

Understanding How Low Levels of Early Lead Exposure Affect Children's Life-Trajectories

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We study the impact of lead exposure from birth to adulthood and provide evidence on the mechanisms producing these effects. Following 800,000 children differentially exposed to the phaseout of leaded gasoline in Sweden, we find that even a low exposure affects long-run outcomes, that boys are more affected, and that changes in non-cognitive skills explain a sizeable share of the impact on crime and human capital. The effects are greater above exposure thresholds still relevant for the general population, and reductions in exposure equivalent to the magnitude of the recent redefinition of elevated blood-lead levels can increase earnings by 4%.

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I. Introduction^{*}

Exposure to lead is a major public health concern. It is well documented that high levels of exposure cause adverse and often irreversible health effects, which can even be fatal. Today, exposure leading to clinical symptoms is relatively rare, primarily due to policies first limiting and finally banning lead in gasoline. However, the general population is still exposed to lower doses of lead from a variety of sources including food, air, toys, contaminated soils, lead-based paint, and water.¹ Recently, in Flint, Michigan, exposure to lead due to deteriorating lead water pipes was made salient when an estimated 6,000 to 12,000 children experienced an increased lead exposure, and the share of children aged below 6 testing positive for elevated blood lead (BPb) levels ($>5 \mu\text{g}/\text{dL}$) more than doubled (Hannah-Attisha et al., 2016). The WHO reports that 40 percent of all children globally have elevated BPb.²

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¹ For example, the US Department of Housing and Urban Development estimates that 38 million homes constructed before 1978 still contain lead-based paint.

² 5 $\mu\text{g}/\text{dL}$ blood is the new reference value for elevated BPb levels established by the CDC in 2012. The reference value was 60 $\mu\text{g}/\text{dL}$ in 1960, 40 $\mu\text{g}/\text{dL}$ in 1970, 30 $\mu\text{g}/\text{dL}$ in 1975, 25 $\mu\text{g}/\text{dL}$ in 1985, and 10 $\mu\text{g}/\text{dL}$ in 1991.

Lead exposure is believed to be particularly harmful to infants and toddlers due to their rapidly developing central nervous systems, to an increased exposure due to hand-to-mouth activities and, conditional on exposure, to a higher absorption rate (Etzel, 2003).³ However, credibly estimating the effect of childhood lead exposure is typically hindered by a lack of large sample data that both links relevant outcomes to exposure and that at the same time is detailed enough to include information on all relevant confounders. The age of the housing stock and neighborhood poverty status constitute key predictors of elevated BPb levels in children in the US (Roberts and English, 2016). Hence, any unobserved individual and neighborhood attributes correlated with poverty status and child outcomes will likely conflate the correlation between lead exposure and child development. While a recent literature documents negative causal effects on child outcomes (e.g. Reyes, 2007; Rau, Urzúa and Reyes, 2015; Billings and Schnepel, 2015; Aizer et al., 2016; Gazze, 2016; Ferrie, Rolf and Troesken, 2012), little is known about the mechanisms through which childhood lead exposure maps into adult outcomes.⁴

³ Appendix A1 provides more details on the biological pathways through which lead exposure may affect child development.

⁴ Reyes (2007) uses state-level panel data and shows that the gasoline lead content reductions in the United States 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) finds a strong positive effect of childhood lead exposure on measures of delinquency for 8,000 children followed up to the age of 17 in the NLSY. Rau, Urzúa and Reyes (2015) examine the effects of proximity to a toxic waste site (containing large amounts of lead, mercury, and arsenic deposits from mining operations) on academic achievements in a Chilean community, finding that a longer residential distance to the waste site lowers the BPb level and improves math and reading test scores. Billings and Schnepel (2015) exploit the substantial measurement error in capillary BPb tests to study a sample of children who were 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, Currie, Simon, and Vivier (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 the reading test scores at the age of 8. Gazze (2016) exploits similar state mandates and finds that the share of children 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,

The aim of this paper is to provide consistent estimates of the long-run effects of early childhood lead exposure and explain the mechanisms of these effects. The context of our study is Sweden and our work draws on several strengths of the Swedish setting. First, the access to rich population micro data allows us to measure local air lead levels during the first three years of life for about 800,000 children and document their outcomes up to three decades later. The data contain measures of both cognitive and non-cognitive skills which allows us to decompose the effect on adult outcomes into components attributable to changes in these skills. Our novel measure of local air lead levels builds on data from moss (bryophyte) samples collected throughout Sweden by the Environmental Protection Agency. Using several independent data sources, we verify that changes in local moss lead (MPb) levels and the lead policy can be used to accurately predict early childhood BPb levels.

Second, we deal with correlated unobservables by focusing on children born between the early 1970s and mid 1980s when, due to the phaseout of leaded gasoline, lead levels per liter of gasoline were rapidly reduced by 80 percent. Since gasoline lead was the main source of lead exposure in the general population, there was a sharp decrease in children's BPb levels from the 1970s until the mid-1990s, when leaded gasoline was banned. Due to major differences in the initial lead levels, primarily due to pre-existing differences in traffic density, the reforms induced a substantial variation across localities in the size of the reduction in lead exposure which we exploit in our empirical analysis.

Third, Sweden banned lead in other important sources of exposure (e.g. paint and water pipes) as early as in the 1920s. Together with a low traffic density, this implies that the BPb levels, already before the phaseout of leaded gasoline, were much lower than in the US at the same time. We are thus able to document the effects of lead in a low-exposure setting.

cognitive ability, and homicide (Clay, Troesken and Hains 2014; Troesken 2008; Ferrie, Rolf and Troesken 2012; Fiegenbaum and Muller 2016).

This contextual feature is particularly useful as it helps provide evidence about the effects at exposure levels that are still common in many countries, including the US, and also allows us to shed some light on a question of key policy relevance: whether there are thresholds below which lead exposure has little or no effects on child development.

Across datasets, outcomes, and identification strategies, the results all point toward even low levels of childhood lead exposure hurting the short and long-term outcomes. Our main empirical strategy leverages the policy-induced changes in (within-neighborhood) exposure by focusing on the major reforms that took place in 1980/81 and estimating a flexible difference-in-differences event study model. This strategy lays bare the timing of the policy variation and allows us to visually assess how the reform effect evolves across birth cohorts. Our research design and results are most easily summarized in Figure 1 where we estimate the long differences (5 years) in MPb exposure and the outcomes for cohorts born around the time of the 1980/81 reforms using a local linear regression. It is clear that pre-reform neighborhood lead exposure is a strong predictor of subsequent changes in neighborhood lead exposure. After the reform, the exposure is equalized across neighborhoods, and the lead levels decrease drastically in neighborhoods with high pre-reform levels of exposure, while there are much smaller reductions across the cohorts born in areas with low pre-reform exposure. We also see that compared to older peers born in the same neighborhoods, the relative academic achievements of children improve among those born in high pre-reform exposure neighborhoods, but are unchanged in low pre-reform exposure neighborhoods. In the paper, we also show that a lower exposure to lead raises compulsory school GPA and reduces crime. Similar to other studies examining the importance of early experiences in the very long-run, we find that the effects in adulthood are stronger in boys (e.g. Nilsson, 2008; Chetty et al., 2016; Conti, Heckman and Pinto, 2016; Heckman and Karapakula, 2019), and for children from low-income households.

Figure 1 also illustrates another of our key findings: the effect becomes much weaker below an MPb level of around 30 mg/kg. This exposure threshold corresponds to an estimated early childhood BPb level of 5 µg/dL. In our more detailed analysis of the 1980/81 reforms where we provide cohort-by-cohort estimates of the effects of exposure, we find 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 living in neighborhoods *below the exposure threshold* were not affected by the reforms, whereas (iii) the outcomes of children in neighborhoods *above the exposure threshold* improved significantly. To put our estimates into perspective, a one-unit decrease in blood-lead above the exposure threshold significantly increases the probability of completing high school by 4 percent among males.

Interestingly, in our low-exposure setting, we find no significant or consistent pattern with respect to cognitive skills. However, non-cognitive skills follow the same nonlinear dose-response pattern as human capital and crime. When decomposing the effect on adult outcomes into components attributable to the policy-induced changes in these skills, we find that up to 40 percent of the changes in human capital can be accounted for by the estimated effects of lead exposure on the measured non-cognitive traits.

Our results add to a broad literature in social and medical sciences examining the effects of lead exposure in childhood on human capital accumulation. Much of this literature focuses on the effects on grades, and cognitive or behavioral test scores for children using cross-sectional data. There is less evidence of the effects on adult outcomes and few previous studies report estimates that can be interpreted as causal. Our most important contribution is that we examine the effects on adult outcomes at exposure levels that are still relevant, while the earlier work examines child outcomes or effects at much higher levels of exposure. In fact, many of the earlier studies examine children who by today's standards experienced extreme

levels of exposure.⁵ In addition, while previous studies tend to examine the outcomes separately, we provide comprehensive results for a broad set of adult outcomes using an identical approach and population-wide data. Moreover, previous studies focus on estimating the effects of childhood lead exposure and do not attempt to explain their sources. While animal studies suggest that lead interferes with development, we know little about the pathways to later human capital outcomes, or whether different types of skills and behavior respond more strongly to a given level of exposure. We provide direct evidence of two key mechanisms highlighted in previous work: cognitive and non-cognitive skills. The specific channels are of obvious importance for efficient remediating investments, for example, because cognitive and non-cognitive skills differ in their malleability across a child's lifecycle

⁵ In the 1970s, several studies documented strong associations between lead exposure and cognitive and behavioral outcomes in children without clinical symptoms. However, by today's standards, even these children experienced extreme levels of exposure (e.g., Needleman et al. 1979). For instance, Landrigan et al. (1975) classify children with a BPb at 27 in the "low-exposure" group. Subsequent 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 of effects of low lead exposure is still debated, however (Lanphear et al. 2000). Not only is it unclear at what level damage actually occurs, but the results have been put into question for the use of small and unrepresentative samples (Needleman et al. 1996 study 301 first graders in Pittsburgh, Pennsylvania; Dietrich et al. 2001 examine 195 inner city adolescents from Cincinnati, Ohio; Wright et al. 2008 study 376 children also from Cincinnati), measurement errors, a focus on short-run effects on test-scores, and the inability to control for confounders (e.g., Ernhart 1995; Kaufman 2001a, 2001b; Heben 2001; Needleman and Bellinger 2001). While evidence from animal studies holds information on the toxicity of lead, it is unclear how such findings map back to cost-benefit analyses of policy interventions with respect to the effects of human exposure on adult outcomes. Among the more recent and design based studies, Billings and Schnepel (2015) examine the intervention that occurs at 10 and 20 µg/dL. The cohorts studied in Reyes (2007) were estimated to have a BPb level of 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 needs following reductions in the share of children with BPb>10 µg/dL and 20 µg/dL. Rau et al. (2016) study the effects on academic achievement and earnings where the subjects' BPb levels were, on average, 10 µg/dL, while Aizer et al. (2016) study the effects of low exposure (<10 µg/dL) but their analysis is limited to third grade reading and math test scores. To put these estimates into perspective, Reyes (2007) reports that average childhood BPb levels in the adult US population will have decreased from 10 µg/dL in 2002 to less than 3 µg/dL in 2018.

