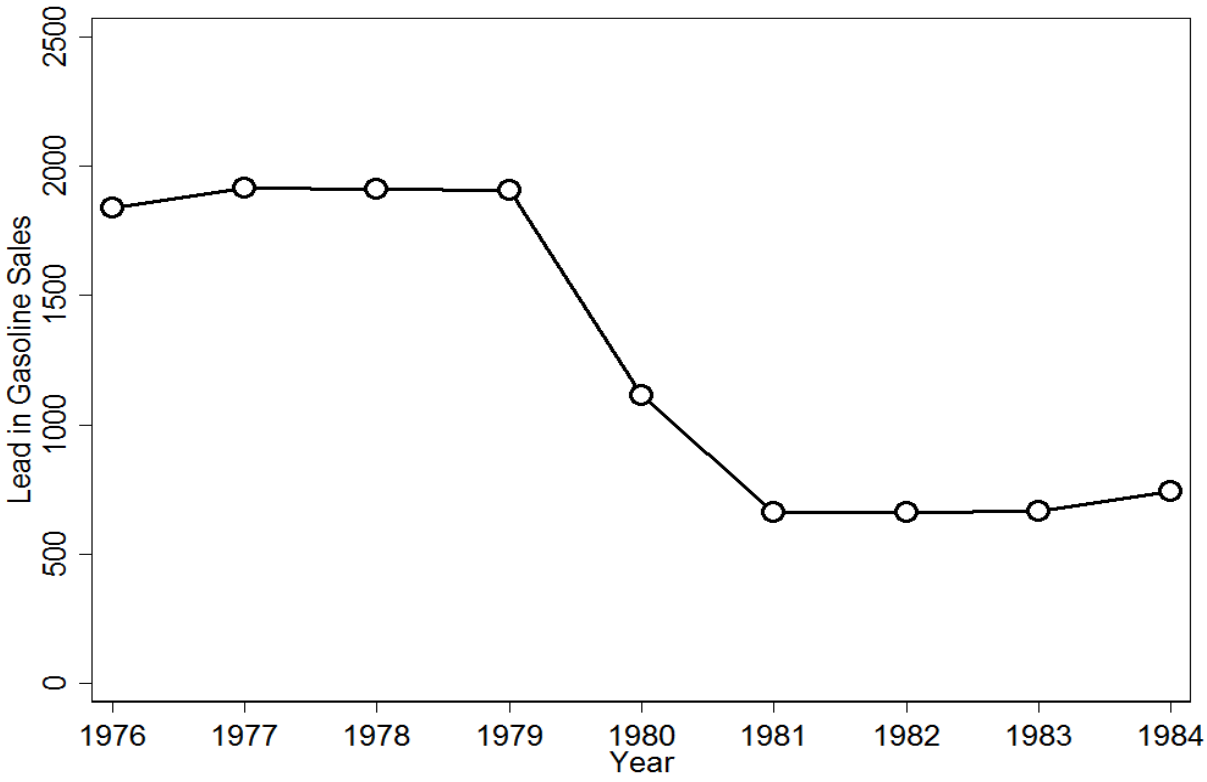

Figure A10. Maximum gasoline lead sales in metric tons per year

Note: The figure provides the maximum total lead sales over the years 1976–1984 using yearly data on deliveries of motor fuel (regular, medium, and premium grade gasoline) to final consumers. We multiply the delivered volumes by the fuel type specific maximum allowed lead content (for regular and premium gasoline, see Figure 1).