(Francesconi and Heckman 2016).⁶ Our results suggest that policies targeting non-cognitive traits among children may be more successful in mitigating the adverse effects of low early lead exposure.⁷

The rest of this paper is structured as follows. Section 2 discusses our data and shows how air lead exposure is linked to children's BPb levels. Section 3 describes our research design. Section 4 presents the empirical results. In Section 5 we interpret our findings and decompose the effects of early lead exposure by source and Section 6 concludes.

II. Setting and measurement

Lead started to be added to gasoline in Sweden in 1946 (Danielson, 1970). Adding lead was an easy way of reducing engine knocking, boosting octane ratings, and helping with wear and tear on valve seats in the engine in early car models. In response to the increasing lead emissions from motor vehicles, illustrated in Figure 2, Sweden initiated a staggered phase-out of leaded gasoline in 1970 in order to protect the environment and future public health. At the time of the reforms, the concurrent level of exposure was well below the limit of concern, and it was therefore not considered to be an immediate public health concern. Policy makers were concerned that with the projected increase in vehicles and the increasing trend in gasoline lead content, lead exposure would become a public health issue if no action were undertaken.

⁶ Our finding that non-cognitive skills dominate cognitive skills in mediating the effects of early lead exposure on human capital is also important since the effects of lead on grades and achievement test scores have previously often been interpreted as measuring the impact on cognition. Yet, it has been shown that personality traits constitute a substantial explanation for grades and achievement test scores (Borghans 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 is the key explanatory factor behind the crime reducing effects of the Perry Preschool program.

⁷ More broadly, our study is also related to the large body of research on the effects of air pollution on health and human capital (e.g. Chay and Greenstone 2003; Currie and Neidell 2005; Isen, et al. 2017; Lüchinger 2009; Jans et al. 2014).

Lead is well known to be highly toxic to humans, irrespective of how it enters the body and it affects brain development and organ function. Appendix A summarizes the evidence in the medical literature, in particular the specific mechanisms that affect development.⁸ The major lesson learnt from this review is that: (i) Early exposure poses the greatest risk because of a higher absorption rate among children and, moreover, conditional on absorption, lead is more likely to affect the developing nervous system than the mature brain; (ii) BPb 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 this age; (iii) While neither the lead exposure nor the take-up differs by gender among infants and toddlers, the evidence is mixed regarding gender differences in early vulnerability. Burns et al. (1999) find no evidence of gender differences in the impact on cognition, but fMRI scans show that early exposure is correlated with a lower brain volume, particularly among boys and in the prefrontal cortex (PFC). PFC activity is correlated with the ability to plan and imagine consequences of one's actions, impulsive behavior, self-control, and the ability to delay gratification (Cecil et al., 2008).⁹

Although 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 percent. Gasoline lead was the main source of lead exposure in the general population and as we can see in Figure 2, there was a sharp decrease in children's BPb from the 1970s until the mid-1990s when leaded gasoline was banned altogether.

⁸ Appendix A also outlines a conceptual framework, based on the seminal work by Cuhna and Heckman (2007, 2009), which serves to explain how even early exposure to low levels of lead may affect the outcomes in adulthood.

⁹ However, as regards the more common measures of development, it is unclear whether the gender differences detected by in the fMRI scans are due to biological differences in the sensitivity to lead or simply reflect correlated unobservables. Moreover, both Burns et al. and Cecil et al. examine children with high BPb levels ($>10 \mu\text{g/dL}$).

The rest of this section explains how we use the phaseout to measure lead exposure and document the link between exposure and children's BPb levels. We also discuss our administrative data.

A. Measuring exposure

Unfortunately, there is no large sample data-set that monitors trends in BPb levels among young children or the general population in Sweden during the phaseout of leaded gasoline. Instead, to measure early lead exposure, we use novel data from the Swedish Environmental Protection Agency (EPA), which has monitored heavy metal air pollution every fifth year since 1975 using a nationwide grid of moss (bryophytes) samples. The use of mosses as biomonitoring tools for heavy metal air pollution was developed in Sweden in the 1960s in pioneering work by Rühling and Tyler (1968, 1969) and is now well established. Since 1995, 28 countries participate in a bi-decennial survey designed to study regional differences and trends in heavy metal deposition throughout Europe.

Moss is particularly suitable for monitoring air pollution levels for several reasons. The lack of roots implies that take-up solely depends on the surface absorption of pollution through precipitation and the dry deposition of airborne particles. The absorption and retention of metals is high, and mosses can be found in abundance in nearly all environments. It is further possible to distinguish temporal patterns in pollution levels.¹⁰ Biomonitoring has several advantages over regular pollution monitors, the main ones being their simplicity, accuracy, and low cost which allow many sites to be included in the surveys.

¹⁰ The annual growth segments are easily distinguishable and the transportation of metal across segments is minimal.

The EPA collects samples from around 1,000 sites all over Sweden using a systematic procedure.¹¹ The growth segments of the last three preceding years are pooled and analyzed by the EPA and provide a measure of average local air lead levels. We use the samples collected in 1975, 1980 and 1985, which reflect the lead deposition during the years 1972-1974, 1977-1979, and 1982-1984. The selection of these years is made for two reasons. First, the maximum concentration of lead in gasoline permitted decreased sharply between these years. As shown in Figure 2, the maximum lead level permitted in gasoline was reduced from 0.7 to 0.4 g/L in 1973. This change applied to all types of gasoline. In January 1980 (1981), there was an additional major cut for regular (premium) gasoline from 0.4 to 0.15 g/L. In 1986 (1995), lead was banned for regular (premium) gasoline. Second, the sample is restricted to cohorts that have reached adulthood and for whom exposure in early childhood has been 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 of lead exposure for each period and parish (henceforth “neighborhood”), we calculate the inverse distance weighted average air lead level for each neighborhood using the population weighted neighborhood centroids and the 10 closest sampling sites.¹² The average neighborhood has about 3,000 inhabitants, which is slightly larger than the US Census Block Groups.

¹¹ The sampling sites have been chosen with care; they should be located at least 300 meters away from major 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 m².

¹² To test the sensitivity of the results, we also used the five nearest sample points. The differences between these definitions are small and they are highly correlated ($\rho = .99$). To cross-validate our approach, we also estimated the level of lead at each sampling point, as opposed to neighborhood, after excluding the sampling point of interest. We did this for all sample sites, and then correlated the actual and predicted air lead levels. The high correlation ($\rho = .82$) indicates that the interpolation method provides reasonably accurate predictions of local air lead levels.

There is a great deal of variation in lead levels between neighborhoods. Figures 3a and 3b present maps for neighborhood lead concentrations in 1975 and 1985, using our definition of exposure. From these figures, it is clear that the MPb concentrations fell sharply between 1975 and 1985, and that the MPb was highest in the densely populated coastal regions. Figure 4 shows that the entire distribution of lead levels shifted dramatically between 1975 and 1985 and became more compressed. Figure 5 shows the distribution of within-neighborhood changes in lead exposure between 1975 and 1985. Most neighborhoods experienced decreases in MPb levels of between 15 and 45 mg/kg, with an average reduction of 30 mg/kg. Most of the reduction in lead exposure is due to within-neighborhood rather than between-neighborhood differences.¹³ These sharp within-neighborhood differences in exposure across cohorts constitute a key feature of our research design.

B. The relationship between MPb and children's BPb

In the main analysis we exploit all data on local lead levels, as defined in the previous section, and link them to outcomes of all the children in our sample. Before turning to the main analysis, we first shed light on the important link between moss lead and children's blood lead. Finding that MPb levels are uncorrelated with BPb levels would cast doubts on the relevance of our measure of exposure.¹⁴ We report the key findings on the relationship below, while Appendix B1 provides a more detailed discussion of the BPb data, the estimation procedure, and gives additional robustness checks.

¹³ The between and within SDs are 10.78 and 14.35, respectively.

¹⁴ That said, the two measures partly capture different aspects of exposure. While BPb levels, the most commonly used measure of lead exposure in the literature, reflects current exposure (the half-life is 2-3 months), the moss samples measure long-term exposure (averaged over the three preceding years). This is arguably one advantage of the moss-measurement, but partly for this reason, we also do not expect MPb levels to be perfectly correlated with BPb levels.

As noted above, there is no comprehensive data with measures of blood-lead available in Sweden. However, since 1978, venous blood samples have been collected from about 120 primary school children (between 8 and 10) per year in two municipalities in southern Sweden.¹⁵ One of these municipalities, Landskrona, also collected around 50 moss samples using the same procedure as in the national moss sampling survey in 1984 and 1995.

The two datasets from Landskrona provide an opportunity to assess the strength of the relationship between our measure of local air lead exposure and children's BPb. Table 1 Panel A reiterates and extends the findings in Nilsson et al. (2009), which links the average lead level of the nearest moss samples to the children in Landskrona using their residential location. The table reports the estimated elasticity from regressions of BPb levels on MPb levels using four different specifications. We can see that controlling for individual characteristics, time and neighborhood fixed effects, the estimates in Panel A suggest an elasticity in the pre-ban period of 0.47. This implies that a 10 percent reduction in MPb corresponds to a 4.7 percent decrease in primary school children's BPb levels. Moreover, there are no differences in the relationship between MPb and BPb levels in boys and girls, which is central for the interpretation of our main results. Our estimates suggest that the drop in local air lead levels between 1982 and 1994 can account for as much as 50 percent of the change in children's BPb levels.¹⁶

¹⁵ The methods and results of the trends in childhood lead exposure are described in detail in Strömberg et al. (2003).

¹⁶ Here it is instructive to discuss the role of other sources of lead exposure in Sweden. First, it is important to know that lead-based paint was banned in Sweden in the early 1920s (unlike in, for example, the US at the end of the 1970s, and the UK in the early 1980s). Second, lead water service pipes are a non-issue in Sweden. In addition, even after the major gasoline lead reforms were implemented in the late 1980s, more than 80% of the airborne lead stemmed from vehicle exhaust emissions. That said, other changes may have contributed to the decline in lead exposure. In our context, it has been suggested that exposure from food has contributed to the decline in lead exposure. This is partly due to the fact that when the gasoline lead content decreased, the take up of lead in plants and animals also decreased. Since most of what children eat in Sweden, as in most other developed countries, is not produced locally, this implies that local airborne lead levels will not be

That said the blood lead data has its limitations in terms of the small sample size and geographical dispersion. It is also important to bear in mind that the relationship between environmental lead exposure and young children's BPb levels is significantly stronger due to greater exposure and higher absorption. For example, Reyes (2007) finds that the elasticity between lead in gasoline and BPb in children aged 0-6 is around 30 percent higher than among children aged 6-12. In addition, there are several potentially important layers of measurement problems (mapping moss observations to neighborhoods, neighborhood lead to actual exposure, adjusting for the varying relationship by children's age, etc.) that may distort the MPb-BPb predictions.

A relevant question is therefore whether moss lead levels predict blood lead levels among younger children in other parts of Sweden? To address this issue, Appendix B2 provides results from an out-of-sample validation exercise using observed BPb levels in preschool children in central Stockholm in 1979 and 1992, and in a Stockholm suburb in 1991. In summary, the MPb levels give an accurate prediction (prediction errors of about 0.4 µg/dL) of the average BPb levels among preschool children, even in Sweden's most densely populated city center. This important result validates the use of the MPb data for the estimation of the impact of early lead exposure on long-term outcomes.

Our main research design exploits the sudden changes in lead exposure across cohorts born around the 1980/81 reforms. Since the size of the change in exposure is connected to the pre-reform exposure levels, which to a large extent were caused by pre-existing population/traffic density, it is important to examine whether also children's BPb levels were

perfectly correlated with local BPb levels, despite the fact that the policy changes are the cause of the decline of lead in food. A second more gradual change that may also have contributed to the BPb declines other than airborne lead is that the lead soldering of cans has decreased in favor of welded food cans. There was no mandate for this and we have found no evidence indicating that the change in the lead-soldered food cans has contributed in nearly as significant or abrupt ways to the BPb levels as the phase-out of leaded gasoline.

differently affected depending on whether they lived in more or less densely populated areas. As a reality check of the validity of our empirical design, we therefore estimate the effects of the reform in 1980/81 on children's BPb using data collected from Trelleborg municipality¹⁷ by Strömberg et al. (2003). While Trelleborg is one of the most densely populated municipalities in the Sweden, it also contains more rural areas, allowing us to compare trends in BPb levels around the reforms among children living in more or less populated areas.

Figure 6 summarizes our findings. The hollow circle line in Figure 6 shows the total maximum gasoline lead sales (in metric tons) around the time of the reform.¹⁸ The two other lines show local average BPb levels reported separately for children residing in urban areas and in rural areas before, during, and after the implementation of the reform. The three time series in Figure 6 follows a strikingly similar pattern.¹⁹ Before the reform, the BPb levels in urban Trelleborg averaged 6 µg/dL. Between 1979 and 1983, at the same time as the gasoline lead content plummeted, so did the BPb levels among urban children with little or no changes before the reform, or in the years following the reform. Among rural children, the BPb levels also decreased following the reform, but not as sharply. After the full implementation, the BPb levels of urban and rural children leveled off and converged at around 4 µg/dL.

Table 1 Panel B provides difference-in-differences estimates, comparing the impact of the reform on BPb in urban vs. rural areas of Trelleborg before and after 1980/81, controlling for individual, area and time effects. In column (1), we see that children in densely populated

¹⁷ Trelleborg is one the southern-most municipalities in Sweden (i.e. one of the most densely populated regions in the country) with highest lead exposure and has no major lead-emitting industries. Trelleborg constitutes the southern major junction of the European Route E6 which passes through the urban parts of the municipality.

¹⁸ Elinder et al. (1986) find that the air lead levels in Stockholm decreased by 50% between 1979 and 1982, which is exactly the effect that was predicted in the preparatory work of the reform (SOU 1979:34, p. 35).

¹⁹ We follow Strömberg et al. (1995) and apply a two-year lag when relating BPb levels to the gasoline lead content, allowing for lasting environmental contamination and the slow excretion of lead stored in bones.

areas experienced larger drops in BPb levels than children in sparsely populated areas following the reforms. Column (2) allows the reform effect to differ during the phase-in (1980-1981) and full reform (>1982) periods, and column (3) shows results from the same specification after adding year of blood sampling fixed effects. In line with Figure 6, the impact of the reform is significantly stronger among urban children and the effect increased as the reform was rolled out (-27%). Column (4) shows that the reform has no significantly different impact on the BPb levels among boys and girls.

In summary, the key results in this section are that changes in MPb levels strongly predict children's BPb levels, and that the phaseout resulted in differential changes not only in MPb levels but also in children's BPb levels. Moreover, there are no gender differences in the relationship between MPb and children's BPb. Next we describe the administrative data used in our main analysis.

C. Administrative data

Our outcome analysis sample includes 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 1973-1974, 1977-1979 and 1982-1984.²⁰ The moss samples measure the local lead deposition at ages 1-3 (e.g. mosses collected in 1975 reflect the lead levels in 1972-1974), which correspond to a particularly sensitive period in human development and a period with a particularly high uptake of lead. We focus on children who have completed compulsory school (9 years) and who were born in Sweden, so that their neighborhood of birth (and hence, their childhood lead

²⁰ The data originates 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. While all databases draw from the same population-based registers at Statistics Sweden (SCB), containing the information necessary to identify our main sample as well as standard background characteristics, the databases differ in terms of the specific outcome measures they comprise.

exposure) is known. Sweden is one of the OECD countries with the highest age for university graduates (the average age is 29) and more than 40 percent of each birth cohort enroll in college (OECD 2014). Hence, we focus on outcomes which we are able to measure at the same age for all cohorts. We measure educational outcomes using the compulsory school grade point average (GPA) (aged 16, percentile ranked within graduation cohorts) and the probability of graduating from high school (aged 19).

Criminal behavior is measured using data on criminal convictions available between 1985 and 2008, provided by the National Institute for Crime Prevention (BRÅ). The data includes details on the type of crime as well as the exact date of the offense for all individuals up to the age of 24.²¹ A conviction may include several crimes but all crimes are recorded. Speeding tickets, and other minor crimes not severe enough to warrant a trial, are not covered. We are interested in the probability of being convicted at least once for any type of crime and in the probability of being convicted for the more specific and most common crime categories: violent and property crime. Violent crime represents the full spectrum of assaults from minor assault to murder. Property crime covers the full spectrum of thefts from shoplifting to robbery.

We shed light on the mediating factors using unique data on cognitive and non-cognitive skills measured at military enlistment. All Swedish men were obliged by law to participate in the military draft. Around 90 percent of all males born in Sweden went through the draft at the age of 18 or 19.²² The cognitive test scores were Stanine (Standard Nine) and

²¹ The age of criminal responsibility in Sweden is 15, meaning that we do not observe crime before the age of 15.

²² In principle, only those with a physical or intellectual disability were exempt from the enlistment tests. Due to reforms in the enlistment procedures affecting the cohorts born during the 1980s, the share of males tested decreased to 85% in 1984. Excluding the post-1980 cohorts does not change any of our conclusions from the main analysis. However, as we discuss below, including the later cohort allows us to test effects on cognitive and non-cognitive skills in the analysis of the 1980/81 reforms.

similar to the AFQT in the US. The test assessed the subject's cognitive ability based on subtests of logical, verbal, spatial, and technical abilities. These subtests were combined by the military to produce a general cognitive ability measure on a discrete 1 to 9 scale. We standardize the score within each cohort of draftees to account for minor changes in the tests over time.²³

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 personality traits²⁴ to which they are related: (i) *Psychological energy* (perseverance (*C*), ability to fulfill plans (*C*), to remain focused (*C*)), (ii) *Emotional stability* (ability to control and channel nervousness (*N*), tolerance of stress (*N*), and disposition to anxiety (*N*)), (iii) *Social maturity* (extraversion (*E*), having friends (*E*), taking responsibility (*C*), independence (*O*)), and (iv) *Intensity* (the capacity to activate oneself without external pressure (*C*) and the intensity and frequency of leisure activities (*O*)). Using principal component analysis, we combine the sub-scores into a general measure of non-cognitive ability and use it as our main measure of non-cognitive ability.²⁵

Table 2 provides summary statistics for the main analysis sample. For example, we can see that 79 percent of the children in our sample completed high school and that a large proportion of the children, 16.4 percent, have been convicted at least once by the age of 24.

²³ The test has been subject to evaluation by psychologists and appears to be a good measure of general IQ (Carlstedt 2000).

²⁴ (*O*)peness to Experience, (*C*)onscientiousness, (*E*xtraversion, (*A*greeableness, (*N*euroticism.

²⁵ The correlation between the non-cognitive index and the non-cognitive measure provided by the military is high (0.95), but the procedure the military uses to create their index is unknown, which is why we prefer to use the PCA index as our measure of non-cognitive skills. The PCA gives approximately the same weight to the different non-cognitive sub-skills. The correlation between the non-cognitive skills index and cognitive skills is 0.3. The correlation between cognitive skills (non-cognitive) and 9th grade GPA is 0.64 (0.4). Nilsson (2017) provides an account of how the military non-cognitive sub-scores relate to the Big-Five traits of personality (c.f. Bihagen et al. 2012). Lindqvist and Vestman (2011) relate the cognitive and non-cognitive skills to labor market outcomes.

The number is well in line with other studies using data from the same sources (e.g., Grönqvist et al. 2016). The average child grew up in a neighborhood with MPb exposure equal to 34mg/kg, and around 40 percent of the children have at least one parent who has completed college. Next we describe how we estimate the effect of childhood lead exposure on the human capital and crime outcomes.

III. Research design

Our goal is to identify the effect of lead exposure on children's life trajectories. To better understand our novel measure of lead exposure, we start with a thorough explorative analysis that covers all cohorts using linear and semi-parametric fixed effect models (which we sometimes refer to simply as our panel data analysis). Guided by the results from this analysis, we then home in on the cohorts born in the period between 1976 and 1984 and provide a detailed account of the impact of the major reforms in 1980/81 which reduced the maximum lead content from 0.4 to 0.15 g/l. We describe the different approaches below.