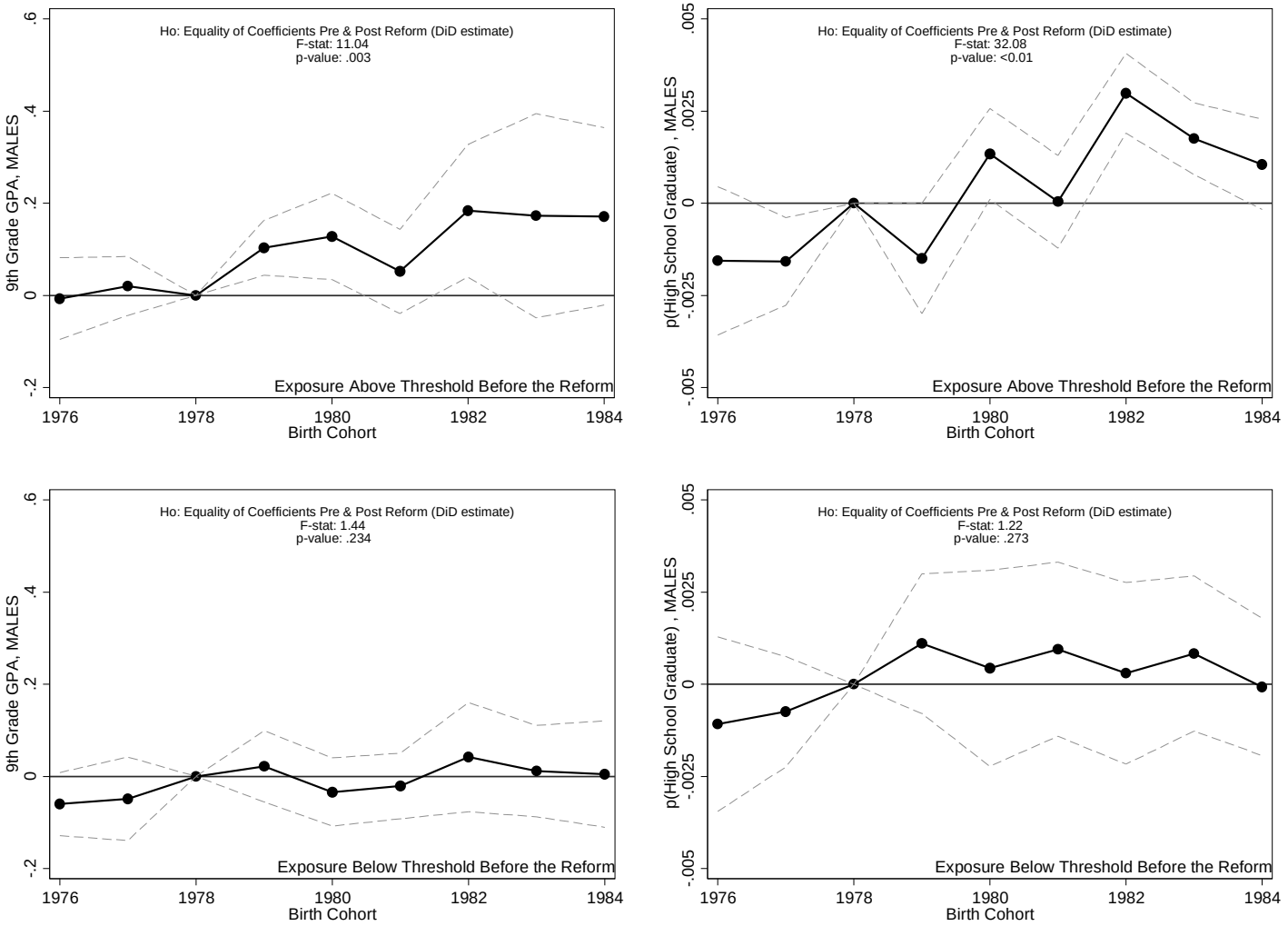

Figure A11. Human capital above and below the threshold (MALES)

Note: These figures show the point-estimates, point-wise 95% confindence bands from the estimation of the cohort-specific $\gamma's$ from equation (5) on the male sample together with the F-stats and p-values for a test of the equality of the γ coefficients before and after the reform (i.e. difference-in-differences for 1976–1978 vs.1979–1984 cohort coefficients). The top (bottom) left figure provides the estimates for 9th grade GPA among children born in neighborhoods with a lead level of above (below) 30mg/kg just before the reform. The top (bottom) right figure provides the estimates for high-school completion among children born in neighborhoods with a lead level of above (below) 30mg/kg just before the reform. The point estimates (and standard errors) provided in these four figures constitute the basis for the top panel of Figure 4. See text and notes to Figure 4 for further details.

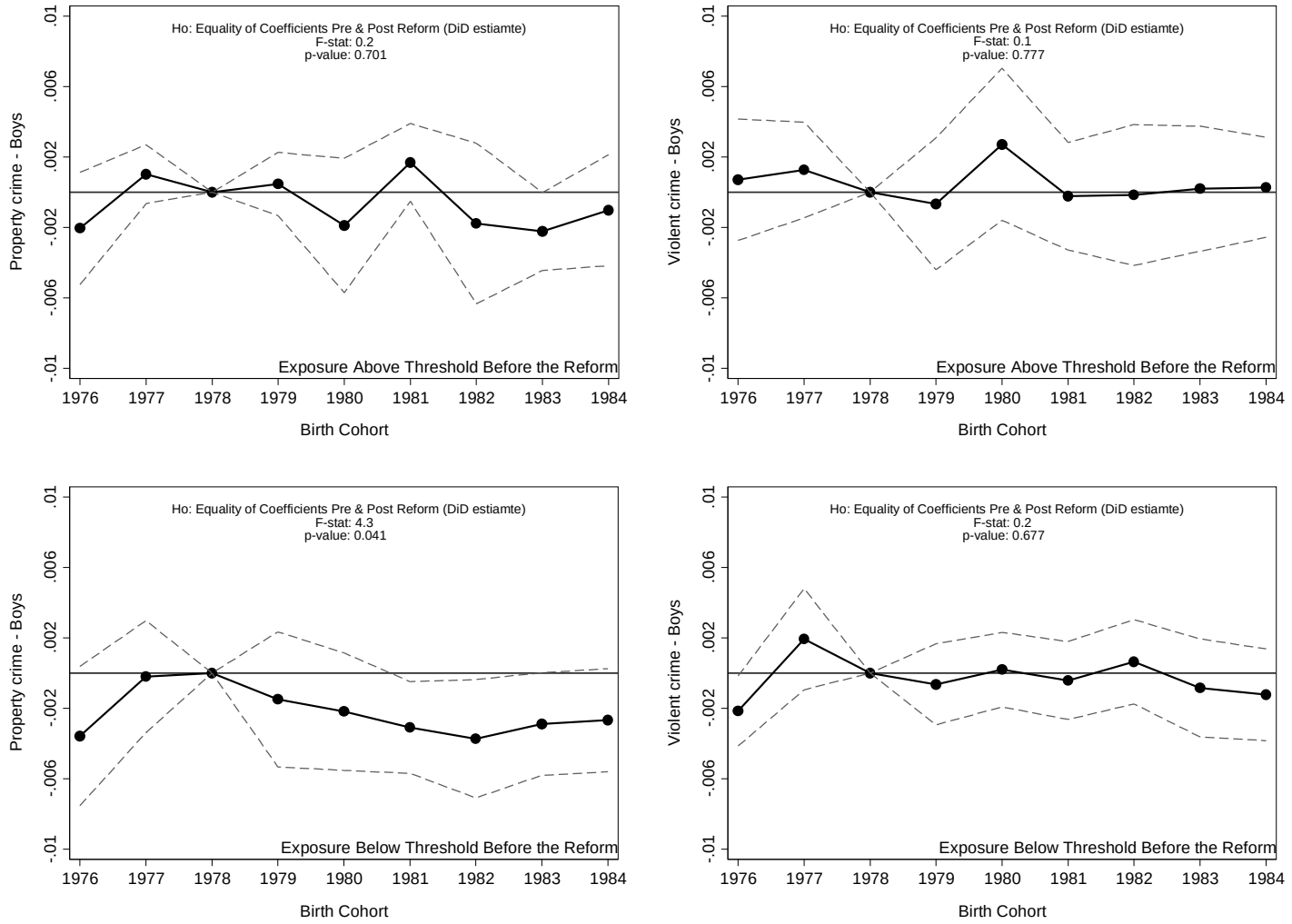

Figure A12. Violent and property crime above and below the human capital threshold (MALES)