Our initial analysis of the data exploits the panel structure and estimates variations of the following model estimated on individual data aggregated to the neighborhood-cohort level

$$(1) \quad Y_{nc} = \alpha + \beta Lead_{nc} + \theta X_{nc} + \delta Z_{mc} + \lambda_n + \lambda_c + \varepsilon_{nc}$$

where n , c and m denote neighborhood, cohort and municipality, respectively. Y_{nc} is the outcome of interest and $Lead_{nc}$ is the local air lead level measured in mg/kg moss. We include neighborhood fixed effects (λ_n) that account for persistent differences across neighborhoods over time, and birth cohort fixed effects (λ_c) that control for nationwide trends in the outcomes. To increase the statistical precision and test the sensitivity of our specification, the regressions also control for parental and individual background

characteristics (X_{nc}) aggregated to the neighborhood level, and municipality level covariates (Z_{mc}) (employment rate and population size). ε_{nc} is the error term representing unobserved determinants of children's long-run outcomes. All regressions are weighted by the number of children in each cell, and the standard errors are clustered at the commuting zone (CZ) level to account for both spatially and serially correlated errors within the 74 CZs.²⁶ $\hat{\beta}^{OLS}$ provides the effect on adult outcomes of an additional unit of lead exposure (mg/kg) in early childhood.

While model (1) restricts the effect of lead exposure to be linearly related to the outcomes, the benchmark dose-response model in toxicology is a threshold model which posits that the effect of toxins follows a "hockey stick" shape across the distribution with diminishing marginal effects as exposure decreases. Put differently, below certain thresholds, further reductions in exposure may no longer affect the outcome of interest. Although some indicative health thresholds for lead have been established for children at varying levels depending on the outcome,²⁷ no thresholds have been proven for more subtle effects on neuro-cognitive development or behavior.²⁸ The literature that attempts to address the

²⁶ There are 2,559 neighborhoods in our data. Statistics Sweden divided these into 74 commuting zones. As shown below, we also explored alternative ways of accounting for correlated errors, including clustering at the nearest lead sampling point, the neighborhood level, and the neighborhood-by-cohort level. Clustering at the CZ level is our preferred option, not only because it provides the most conservative standard errors, but also because of its transparency and relevance (CZs are local labor markets defined by Statistics Sweden), and that clustering at the CZ also accounts for an arbitrary correlation in the errors across neighborhoods within the same CZ.

²⁷ For instance, encephalopathy, seizures and coma (>70-80 µg/dL), renal (kidney) failure (50 µg/dL), hemoglobin disruption (25 µg/dL) (Agency for Toxic Substances and Disease Registry 2007).

²⁸ The extent to which this is due to the limitations of the cross-sectional research designs, or that previous studies generally test for nonlinearities at high levels of exposure, or both, is unclear. Theory does not provide 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 changes in exposure at low levels of exposure have stronger effects on test scores than changes at high levels of exposure (see e.g., 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 the composition of children that are exposed to low

identification issues and directly test for nonlinearities has not detected any thresholds but has examined effects at much higher exposure.²⁹ The setting of our study offers a rare possibility to shed some light on the role of nonlinearities at levels that are still common in developed countries. This is done by estimating semi-parametric fixed effect models (c.f. Baltagi and Li, 2002; Libois and Verardi, 2013) that allow the exposure-response function $f(\text{Lead}_{nc})$ to be estimated in a fully flexible way. The semi-parametric model is computationally intensive, and to ease the computational burden, we collapse the individual level data at the neighborhood-by-measurement year level and weight by the number of children in each cell.³⁰

Both the linear and semi-parametric fixed effects models rest on the same key underlying assumptions: unobserved determinants of long-run outcomes should not co-vary with changes in lead exposure conditional on the covariates. This assumption is violated if unobserved factors trended differently across localities depending on whether the neighborhoods experienced large or small drops in lead exposure following the reforms. Our analysis of children's BPb data in Section 2 highlights a potentially important factor: the

levels of lead is likely to differ from the composition of children exposed to higher levels. 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 have been exposed, namely children living in low-standard housing with deteriorating water pipes, lead paint walls, or living close to lead-emitting industries. Therefore, it is possible that the bias from unobservables is stronger in a low-lead setting than in a high-lead setting, generating a steeper dose-response curve in settings where the sources of exposure are more likely to reflect unobserved family characteristics.

²⁹ Reyes (2011) directly examines nonlinearities in the effects of early lead on violent crime, but finds no threshold effects at average BPb levels between 10 µg/dL and 20 µg/dL.

³⁰ The non-parametric exposure-response function, $\hat{f}(\cdot)$, is estimated using local linear regression and we present the non-parametric slope together with bootstrapped 95% confidence intervals (1,000 repetitions). Specifically, we estimate a narrow bandwidth (.3) robust local linear regression model (LOWESS) using Cleveland's tri-cube weighting function. To further reduce the influence of outliers, we follow standard practice and exclude the bottom and top 1% of the observations in the exposure distribution. We also tested varying the bandwidth and removing the (Cleveland) weighting functions. Overall, the interpretation of the results for the key human capital outcomes is not particularly affected by these sensitivity checks.

variation in lead exposure induced by the reforms is clearly linked to population density. If unobserved factors evolved differently between urban and rural areas, the baseline fixed effects estimates may be biased. On the one hand, it is well documented (c.f. Thomas et al., 1999) that the general dramatic drop in lead exposure that occurred during the observation period was due to the policies mandating reductions in the maximum gasoline lead content. This suggests that it may be plausible to consider the major changes in lead exposure as orthogonal to trends in unobserved local conditions. On the other hand, because the panel data analysis uses *all* changes in lead exposure during the observation period to identify the parameter of interest, it is conceivable that the estimates at least partly capture secular changes in other local conditions correlated with both lead and children's outcomes.³¹

This concern motivates our main empirical strategy, which is a flexible continuous difference-in-differences research design that proxies changes in exposure in neighborhood n using the lead exposure level just *prior* to the 1980/81 reforms.³² Henceforth, we will sometimes simply refer to this as the reform analysis. The model, estimated by ordinary least squares (OLS), is specified as follows

³¹ *A priori*, the direction of any bias is unclear. If well-educated parents disproportionately moved to more rural areas to avoid pollution at the same time as lead levels decreased, then the panel fixed effect estimator could overstate the effects of the reductions in lead. Alternatively, if parents with a low education were closer to the margin of moving to more rural areas, an increase in environmental awareness could underestimate the estimated effect of the reductions in lead exposure.

³² We focus on the 1980/81 reforms for three reasons. First, several years had passed since the previous reform was implemented (1973), and there were several years until the next reform was implemented (1986). This gives us a reasonably long pre-reform period, which we use to examine pre-reform trends. Second, it enables us to examine the same outcomes as in the panel analysis. Third, the 1980/81 reforms give us a well-defined pre-reform measure of exposure (1977-1979). The 1973 reform is not as useful since the closest MPb exposure measure captures a mix of pre and post-reform levels of exposure (1972-1974). The 1986 reform is less useful as it only targeted regular grade gasoline and the changes in lead exposure are much more gradual than following the 1980/81 reforms which targeted all grades.

$$(2) \quad Y_{inc} = \sum_{c=1976}^{1984} (\lambda_c \times Lead_n^{Pre-Reform}) \cdot \gamma_c \\ + \theta X_{inc} + \delta Z_{mc} + \lambda_c + \lambda_n + \varepsilon_{inc} .$$

where $Lead_n^{Pre-reform}$ is the (pre-determined) MPb level just before the reforms in neighborhood n . To map out the changes in outcomes for cohorts born around the time of the reform, we interact pre-reform exposure with cohort of birth (λ_c). All common variables are as in equation (1), except that we are now also able to control for total annual municipality gasoline sales to account for other traffic-related pollutants and traffic density.³³ ε_{inc} is the error term, and the standard errors are clustered at the neighborhood-by-cohort and CZ level in all specifications.³⁴

Similar to model (1) identification in this reduced form model requires common trends. One key advantage of the reduced form model (2) is that we do not have to restrict the sample to only the cohorts for whom we have moss-lead exposure data, and estimate this model using data on all cohort born between 1976 and 1984. Even more importantly, model (2) lays bare the timing of the policy variation and allows a visual assessment of the plausibility of the common trends assumption.

Building on the reduced form results from model (2) in combination with the long-difference first-stage results shown in Figure 1, we can take an additional step and provide two-stage least squares (2SLS) estimates for the “full-reform effect” of early lead exposure. To see how, first note that unlike the reduced form model the longer time interval between the moss sampling occasions prohibits us from estimating the cohort-by-cohort

³³ Gasoline lead sales are useful since we do not have any direct measures of the other traffic related pollutants (Cu, Zn, Cd) for the 1980-1982 cohorts. For the 1980-82 cohorts, we impute the Zn, Cu, and Cd levels using the average of the 1980 and 1985 measurements in the neighborhood of birth.

³⁴ Clustering at the cohort-by-neighborhood and commuting zone level aligns the analysis with the baseline (cohort-by-neighborhood) panel data specification.

first-stage effects of the reform. To implement the 2SLS analysis, we therefore divide the children into three groups based on cohort of birth: the pre-reform (1976-1978), phase-in (1979-1981), and full-reform (1982-1984) cohorts. We then interact the phase-in and full-reform dummies with the pre-reform moss-lead level in the neighborhood of birth, and estimate a system of equations of the following form

$$\begin{aligned} \text{Lead}_{nc} &= \pi_1 (\text{FullReform}_c \times \text{Lead}_n^{\text{Pre-Reform}}) \\ (3) \quad &+ \omega_1 X_{inc} + \omega_2 Z_{mc} + \rho_c + \rho_n + \tau_{inc} \end{aligned}$$

$$Y_{inc} = \delta \cdot \widehat{\text{Lead}}_{nc} + \theta X_{inc} + \delta Z_{mc} + \lambda_c + \lambda_n + \varepsilon_{inc}$$

where the excluded instrument is the interaction term between the full-reform cohort dummy, FullReform_c , and the pre-reform moss-lead level in the neighborhood of birth, $\text{Lead}_n^{\text{Pre-Reform}}$.³⁵ In addition to addressing the endogeneity concerns, the 2SLS model also accounts for measurement error in the exposure variable. The model relies on the standard assumptions regarding the 2SLS estimator: that the instrument is uncorrelated with the error and does not directly affect long-term outcomes other than through its effect on actual lead levels. At the very basic level, this assumption is simply the same as in models (1) and (2): unobserved correlates of exposure should not give rise to differential secular trends in the

³⁵Note that $\text{Phasein}_c \times \text{Lead}_n^{\text{Pre-Reform}}$ interaction is not identified since we do not have any measures of early lead exposure for the phase-in cohorts, only for the pre- and full-reform cohorts. In practice, we set the observed moss-lead level in these phase-in cohorts to the pre-reform moss-lead level in the neighborhood of birth. The Phase-in \times Pre-reform Lead, and the Pre-reform Lead variables are included in X_{nc} in the above equations. The π_1 and δ coefficients are thus estimated in comparison with the pre-reform cohorts, not the phase-in cohorts.

outcomes of interest. Since all models are based on this assumption, it is useful to start by exploring our data to challenge this assumption before moving on to the results.

A. Assessing the identifying assumptions

Many unobserved factors may give rise to differential trends in the setting we consider. Two of the most obvious candidates are the local business cycle and changes in population density, since both may be linked to local lead levels via emissions and may also co-vary with child outcomes. To provide a first assessment of the risk of bias from these factors, we collected municipality level data on population density and employment from the bi-decennial Censuses. Figure C.1 shows that changes in local lead exposure between 1975 and 1985 appear to be unrelated to changes in population density and local employment. As we will see below, controlling for these variables in the regressions does not change the magnitude of the estimates in a meaningful way.

Other pollutants predictive of children's long-term outcomes and also correlated with local lead levels represent another potential source of bias. The fact that we focus on changes in air lead levels induced by government regulations that specifically targeted the gasoline lead content should mitigate much of this concern. Fortunately, since the moss data also holds information on other common heavy metals, we are able to make an initial assessment of the likely severity of this problem. Of all the observable pollutants in the data, only Cadmium (Cd) displays nearly as great and widespread changes during the observation period as lead. Cd has previously been found to be associated with adverse health outcomes (kidney damage, bone disease).³⁶ The data also includes information on levels of Copper (Cu) and Zinc (Zn).

³⁶ Early Cd exposure has been shown 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 Schmeidler 2009). Air Cd and air Pb concentrations display a fairly high correlation at the neighborhood 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

Cu and Zn have been used as marker elements for motor vehicle emissions in previous studies and are therefore useful to include in the analysis as proxies for traffic density.³⁷ As we will show in the next section, the estimates are similar when controlling for these key pollutants, suggesting that there is less risk that other pollutants bias our results in any appreciable way.

There is also a risk that changes in local lead levels changes the composition of children growing up in the neighborhoods which could compromise our approach.³⁸ We investigate the importance of sorting by regressing predetermined characteristics of parents on their children's lead exposure.³⁹ Finding that key observed parental characteristics are

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; Olsson et al. 2005). Cd accumulates in crops, fish and livestock. But since only a small proportion of the food that children in Sweden (and elsewhere in most developed countries) eat is produced locally, it is *a priori* not expected that the intertemporal changes in *local* air-Cd levels in early childhood are necessarily associated with future outcomes.

³⁷ Brake lining wear is the major source of road traffic emitted Cu, and Zn concentrations are high in tiers (e.g., Hjortenkrans et al. 2007; Johansson et al. 2009).

³⁸ For example, if parental preferences for clean air are associated with higher parental investments in children, systematic residential sorting could overstate the effects of early lead exposure. On the other hand, pollution tends to be higher in densely populated areas and, at the same time, metropolitan areas often attract parents with more resources, provide better access to high-quality childcare, schools, healthcare and other amenities that are positively associated with child outcomes. Such local amenities could, in turn, for example, via gentrification, underestimate the role of childhood lead exposure.

³⁹ These parental characteristics can be considered as predetermined in the present context since >95% of the parents were born before 1960, i.e. before environmental lead exposure became a serious environmental problem in Sweden. Using mosses collected from 1860 until 1968, Röhling 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 40 mg/kg moss. They conclude that the first rise was probably due to industrial pollution, possibly due to the increased use of coal, and that it is *more than likely* that the second rise was caused by the rapid increase in the 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

correlated with local lead levels would increase the risk that also sorting on unobserved factors invalidates our results. The correlation between lead exposure and parental characteristics is shown in Table 3. We focus on characteristics that are likely to be among the strongest predictors of both sorting and child outcomes: income, education and age. Panel A shows the cross-sectional correlation, and in Panel B we add neighborhood fixed effects to the specification. Overall, although the point estimates are not statistically significant, the estimates in Panel A suggest that higher lead exposure for children is associated with lower levels of parental education, lower pre-birth maternal income, and lower maternal age at birth. For example, the cross-sectional estimates suggest that a 30 mg/kg increase in children's exposure (i.e. the average reduction in MPb levels across neighborhoods during the period we study) to lead is associated with a 1.5 percentage point *lower* probability of having a parent with a college education (-0.0005*30). This may imply that cross-sectional regressions are likely to overstate the effects of lead exposure on children's outcomes. After taking fixed differences across neighborhoods into account, the coefficients generally drop substantially and often change sign (e.g., a 0.3 percentage point *increase* in parental college graduation).

However, once more note that neither the point estimates in Panel A nor in Panel B are statistically distinguishable from zero, and that parents' baseline socio-economic characteristics show no consistent pattern with respect to their children's lead exposure during the phaseout. This is an interesting and important finding since it provides support for the identifying assumption: changes in key pre-determined factors influencing children's long-term outcomes (parents' earnings, age, and level of education) are, at least in the setting

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 influence the children's outcomes as adults.

we consider, not correlated with changes in their children's lead exposure. Having established this let us now proceed to our main results.

IV. Early lead exposure and long-term outcomes

In this section, we present our main results. To preview our findings, we show that even low levels of lead exposure deteriorate children's outcomes and that the effects are more pronounced in boys. Thereafter, we examine the potential importance of thresholds and find that the marginal effects increase with exposure. Building on these insights, we subsequently focus on the major reforms in 1980/81 and apply the difference-in-differences framework that uncovers the policy variation that allows us to visually assess how the effect of pre-reform lead exposure evolved across birth cohorts. Several heterogeneity and robustness checks follow.

A. Results from the linear panel data model

Table 4 presents the results from the linear panel data specification examining the effect of lead exposure on children's GPA, high school completion, and criminal convictions. In the first row, we only control for cohort and neighborhood fixed effects. Thereafter, we assess the role of unobserved factors by successively adding more controls. If we were to find that the point estimates change when we include key covariates, it would make us concerned that unobserved factors might bias the results. Our preferred specification is found in the bottom row and provides the neighborhood fixed effect estimates of an increase in MPb by 10 mg/kg, after including the full set of controls (individual, parental, municipal and other traffic-related pollutants).

The pattern is clear: Higher levels of exposure in early childhood are linked to lower GPA, a reduced likelihood of high school completion, and a greater risk of crime. Overall,

adding the controls hardly changes the coefficients at all. The only exception is the estimate for violent crime, which is always positive but eventually becomes insignificant. The coefficient for property crime is positive and stable across the different specifications, but is always statistically insignificant in this linear specification.

We subjected these results to a battery of robustness checks. The results from these analyses are reported in Appendix C2. Probably the most important of these is the role of differential trends across urban and rural areas. To investigate this we: (i) estimated regressions controlling for area-specific trends, (ii) excluded the most populated areas, and (iii) allowed for initial population density specific trends. In line with the results from Section 3.1, we find that the estimates from these exercises are similar to the baseline. These results do not fully rule out the risk of differential trends, but as a starting point it provides some confidence for our research design. In Appendix C2, we also address within-neighborhood sorting and account for correlated errors in alternative ways. These specification checks do not change the results in any significant or systematic ways.

Subgroup heterogeneity

Having established that our baseline results are not particularly sensitive to potentially important changes in the specification, we continue by investigating whether the effect of lead exposure differs by gender and socioeconomic status (SES). As discussed in Section 2, recent studies have documented a greater vulnerability in males to adverse early conditions, but the evidence of gender differences in the impact of lead is mixed. Hence, it is of interest to examine differences across genders in our low exposure setting. It is also highly relevant to examine potential heterogeneity in the effect depending on parental SES. Environmental policy is likely to benefit children's health in the poorest households the most because of higher levels of exposure due to sorting, less information about risks, different preferences

and/or possibilities for compensatory investments, and possible compounding effects through an interaction with other health conditions that are more common among the poor.⁴⁰ Whether early exposure to environmental polices affect adult non-health outcomes more among males and the poor is still an open question.

Panel A of Table 5 reiterates our baseline estimates, while panels B and C provide the same estimates for boys and girls separately. It is striking that the baseline results not only for crime but also for human capital are primarily concentrated among boys. Panels D and E report results from separate regressions by parental income.⁴¹ The point estimates are greater for low SES children, indicating that the benefits of the reductions in lead exposure are more pronounced among relatively disadvantaged children. However, in most cases it is not possible to statistically distinguish the effects across high and low-income groups.

⁴⁰ Parental resources may potentially help mitigate some of the negative effects of adverse conditions in early life (see e.g., Currie and Hyson 1999; Case et al. 2002; Cunha and Heckman 2007). Moreover, several studies have found that low SES children run a higher risk of being exposed to environmental hazards; either through residential segregation or by less care taken by polluters to reduce the risk of exposure in neighborhoods where families with little political and/or economic influence live (see e.g., Davidson and Anderton 2000). Information differentials about the health effects of pollution exposure between low and high SES households might also result in differential childhood lead exposure even within the same localities, since it may induce differential avoidance behavior across social groups (c.f. Neidell 2004). A SES gradient in the long-term effects of early childhood lead exposure could also be expected if parents from different social groups have different preferences or varying possibilities to compensate the impact of lead on subsequent outcomes. Finally, since children from poorer backgrounds more often suffer from other health problems, a SES gradient on the effect of early childhood lead exposure could result in interactive effects of lead exposure and other health problems (c.f. Currie et al. 2010; Jans, Johansson and Nilsson 2016). It would be interesting, although it is not possible to do so here, to fully differentiate the relative importance of the competing underlying mechanism behind the SES gradient. However, note that it is not necessary to do so from an identification perspective.

⁴¹ For the human capital outcomes, we split the sample by median parental income in 1971, 1976 or 1982 (i.e. on average two years before the first children in each cohort were born). For crime outcomes, we do the same in 1985 (the first year parental earnings are observed).

Since the effect of low lead exposure is mainly concentrated among boys, we will, for brevity, focus on boys from here on, although the results for girls are reported in the Appendix.

Effects on cognitive and non-cognitive skills

Our findings of much clearer effects of early lead exposure on long-term outcomes in males, combined with no differential effect across gender concerning blood lead levels, suggest that boys are more strongly affected by a given level of exposure than girls at least at low exposure levels. This finding is consistent with the results in Cecil et al. (2008) which show that early childhood lead exposure is correlated with a lower brain volume at age 20, particularly among boys, and particularly in the prefrontal cortex (PFC). PFC activity, in turn, is strongly correlated with non-cognitive traits such as conscientiousness and grit.