Note: These figures show the point-estimates, point-wise 95% confindence bands from the estimation of the cohort-specific $\gamma's$ from equation (5) on the male sample together with the F-stats and p-values for a test of the equality of the γ coefficients before and after the reform (i.e. difference-in-differences for 1976–1978 vs.1979–1984 cohort coefficients). The top (bottom) left figure provides the estimates Violent Crime among children born in neighborhoods with a lead level of above (below) 30mg/kg just before the reform. The top (bottom) right figure provides the estimates for Property Crime among children born in neighborhoods with a lead level of above (below) 30mg/kg just before the reform. The point estimates (and standard errors) provided in these four figures constitute the basis for the Bottom Panel of Figure 4. See text and notes to Figure 4 for further details.

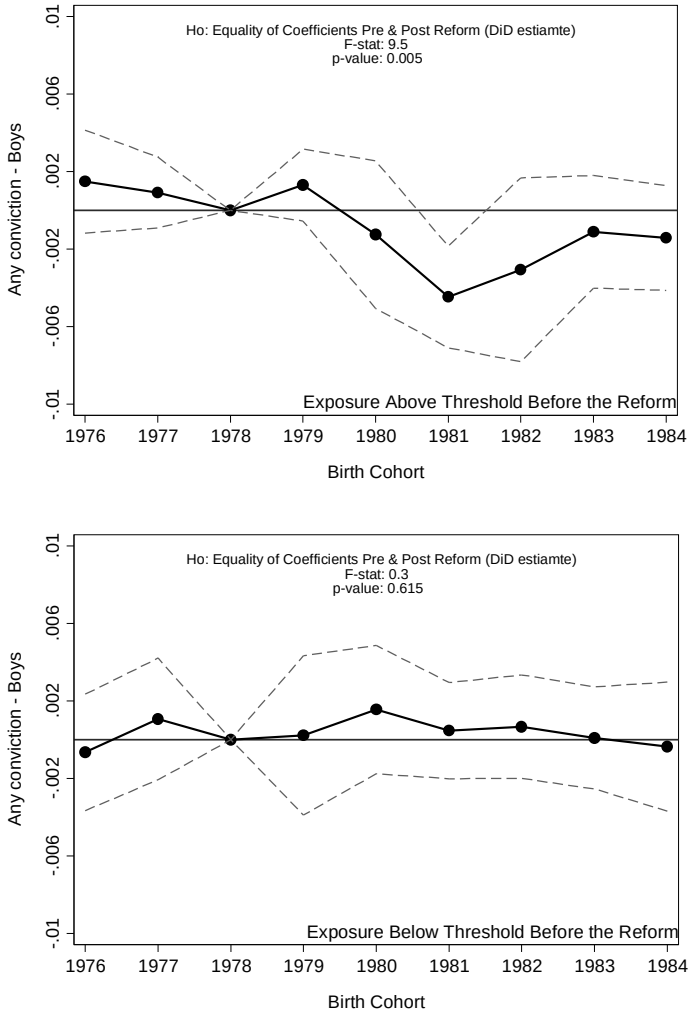

Figure A13. Any crime conviction in neighborhoods above and below the human capital threshold (MALES)

*Note:*See Figure A12 for details.