However, previous research has not quantified whether cognitive or non-cognitive skills are most important when accounting for the effects on later outcomes, or whether the different skills respond more strongly to a given level of exposure. A key contribution of this paper is to shed light on the underlying mechanisms. For this purpose, it is first necessary to document the direct effect of lead exposure on skills in our setting. Table 5 Panel F provides the linear panel data estimates for cognitive and non-cognitive skills in the full sample and separately by parental income. There is a significant negative relationship between early lead exposure and non-cognitive skills, but not with cognitive skills. The point estimates for children in high- and low-income families are similar and statistically indistinguishable. We return to, discuss, and build on these findings in a formal mediation analysis later in the paper.

B. The role of nonlinearities

While the evidence in previous studies is inconclusive as to whether the marginal effects of lead exposure vary over the distribution of exposure, knowledge of whether there are “safe” thresholds exist below which further reductions in exposure do not influence child development is of clear policy relevance.

To shed light on this, we estimate a panel data model similar to equation (1) but allow for a fully flexible functional form in the relationship between lead exposure and the outcomes. Figure 7 presents the non-parametric estimates of the slopes for non-cognitive (top) and cognitive skills (bottom). For non-cognitive skills, there is a clear nonlinear shape. Below around 30 mg/kg, the slope is flat, while above this level, it is consistently and significantly downward sloping.⁴² In contrast, the exposure response pattern for cognitive skills is flat and insignificant.

Figure 8 shows a hockey stick shape for the high school graduation rate which is similar to the shape for non-cognitive skills. Below around 30 mg/kg, further reductions in exposure have limited or no effects on graduation rates. Above 30 mg/kg, there is a clear and statistically precise negative relationship between early exposure and the probability of graduating from high school. For GPA, there is a tendency towards a nonlinear pattern at the bottom of the distribution (~20mg/kg), but we cannot reject linearity. Figure C2 provides the same estimates for females. For females, there is a tendency towards a nonlinear effect at the very top of the exposure distribution for GPA but, as expected from the linear model estimates, the overall pattern is much less clear for females than for males.

Figure 9 provides results for criminal convictions. For any crime, there is a similar pattern as for high school completion, but the relationship is less precisely estimated. For

⁴² We also examine the effects on the four sub-scores used to construct the non-cognitive index. Semi-parametric estimates for each of the sub-scores are presented in Figure C5. The estimates in this figure suggest that Psychological Energy (Conscientiousness), Emotional Stability (Neuroticism) and Social Maturity (Extraversion) are all related to early childhood lead exposure.

property crime, the relationship is clearer. Above approximately 65 mg/kg, there is a distinct and statistically significant positive relationship between lead exposure and property crime. Below 65 mg/kg, the regression line is flat and precisely estimated, suggesting that 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 (~ 30 mg/kg). To formally test for the location of these thresholds, we follow standard practice and estimate a series of piece-wise linear regressions that allow for a change of slope at different assumed thresholds (c.f. Hansen, 1999). Based on these regressions, Hansen's Likelihood Ratio (LR) test provides "no-rejection regions" containing the best estimate of the true location of the threshold.⁴³ Figures C3 and C4 show the LR statistic threshold locations. In general, they verify the location suggested by the more flexible semi-parametric regressions. An alternative interpretation for this pattern could be a nonlinear relationship between MPb and BPb. However, piece-wise linear spline regressions reveal no evidence of meaningful nonlinearities in the moss-blood relationship at the suggested thresholds (see Table B2).

In summary, the semi-parametric estimates reveal clear signs of nonlinearities in the relationship between early childhood lead exposure and adult outcomes. Since the average BPb level for the cohorts in our analysis sample was already initially lower than 10 µg/dL (Appendix B3), the threshold for the adverse effects of lead exposure on adult outcomes is, if

⁴³ The criterion for locating the true threshold is the assumed threshold at which the sum of squared residuals in the piecewise linear regression is minimized. Hansen's (1999) 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.

anything, located significantly lower than 10 µg/dL. We verify this in Section 5 where we use the estimates of the moss-blood link to find out which blood-lead levels the estimated neighborhood moss-lead thresholds corresponds to.

C. The effects of the 1980/81 reforms

The results presented so far rely on the variation in MPb exposure over the entire period of our study. An advantage of the full-sample analysis is that both effects of high and low levels of exposure are covered which, as shown in the previous section, is at least important for the interpretation of the relationship between lead and the crime outcomes. However, a potential concern with using all the data on changes in exposure is that it mixes reform induced variation in exposure with other (potentially endogenous) sources of exposure that may also have changed over time. To address this important concern, we now provide an analysis that focus on the 1980/81 reforms. A major benefit of targeting a clearly defined policy is that we have good information on the timing of exposure changes, which in turn gives us clear priors that allows for a sharp assessment of the plausibility of the common trends assumption.

While we have already described the basic intuition for our strategy in Figure 1, we now use the more granular cohort-by-cohort data and estimate model (2) using the children born in 1978 as the reference group. The 1978 cohort was the last cohort that had passed the critical age (24 months) after which the lead take-up (and BPb) starts to decline (Canfield et al., 2003) when the reform was fully implemented. Hence, the 1976 through 1978 cohorts represent cohorts “unexposed” to the reform. The children born in 1979 were “partially” treated because they were exposed to the 1980 reform from the age of 1, and the 1981 reform from the age of 2. The cohorts born after 1981 were fully exposed to the reform.

The coefficients for the interaction terms in model (2), γ_c , map out the dynamic reduced form effect (across birth cohorts from the same neighborhood) of the reform on adult

outcomes of being exposed to an additional unit higher pre-reform MPb level relative to the 1978 cohort. We plot the estimates to illustrate how the effects of exposure to the reform evolved for cohorts experiencing the critical ages before, during, and after the reforms. These estimates illustrate the exact timing of changes in later outcomes in relation to the age of exposure to the 1980/81 reforms. Allowing for a differential take-up by age, lasting environmental contamination and a slow excretion of lead stored in bones, we expect the pattern of the estimates to mirror the impact of the reform on BPb levels (Figure 6).

However, since we take our baseline panel data results seriously, we want to put them to a strenuous test using the reform analysis framework. In particular, before the 1980/81 reforms, the median pre-reform MPb level was 29 mg/kg. Hence, at least for the human capital outcomes, the estimated threshold documented in the semi-parametric analysis in Section 4.2 is well within the boundaries of the pre-reform exposure levels. This provides us with four predictions of how $\hat{\gamma}_c$ should evolve across cohorts, depending on the outcome, and the pre-reform lead levels in the neighborhoods of birth. If our baseline panel data results hold true, we expect the human capital outcomes: GPA, high school completion, and non-cognitive skills (Domain I) 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. For cognitive skills, there is no clear relationship at all.

Hence, if our baseline panel data results hold true, we expect that cognitive skills, property and violent crime (Domain II) for

- iii) children in neighborhoods *above* 30 mg/kg should all remain unchanged after the reforms, and
- iv) children in neighborhoods *below* 30 mg/kg should all remain unchanged after the reforms

Accordingly, we provide separate estimates of model (2) after splitting the sample at the human capital threshold (30 mg/kg). Figure D1 provides a visual summary of the predictions and hypothesized outcome patterns from an exogenous decrease in air lead levels.

Note that the predictions are derived from the analysis in Section 4.2 and hence, the results from this exercise also provide information about the validity of the panel data estimates. If these results are unrelated to the phaseout and simply driven by differential secular trends, we would not expect to see any systematic changes in the children's outcomes coinciding with the reform.

For brevity, comparability, and a clearer overview of the results, we summarize the estimates from the twelve separate estimations of model (2) on (standardized measures of) the Domain I and Domain II outcomes in Figure 10A. The figure plots the difference between the local averages of the γ_c coefficients (weighed by the inverse of the standard errors) in above vs. below-threshold neighborhoods. The thick black line shows the difference for the Domain I outcomes, and the thin black line shows the difference for the Domain II outcomes. Appendix D provides the full set of results with pointwise confidence bands and tests of equality of the pre-post coefficients, as well as a step-by-step guide to the construction of the summary graph in Figure 10A.

The results summarized in Figure 10A follow the outlined predictions. We can see that the coefficients for the cohorts exposed to the reform at older ages do not differ before the reform between children in above and below-threshold neighborhoods, for either Domain I or II outcomes, and are not statistically distinguishable from zero. For Domain I outcomes, it is clear that the coefficients in above and below-threshold neighborhoods start to diverge around the 1979 birth cohort (aged 1 at the first reform). The fact that the trend break coincides with the policy change suggests that the policy change itself is driving the changes in the outcomes that we see. While a test of equality of the pre vs. post-reform coefficients yields a *p*-value below .05 in the *above*-threshold neighborhoods for males, there are no consistent or significant changes for those born in *below*-threshold neighborhoods.⁴⁴ For Domain II outcomes, as predicted, 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.⁴⁵

An alternative approach to concisely summarizing the effects of the 1980/81 reforms on long-term outcomes can be seen in Figure 10B, which shows the estimates from a triple-difference (DDD) version of model (2) using a summary index of the measures of human capital (i.e. Domain I) predicted to be affected by the 1980/81 reforms. We follow

⁴⁴ F-test in above-threshold neighborhoods: 11.04 for GPA, 32.08 for high school completion, 7.65 for non-cognitive skills;

F-test in below-threshold neighborhoods: 1.44 for GPA, 1.22 for high school completion, 0.02 for non-cognitive skills.

⁴⁵ Two exceptions are worth noting. The first is the estimates for property crime where the F-test suggests that there could be a reduction in property 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 Appendix D Figure D5 despite the uncertainty of the location (if any) of the threshold and the failure to detect nonlinearities. From Figure D5, 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 noisier any crime category, there exists a threshold at similar locations as for human capital outcomes. However, we interpret this finding with caution given the poor precision of the estimates in the top panel of Figure 8 and the top left graph in Figure C4.

Kling, Liebman, and Katz (2007) and construct the summary index for each individual by taking an equally weighted average of the standardized measures of GPA, high school completion, and non-cognitive skills.⁴⁶ Then, we regress the index on the full sample DDD version of model (2), interacting all variables on the right-hand side with an above-threshold neighborhood indicator variable. The cohort-specific γ coefficients from this model capture the reduced form impact on the outcome index of an additional unit of pre-reform lead exposure in neighborhoods above relative to those below the threshold. We plot these coefficients and 90% pointwise confidence intervals, and perform a test of equality of the pre vs. post-reform coefficients in Figure 10B.