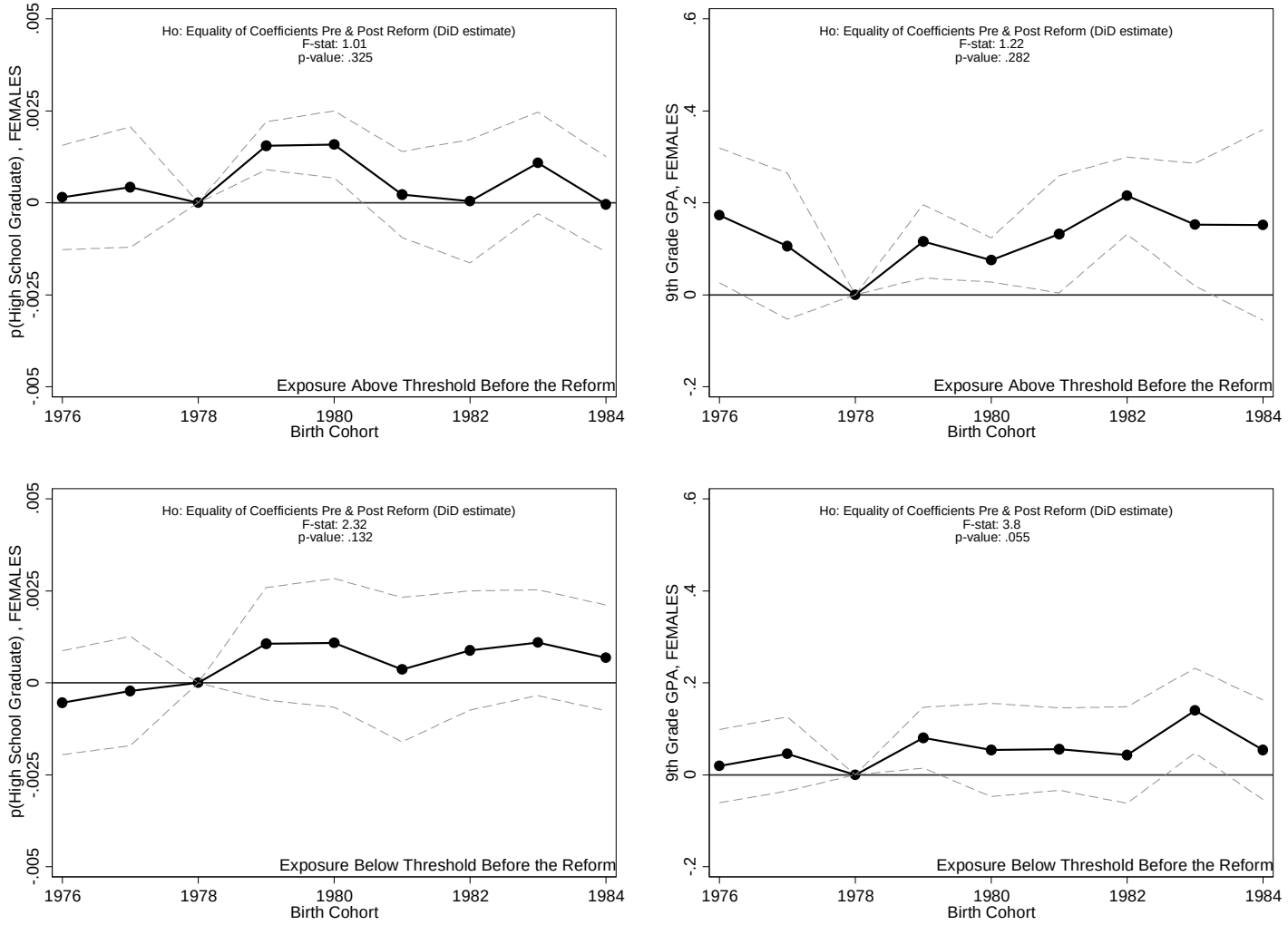

Figure A14. Human capital above and below the threshold (FEMALES)

Note: These figures show the point-estimates, point-wise 95% confindence bands from the estimation of the cohort-specific $\gamma's$ from equation (5) on the female sample together with the F-stats and p-values for a test of the equality of the γ coefficients before and after the reform (i.e. difference-in-differences for 1976–78 vs.1979–1984 cohort coefficients). The top (bottom) left figure provides the estimates for 9th grade GPA among children born in neighborhoods with a lead level of above (below) 30mg/kg just before the reform. The top (bottom) right figure provides the estimates for high-school completion among children born in neighborhoods with a lead level of above (below) 30mg/kg just before the reform.

Table A1. Descriptive statistics for outcomes used in the study

	Definitions	Mean	Std. dev.
GPA	Compulsory school grade point average (percentile ranked) [typically at age 16]	50.09	28.8
Cognitive Skills	Cognitive test score at military enlistment (percentile ranked) [age 18; boys only]	50.0	28.5
Non-Cognitive skills	Non-cognitive test score at military enlistment (percentile ranked) [age 18; boys only]		
High School	=1 if completed high school, 0 otherwise	.89	.31
Ever convicted	=1 if ever convicted in a criminal trial up to age 24, 0 otherwise	.164	.370
Property crime	=1 if ever convicted for a property crime (penal code: BRB Chapter 8) up to age 24, 0 otherwise	.070	.155
Violent crime	=1 if ever convicted for a violent crime (penal code: BRB Chapter 3) up to age 24, 0 otherwise	.024	.256

Table A2. Parents' predetermined characteristics and their children's lead exposure

PANEL A:	Parents completed college	Mother completed college	Father completed college	Parents' earnings 2 years before birth	Father's earnings 2 years before birth	Mother's earnings 2 years before birth	Age of mother at birth
Lead exposure (mg/Kg)	-.0005	-.0004	-.0003	.0004	.0004	-.0001	-.0004
	(.0004)	(.0004)	(.0004)	(.0003)	(.0003)	(.0006)	(.0029)
Neighborhood fixed effects?	No	No	No	No	No	No	No
PANEL B:	Parents completed college	Mother completed college	Father completed college	Parents' earnings 2 years before birth	Father's earnings 2 years before birth	Mother's earnings 2 years before birth	Age of mother at birth
Lead exposure (mg/Kg)	.0001	.0001	-.0000	.0003	.0003	.0003	-.0014
	(.0001)	(.0001)	(.0001)	(.0003)	(.0002)	(.0003)	(.0028)
Neighborhood fixed effects?	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: Standard errors (in parenthesis) are clustered at the commuting zone level. All regressions control for cohort of birth fixed effects.

Appendix A.II.: Sibling fixed effects

Table A3 shows results from sibling fixed effects estimates. Panel A provides OLS estimates for the sibling sample. Due to the gender specific effects and that we can only measure lead exposure in specific years, the sampling of siblings is somewhat more complex than in a standard siblings fixed effects model. In particular, we restrict the sample to mothers with at least two but at most three males born during the observation windows (cohorts 72–74, 77–79, 82–84). In addition, to exploit the policy driven variation rather than (potentially endogenous) changes in exposure from families that move between births, we require that at least one of the brothers must be born in a different lead measurement period (remember that we measure average lead over the three preceding years in 1975, 1980 and 1985).

For a comparison with the full sample, Panel A of Table A3 first provides the OLS estimates using this restricted sibling sample. Overall, the OLS estimate of the full sample is reasonably similar to the sibling sample estimates for males. Compared to the sibling sample, the full sample GPA and high school completion estimates are slightly higher, while the estimates for ever convicted and violent crime are higher, and similar for property crime. Turning to the siblings fixed effects estimates in Panel B, we see that as compared to the OLS estimates, the precision is first of all slightly worse, but the point estimates are not systematically different from the OLS estimates, which is reassuring. Some estimates are larger while others are smaller than the OLS counterparts, and none are statistically distinguishable from each other. Overall, we take this as supporting evidence that the estimates from the baseline empirical strategy for the full sample of children are valid.

Table A3. Sibling fixed effects estimates

	Grade 9 GPA (pct. rank)	P(High school completion)	P(Ever convicted)	P(Property crime)	P(Violent crime)
	(1)	(2)	(3)	(4)	(5)
	Panel A: OLS on Sibling Sample				
Early lead exposure	-.2112**	-.0044*	.0079***	.0016	.0028*
	(.1062)	(.0025)	(.0023)	(.0023)	(.0016)
	Panel B: Sibling fixed effects				
Early lead exposure	-.1410	-.0050	.0065**	.0024	.0015
	(.1810)	(.0035)	(.0031)	(.0023)	(.0030)
Neighborhood FEs	Yes	Yes	Yes	Yes	Yes
Year of birth FEs	Yes	Yes	Yes	Yes	Yes

Notes: The table presents the coefficient on early lead exposure from separate regressions. Panel B includes maternal fixed effects for individuals with up to three siblings. Coefficients and standard errors are scaled by a factor 10. Crime (in columns 3–5) is defined as having been convicted at least once up to age 24. The sample consists of male children born in 1972–1974, 1977–1979 and 1982–1984. Childhood lead exposure is measured as an average over ages 1-3. The baseline model includes controls for each parent's highest completed level of education (4 levels), total family earnings, maternal age at birth, birth order, family size, municipality population size, municipality employment rate, Cadmium, Zinc and Copper. Cluster robust standard errors (at the commuting zone level (74 cells)) are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % .

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

The relationship between blood lead and moss lead

This section briefly reviews 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 that Nilsson et al. (2009) use were 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 were then matched to the (venous) blood lead measurements from 396 children aged between 7 and 10, collected by Strömberg et al. (2003) in the year prior to that during which the mosses were sampled. Using the coordinates of the children's home address, each child is assigned an inverse distance weighted lead exposure level using the 10 nearest moss sampling sites.[42] The raw correlation between this lead exposure measure and children's blood lead level is 0.60, which compares very well with findings in previous studies linking ambient air pollution to actual population exposure.

Table A1 reports 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 B1., 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 suggests that for a 10% increase in the lead level in moss, the blood lead level increases by on average 3%. In columns (2)–(4), individual characteristics, fixed community, year of sampling and finally year*community fixed effects are stepwise introduced. Adding year and community fixed effects is the only control which influences 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=.73), 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 B1. Baseline model for lead

Dependent variable:	ln (BloodPb)	ln (BloodPb)	ln (BloodPb)	ln (BloodPb)	ln (BloodPb)	ln (BloodPb)
Specification:	(1)	(2)	(3)	(4)	(5)	(6)
Time period:	**All**	**All**	**All**	**All**	**Pre-Ban (<1995)**	**Pre-Ban (<1995)**
ln(Moss-Pb)	.2710*** (.0163)	.337*** (.0203)	.1834** (.0914)	.2111** (.0973)	.453*** (.1388)	.463*** (.1183)
Individual controls	no	yes	yes	yes	yes	Only standard
Neighb. fixed effects	no	no	yes	yes	yes	yes
Year fixed effects	no	no	yes	yes	yes	yes
Year* Neighborhood fixed effects	no	no	no	yes	yes	yes
R-squared	0.35	0.49	0.60	0.61	0.58	0.57
No of children	396	396	396	396	242	242

Note: The table reports regression results from OLS estimations of equation (B1). The blood lead is a measure in µg/dL blood. The lead exposure is mg/kg of moss. The full set of individual controls are gender, whether the child's practicing any lead exposing hobbies, ln(hemoglobin) level and school fixed effects. Standard errors are reported in parenthesis and are robust with respect to heteroscedasticity. */**/*** reflects significance at the 10/5/1 percent levels, respectively. Column (1) provides raw correlation, (2) adds individuals controls, (3) neighborhood and year fixed effects, (4) neighborhood by year effects, (5) restricts the sample to the pre-ban period, (6) drops the more esoteric control variables (i.e. hemoglobin level, and lead hobbies, and school FE) which are typically not available in most datasets. Nilsson, Skerfving, Stroh and Strömberg (2009)

The first four columns report the estimated elasticity using the full sample. However, in 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 shows that while the lead in moss continued to decrease throughout the observation period, the children's blood-lead levels leveled off at around 2 µg/dL after the ban on leaded gasoline. This 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 0.45 while the standard errors increase marginally. This result suggests that the relative importance of air-lead exposure was indeed stronger in the period prior to the ban than in the period after, as expected. The pre-ban period is also the period focused on in this paper. The final result also provides suggestive evidence on the validity of using mosses as monitors of air pollution.

Initial blood lead levels

The earliest systematic blood lead levels sampled among children in Sweden were conducted in 1978 by Strömberg et al. (1995). At that time, the blood lead level was just above 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 reports blood lead levels in the US in the same year of on average around 14 µg/dL. Before this point in time, we use the estimates of previous work by Reyes (2007) in order to assess the children's lead levels. She finds that an increase of 1 gram lead per gallon of gasoline increases the blood lead level by approximately 3.3 µg/dL in the general population. Taking her baseline specification literally and combining it with the data in Table 1 and the estimated intercept from Strömberg et al. (1995) B-Pb = 1.67 + 3.325 * grams/gallon) suggests that at the year of birth of the first cohort born in 1972–74, the average blood lead level would on average have been 6.7µg/dL.