The estimates in Figure 10B follow a similar pattern as those in Figure 10A, and the impact of the reform on BPb levels (Figure 6): (i) no evidence of differential trends across the neighborhoods in the pre-reform cohorts; (ii) cumulative increasing differences after the implementation; and (iii) a leveling off some time after the reform was fully in place. These three figures support the common trends assumption, and the differential effects of the reform depending on baseline lead exposure and the outcome of interest. Consistent with lasting environmental contamination, they also indicate a lag of approximately two years before the benefits of the 1980/81 reforms fully materialized. This lag length is identical to the one used by Strömberg et al. (1995) when relating gasoline lead content to children's BPb levels. For women, the coefficients mirror the panel data results, and suggest no clear effects of the 1980/81 reforms (see Figure D8).

Before summarizing the findings so far, we would like to mention two important robustness checks. First, we always use neighborhood of birth rather than neighborhood of residence when assigning exposure in order to minimize the risk of bias due to residential

⁴⁶ We obtained almost identical results when we instead replaced the equally weighted index with an index from a principal component analysis of the three standardized measures.

sorting. However, endogenous residential sorting prior to birth could potentially bias our reform analysis estimates. We already showed in Section 3.1 that there is no evidence of sorting based on actual neighborhood lead levels. To address this concern using the reform analysis framework, we tested whether changes in parental education and income are systematically correlated with the pre-reform lead level in the neighborhood of birth around the time of the reform, and whether maternal mobility between the year before birth and the year of birth is related to the lead level in the residential area of the mother in the year before birth. We found no indications that parental sorting is an important source of bias in this setting.⁴⁷

Second, an alternative to using the pre-reform moss-lead level to predict changes in exposure is to simply use the actual changes in exposure within the neighborhood of birth before vs. after the reform. Although this sounds like a reasonable alternative, the actual variation would include all changes in exposure that happen to coincide with the timing of the reforms. To avoid conflating the estimates with other local policies or changes in local conditions that also affect the long-term outcomes, our baseline model uses the pre-reform moss-lead level as a predictor of the changes. However, as shown for the human capital index outcome in Figure 10C, using actual changes in exposure instead of the pre-reform moss-lead level predictor of changes in exposure provides a similar picture of the impact of the reform.

⁴⁷ These results are available upon request. We also tested for systematic mobility around the reform (i.e. moving to a high/low exposure area), finding no indications of such a pattern. We did find some indications that the probability of moving from a pre-reform higher exposure neighborhood of birth to a lower pre-reform exposure neighborhood during childhood *decreased* somewhat after the reform. Reduced mobility out of pre-reform high exposed areas is consistent with parents valuing the reform induced reduction in lead exposure. The slight reductions in out-migration from pre-reform higher exposure areas may negate some of the reform-induced reductions in lead exposure since the cumulative exposure throughout childhood becomes higher than it would have been in the absence of changes in out-migration. Most importantly, the reduction in out-migration to lower exposure areas after birth is identical in the below and above threshold neighborhoods indicating that mobility after birth is not an important source of bias in our analysis.

To avoid the potential endogeneity concerns when using actual changes in exposure, we also generated a neighborhood-specific predictor of the changes in lead exposure before vs. after the reform using a “leave-out” procedure. Specifically, we excluded all data for the CZ of neighborhood n . Then, we augmented the first-stage in model (3) with a quadratic term in the pre-reform moss-lead level and interacted this with the reform timing dummies (phase-in, full-reform).⁴⁸ Using the individual data for all children (except those in the same CZ), we estimated the augmented first-stage version of model (2). For each neighborhood in the excluded CZ, we then calculated the average predicted change in exposure between the pre- and post-cohorts using only the pre-reform moss-lead level in neighborhood n and the “leave-out” first-stage estimates.⁴⁹ We repeat the procedure for all CZs. The resulting neighborhood-specific treatment intensity variable is the predicted change in neighborhood moss-lead levels based on the experiences of neighborhoods in other CZs with the same pre-reform moss-lead level. This “leave-out” estimate based on the estimated reform changes excluding all data from the own CZ avoids the endogeneity of using actual changes in neighborhood n . Figure 10D shows that using the predicted change in the neighborhood of birth based on the experiences of similar neighborhoods in other CZs provides a similar picture of the evolution of the impact of the reform across birth cohorts.

In summary, the timing of the changes in outcomes shown in this section reinforces our view that the impact on adult outcomes documented in the linear and semi-parametric panel data analysis reflects changes in childhood lead exposure caused by the reforms. Importantly, if the results in our panel analysis were due to unobserved differential trends

⁴⁸ The quadratic specification for the full sample is motivated by the nonlinear first-stage shown in Figure 1.

⁴⁹ The predicted change in neighborhood n is calculated for the 1982-1984 cohorts using estimates from all other commuting zones is given by $\text{ReformChange}_n = \sum_i^N (\widehat{\pi}_1 \cdot \text{Lead}_{n,i}^{\text{Pre-Reform}} + \widehat{\pi}_2 \cdot (\text{Lead}_{n,i}^{\text{Pre-Reform}})^2) / N$ where i denotes children in the neighborhood of birth, and $\widehat{\pi}_1$ and $\widehat{\pi}_2$ are the linear and quadratic estimates from the augmented first-stage equation. We use the same controls in both specifications.

across high and low-exposure neighborhoods, we would not expect to find such a systematic pattern around the timing of the 1980/81 reforms. Since there are no clear or significant effects of the reform on children in the below-threshold neighborhoods, these results provide support for the much weaker relationship below certain thresholds as documented in the semi-parametric regressions. The reform analysis also supports the differential impact of early lead exposure across genders since the effect of the reform on long-term outcomes is much clearer for males, despite no obvious gender differences in exposure (see Table 1, Panel B).

2SLS estimates

Next we build on the reduced form results and present 2SLS estimates of the full-reform effect as outlined in model (3). Due to the absence of reduced form effects for crime outcomes, we focus on human capital outcomes. The full-reform 2SLS estimates are presented in Table 6. Panel A (B) shows the estimate in above (below) threshold neighborhoods. The first-stage *F*-statistic is above 10 in both below/above-threshold neighborhoods, suggesting less risk for a weak instrument bias. For comparison, using the 1976-1984 sample, we also show OLS estimates that do not account for the possible endogeneity and measurement error in early childhood lead exposure.⁵⁰

The 2SLS estimates in the above threshold neighborhoods are significant and positive for all outcomes except cognitive skills. In the below-threshold neighborhoods, despite a strong first-stage, the 2SLS estimates are much smaller, sometimes switch signs, and are always statistically insignificant. Overall, the OLS estimates for the reform sample follow a similar pattern as the 2SLS estimates but are smaller in magnitude, consistent with

⁵⁰ For an accurate comparison with the 2SLS estimates, in the OLS fixed effects regressions we include all cohorts in the full reform sample but interact all right-hand side variables with a phase-in period dummy and report the estimates comparing the pre-vs. full reform changes in exposure only.

measurement error attenuating the OLS estimates. For females, the 2SLS estimates follow the same pattern as before and show a more muted (compared to males) and insignificant impact of low levels of early lead exposure (Table D1).

D. Additional results

Here we report results from supplementary analyses using different outcomes and specifications. The purpose is to provide evidence of relevant questions that have not been addressed in the main analysis. We start by asking whether we can detect effects at other ages of exposure. Then we present evidence of the impact on long-run labor market outcomes. Finally, we show within-sibling estimates and compare these to our baseline model. All the results are reported in Appendix E.

Prenatal exposure, early childhood, or later exposure?

The results presented so far focus on the effects of lead exposure in early childhood (ages 1 to 3). As discussed earlier, this setup is natural given that children's BPb levels peak at the age of 2 due to increased exposure and take-up, and also that it is a period where children are more susceptible to damage due to the varying sensitivity to lead over the child's life cycle.⁵¹ Still, it is possible that even earlier exposure matters. Our early childhood measure of lead exposure is likely to be correlated both with the level of exposure experienced *in utero* and also with later childhood exposure.

⁵¹ In Appendix B1 we summarize the evidence in the medical literature which finds that children's BPb levels follow an inverted v-shaped pattern over the first 60 months of their lives. A key determinant of this age exposure profile is the well documented age differences of hand-to-mouth activities and that infants and toddlers spend more time on the floor/ground than older children, which makes them more exposed to lead particles. Xu et al. (2007) find that the child hand-mouth activity decreases with age, but find no gender differences. Infants and toddlers are also likely to be more susceptible to damage from lead due to the sensitive and rapid period of development.

To provide evidence of the role of prenatal exposure, we tested whether birthweight, low birth weight and prematurity were affected by the 1980/81 reforms. Appendix Figures E1 and E2 show that none of these measures of neonatal health was significantly affected. Although commonly used for assessing the effects of maternal lead exposure (e.g. Zhu et al., 2010), birthweight and gestational age only are crude proxies of neonatal health, and as such our results should only be viewed as indicative for the relative impact of pre- and post-natal exposure to local air lead levels on outcomes later in life.

Evidence of the effects of later childhood exposures can be elicited from the reform analysis results in Figure 10. Children aged three or older at the time of the reform do not seem to have been affected nearly as much as those exposed from younger ages. To see this, note that Figure 10 shows no indication of a trend in outcomes across the children aged three to five at the time of the reform (the cohorts born between 1976 and 1978) (see Figure D1 for intuition). Consistent with the age-BPb profile, this suggests that local air lead levels are a stronger predictor of later outcomes in infants and toddlers than in older children.

Labor market outcomes

While our main analysis focuses on those relevant outcomes for which we are able to follow all cohorts of children over an identical age span, we would ideally also like to estimate the effect on labor market outcomes. However, a key complication is that Sweden is one of the countries in the OECD with the highest age for university graduates (the average age is 29), and a large share of each cohort (40%) graduates from higher education (OECD 2014). This means that many individuals in our sample are still enrolled in education at the ages when we can measure their labor market outcomes.⁵² For this reason, it might not be surprising that in

⁵² Böhlmark and Lindquist (2006) show that credible measures of permanent earnings require current earnings to be measured in the early to mid-30s for men and even later for women.

Appendix F, we find no significant effect of early lead exposure on earnings in 2013 when the individuals are aged between 29 and 40. Future work could use the same analysis to examine the effects on earnings at ages when current earnings better reflect permanent earnings.

Comparing siblings

Previous studies argue that sibling fixed effect estimates may provide information on how parents respond to early health insults in terms of investing in children (Griliches, 1979). The investment strategy that parents employ depends on parental preferences and the production technology available to them. While some parents may prefer remedial investments in response to early health insults, it is possible that other parents reinforce the initial disadvantage by allocating resources to the child in better health. Yi et al. (2015) show empirically that the family acts as a net equalizer in response to early life health shocks across children in China.