A second way of assessing 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 additive separable specification used in the estimation holds for both populations, the relevant blood lead level for children aged 0–6 would, on average, correspond to about 5µg/dL.

A third way of estimating the initial blood lead level is to use the estimates in Strömberg et al. Based on repeated individual blood lead measurements, they find that individual blood lead levels in primary school children on average decrease by around 6% per year. Given the average blood lead 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, on average, be 8.5µg/dL. Since these samples were taken in a region with high lead exposure (based on moss lead values), it seems reasonable to assume that this level represents an upper bound of blood lead levels in the general population of children in these cohorts.

All three approaches provide estimates suggesting that the initial blood lead levels in the sample period were on average below 10µg/dL for the cohorts born between 1972

and 1974. Using the blood lead level standard deviation (1.5 µg/dL) of school children (age 10) in Strömberg et. al suggests that 97.5% of the first cohort of children in our sample had blood lead levels below 8 µg/dL, 9.7 µg/dL, or 11.5 µg/dL depending on the preferred estimate of the early childhood mean blood-lead level of the children born in 1972–1974.

How does lead exposure affect development?

Although lead exposure may have direct health consequences such as anemia, kidney failure or ultimately death, these effects typically show up only at high exposure levels. The possibility that lower doses may also be harmful for brain development was not recognized until recently. Because lead mimics the actions of calcium, it can pass the blood-brain barrier and thereby affect the central nervous system where it affects the brain's prefrontal cerebral cortex, hippocampus and cerebellum (Finkelstein, Markowitz and Rosen 1998). 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 increase the risk for these types of behavioral problems (Weiss and Elsner 1996). Infants, toddlers, and the developing fetus are at the greatest risk of toxicity from low-level exposure because of a higher absorption rate into the blood and, also conditional on absorption, lead is more likely to affect the developing nervous system than the mature brain (Lidsky and Schneider 2003). Blood-lead levels follow an inverted v-shaped pattern between the ages 6–60 months, reaching its peak at the age of 24 months due to the intense hand-mouth activity at these ages (Needleman and Bellinger 2001; Canfield et al. 2003).

All these physiological impacts may hurt child development and thereby risk causing permanent damage to children's long-run human capital formation. The specific pathways are still unclear, however. Several epidemiological studies have documented strong correlations between lead and cognitive ability (Lamphear et al., 2005). The effect of lead on children's behavior is less understood and few studies have been done at levels of exposure <10 µg/dL (Liu et al. 2014). This is important since research has shown that non-cognitive skills are strongly linked to human capital accumulation (e.g. Heckman et al. 2006).

Appendix C: Effects on earnings

Table C1. The effect of earnings in 2013 (ages 29–40)

	ln (Earnings)	Earnings	Standarized Earnings
	Panel A: Girls		
Early lead exposure	.00036 (.00022)	.5202 (.3888)	.00019 (.00014)
Sample mean	7.54	2303.68	.824
	Panel B: Boys		
Early lead exposure	0.000001 (.0003)	-.0895 (.8430)	-.00003 (.0003)
Sample mean	7.989	3219.495	1.152
Neighborhood FEs	Yes	Yes	Yes
Year of birth FEs	Yes	Yes	Yes

Notes: The table presents the coefficient on early lead exposure from separate OLS regressions on ln earnings, Earnings (including zeros), and Standardized Earnings (Earnings/(mean Earnings)) in 2013. Coefficients reflect the effect of increasing lead by 1 unit. The sample consists of children born in 1972–1974, 1977–1979 and 1982–1984. Childhood lead exposure is measured as an average over ages 1–3. Cluster robust standard errors (at the commuting zone level (74 cells)) are shown in parenthesis. *** = significant at 1 %, * ** = significant at 5 %, * = significant at 10 % . See Table 1 for full set of controls.

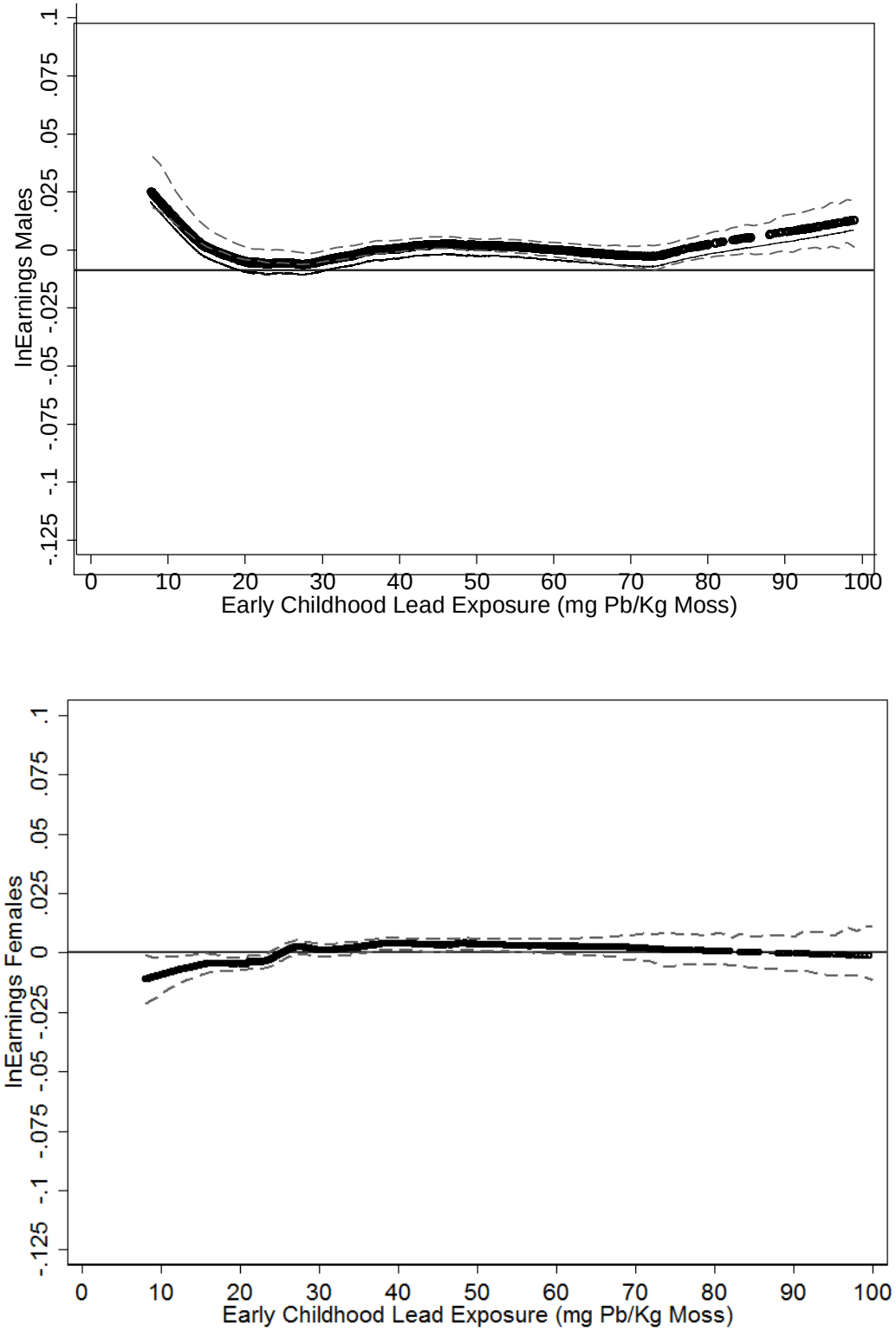

Figure C1. Semi parametric estimates on males and females ln(earnings in 2013) (ages 29–40)

Note: See Figure 2 for details of the estimation procedure.

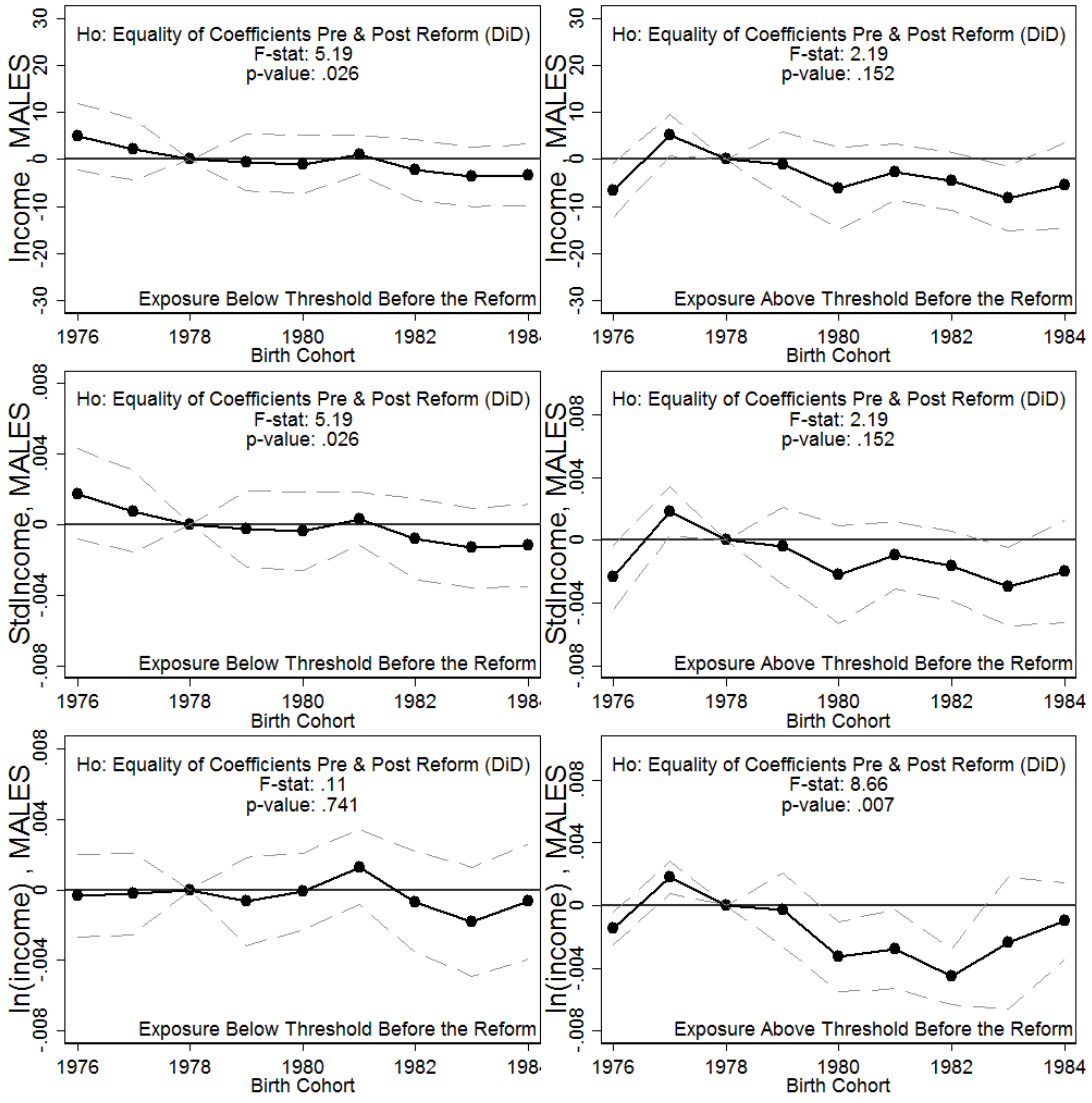

Figure C2. The estimated impact of the 1980/81 reform on earnings in 2013 (MALES)

Notes: These figures show the point-estimates, point-wise 95% confindence bands from the estimation of the cohort-specific $\gamma's$ from equation (5) on the male sample together with the F-stats and p-values for a test of the equality of the γ coefficients before and after the reform (i.e. difference-in-differences for 1976–78 vs.1979–1984 cohort coefficients). The top/middle/bottom left figure provides the estimates for Earnings/Standardized Earnings/ln(Earnings) among children born in neighborhoods with a lead level of Below 30mg/kg just before the reform. Theright figure provides the same estimates for children born in neighborhoods with a lead level of above 30mg/kg just before the reform.

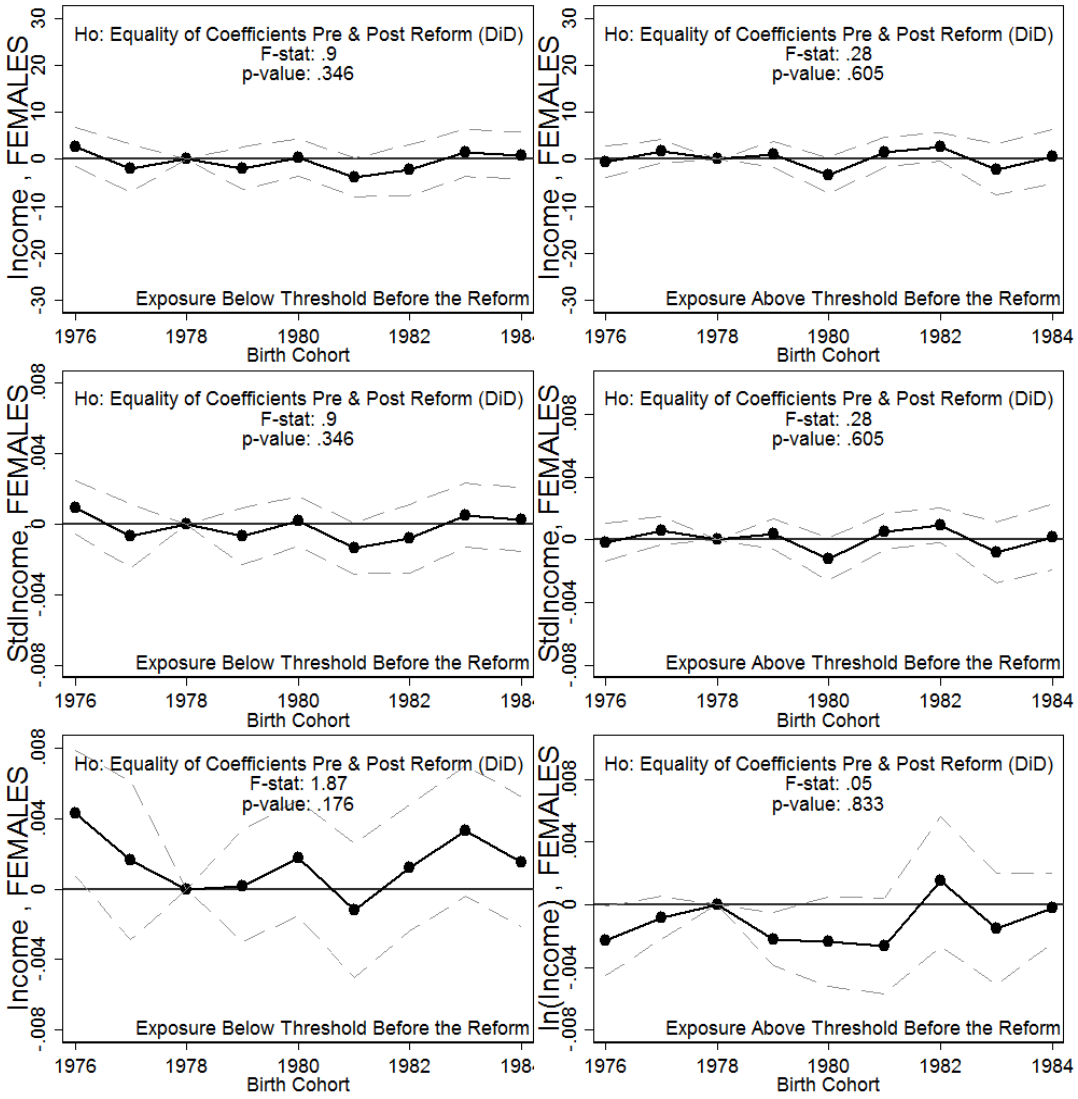

Figure C3. The estimated impact of the 1980/81 reform on earnings in 2013 (FEMALES)

Notes: These figures show the point-estimates, point-wise 95% confindence bands from the estimation of the cohort-specific $\gamma's$ from equation (5) on the female sample together with the *F*-stats and *p*-values for a test of the equality of the γ coefficients before and after the reform (i.e. difference-in-differences for 1976-78 vs.1979–1984 cohort coefficients). The top/middle/bottom left figure provides the estimates for Earnings/Standardized Earnings/ln(Earnings) among children born in neighborhoods with a lead level of below 30mg/kg just before the reform. The right figure provides the same estimates for children born in neighborhoods with a lead level of above 30mg/kg just before the reform.