For this reason, Appendix Table E2 provides sibling fixed effects estimates. Overall, the sibling fixed effect estimates are reasonably similar to the sibling sample OLS estimates. In the above-threshold neighborhoods, the sibling fixed effects estimates are sometimes greater and sometimes smaller than the sibling sample estimates. In the below-threshold neighborhoods, all estimates are much smaller in magnitude and insignificant.

While it is difficult to draw any firm conclusions without actual data on parental investments within the household, the overall similarity across the specifications in Table E 2 may suggest that parental investments do not vary with respect to early lead exposure.

V. Interpretation

The existence and location of moss lead thresholds are relevant to policy in themselves, since many countries and cities use moss to monitor air lead levels.⁵³ In this section, we try to extract further information by translating the moss-lead thresholds to blood-lead thresholds. We also implement a decomposition analysis where we ask what share of the effect on adult outcomes is mediated through the policy induced changes in observed skills and what share is explained by other unobserved skills.

Assessing the magnitude of the estimates

We start by using our estimates of the moss-blood elasticity (see equation (B1) in Appendix B) to translate the estimated MPb thresholds into the more easily interpreted BPb levels. We then use these numbers to calculate the hypothetical annual earnings gains from reductions in BPb levels.

Evaluated at the mean of the independent variables, adjusting the estimates using the age-specific blood-gasoline lead elasticity estimated by Reyes (2007), and assuming that the additive separable specification used in the estimation holds for both populations, we find that the relevant BPb level for property and violent crime (~50 mg/kg) corresponds to an early childhood BPb level of just above 7 µg/dL. The corresponding number for the high school completion and non-cognitive skills thresholds is approximately 5 µg/dL.

It is important to bear in mind that we measure exposure at the neighborhood level and hence, the documented threshold for human capital is associated with a *neighborhood* average BPb level above 5 µg/dL. In principle, it is possible that the entire effect could be caused by large effects on the development among a few children with very high BPb levels. However,

⁵³ 28 European countries measure local lead levels in moss regularly, and many regions and cities also do so at less regular intervals. 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

we find this explanation to be less plausible since the standard deviation in childhood BPb levels is not extremely large, thus suggesting that it would require a massive impact from still relatively low levels of exposure to account for the average effects in the full population. That said, the exactness of this BPb level threshold remains to be confirmed in future research using individual childhood BPb levels, individual adult outcomes, and an exogenous variation in exposure.

Next we use the information on the BPb level thresholds to draw conclusions about the real world significance of the results. This is done by considering the consequences of a decrease in early childhood BPb levels from 10 µg/dL to 5 µg/dL. This change corresponds to the latest change in the guidelines for elevated BPb levels used by the CDC. In the United States alone, 530,000 children below the age of five have BPb levels above 5 µg/dL, and 150,000 out of these are above 10 µg/dL (CDC 2012). Using the above predictions, it is, for example, possible to calculate the hypothetical annual gains from reducing the BPb from 10 to 5 µg among these children.

We impute the effects on mid-30s earnings using the estimated impact on high school completion. The Swedish high school graduation premium on earnings at the age of 32 is 17 percent (Nilsson, 2009). Combining this number with the estimated effects of the 1980/81 reforms on high school graduation rates, we find that reducing early childhood BPb levels from 10 to 5 µg/dL leads to around 4.4 percent higher lifetime earnings (among males). Naturally, this estimate only captures the effect of lead on earnings that operates through high school completion. 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 assumed average annual income of USD 30,000, the benefits in terms of increased earnings from reducing the BPb level in these children would hence be around USD \$198 million annually after the age of 32. This reflects the effect on the average population of children, but

since 60% of all children with a BPb above 10 µg/dL are economically disadvantaged (see Currie, 2009), our results suggest that the expected effects on individual earnings could be greater.

Decomposing the effect by source

Next we assess how much of the effects of early lead exposure on adult outcomes can be explained by the impact of lead exposure on observed skills and how much is unaccounted for. For this purpose, we implement a mediation analysis similar in spirit to Heckman et al. (2013). Using the enlistment data, we decompose the effects of lead exposure on later outcomes into the effects via measured cognitive and non-cognitive skills, and the effect via unmeasured skills. The decomposition method is described in detail in Appendix F, and Figure 11 shows the relative contribution of the cognitive and non-cognitive skills and other (unmeasured) factors to the total effect (normalized to 100 percent).

The top five bars show that the panel data estimates of the effect of low levels of early lead exposure on later outcomes primarily operate through non-cognitive skills. Changes in non-cognitive skills explain between 20 to 30 percent of the effect of changes in lead exposure on human capital, and 25 to 30 percent of the crime convictions up to the age of 24, and the remainder of the effects is accounted for by effects of early lead exposure on other unmeasured skills.

The two bottom bars of Figure 11 provide a decomposition using the 2SLS estimates for high school completion and GPA in the above-threshold neighborhoods. The estimated changes in cognitive and non-cognitive skills from the 2SLS-model accounts for a greater share of the total effects of early lead exposure (~ 40%), but overall the results are well in line with the findings from the panel data model. Non-cognitive skill seems to be a much more

important contributor to the overall effect on children's long-term outcomes than cognitive skills at low levels of exposure.

Given previous research on the link between lead and crime, and lead and prefrontal cortex development (Cecil et al, 2008), we did not preclude to find an impact on non-cognitive skills even in our low exposure setting. However, we were more surprised to find no consistent effect on cognitive skills at age 18 given the many epidemiological studies documenting an association between lead and direct measures of cognitive skills in childhood. This interesting and surprising finding could be due to a number of different reasons: (1) much of the epidemiological literature looks at exposure levels that are much higher than in this context (i.e. $>10\mu\text{g}/\text{dl}$); (2) the epidemiological studies studying associations at lower levels of exposure ($<10\mu\text{g}/\text{dl}$) often suffer from small sample sizes and often find *steeper* dose-response functions at lower exposure which is typically described as "counterintuitive" Canfield et al. (2005), or "a mystery" (Skerfving et al. 2015). An obvious explanation for this could be that confounding becomes more important at low levels of exposure.⁵⁴ (3) The recent design based studies that address the endogeneity concerns and looks at low levels of exposure have used school achievement test-scores in grade 3 or 4 as proxies for cognitive performance. We use direct measures of cognitive ability and note that it has been shown that grades and achievement test scores can be substantially explained by non-cognitive personality traits (Borghans et al. 2015, 2011). (4) The effects on cognitive ability may be

⁵⁴ A potential explanation is that the composition of children that are exposed to low levels of lead is likely to differ from the composition of children exposed to higher levels. To give a concrete example, in the US 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 have been exposed, namely children living in low-standard housing with deteriorating water pipes, lead paint walls, or living close to lead-emitting industries. Therefore, it is possible that the bias from unobservables is stronger in a low lead setting than in a high lead setting, generating a steeper dose-response curve in settings where the sources of exposure are more likely to reflect unobserved family characteristics.

transient rather than permanent. While this seems less likely given the cognitive rank stability that generally emerges after around age 10, it is possible that direct measures on cognitive ability earlier on in the child's life-cycle may have revealed a different pattern. While it is out of scope of this paper to further disentangle these alternative explanations, we hope that our findings contribute to further studies that may be able to shed light on the relative importance of cognitive and non-cognitive skills in accounting for the effects on long-run outcomes.

A few words of caution are also warranted regarding the mediation analysis. First, a key assumption for the mediation analysis is that the change in exposure does not also shift the production function. We investigated this by using our reform analysis framework in model (2) and allowed the returns to the cognitive and non-cognitive skills to vary with the instrument. While the returns to non-cognitive skills is stable and not significantly affected by the changes in exposure, the returns to cognitive skills (for high school completion) becomes significantly lower. Since the decomposition use the estimates of the factor loadings for the full reform sample, the estimated reduction in the return to cognitive skills indicates that the contribution of cognitive skills in Figure 11 could, if anything, be *overstated* in our mediation analysis.

Second, the mediation analysis does not only assume that our baseline estimates reflect a causal relationship, but also that unobserved factors are (conditionally) independent of the link between skills and adult outcomes, and orthogonal to the link between lead exposure and skills. Since unobserved factors are likely to be positively correlated with the outcomes and with skills, this would overstate the role of observed skills relative to unobserved skills. For this reason, we prefer to think of this method as primarily a descriptive tool to better understand our results and for comparison with other studies.

VI. Concluding remarks

We document the effects of early exposure to low lead levels on adult outcomes and try to explain their sources. Our analysis takes advantage of the Swedish phase-out of leaded gasoline, a novel measure of lead exposure, and population-wide administrative data. Contrary to the unexplained but common finding in cross-sectional studies that the marginal effects are greatest at low lead levels (EPA 2013), but consistent with the benchmark toxicological dose-response model, we find that the relationship between early lead exposure and non-cognitive skills, crime, and high school completion becomes much weaker below certain thresholds.

An estimated 535,000 children in the United States have BPb levels at or above the reference value for elevated BPb levels established by CDC in 2012 (5 µg/dL). 150,000 of these children's levels are ≥ 10 µg/dL. Globally, the WHO estimates that more than 40 percent of the children have BPb levels above 5 µg/dL. Our results suggest that early lead exposure may have deleterious effects on the academic performance among children with BPb at levels from 5 µg/dL and above, and criminal convictions from around 7 µg/dL. The magnitude of the effect is of clear economic significance. While the exposed cohorts are still too young to obtain 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, solely via the effects on high school graduation, increases the lifetime earnings by around 4%. If the effects via crime and non-cognitive skills are taken into account, the effects are presumably larger.

In terms of mechanisms, non-cognitive skills seem to play a much greater role than cognitive skills at the low levels of exposure considered. 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 an estimate of the effects of lead exposure on adult outcomes, but also offers insights concerning effective parental and public remedial investments following early lead exposure. Our results suggest that interventions following

low levels of exposure that specifically target non-cognitive skills are likely to be more successful than those focusing on cognitive skills.

Stark disparities in lead exposure continue to exist in the US, where the age of the housing stock together with the neighborhood poverty status constitute key predictors of elevated BPb levels in children (Roberts and English, 2016). Interestingly, we find that boys are more vulnerable to lead exposure. This finding suggests that early lead exposure may be one contributing explanation for why growing up in disadvantaged areas is especially harmful to boys (Chetty et al. 2016). In line with previous studies, we find no gender differences in the relationship between lead exposure and early BPb levels. Hence, an important task for future research is to provide evidence on the causes of the greater impact of early adversity on boys. Is it explained by a general greater vulnerability? Or do parents, teachers, or others differ in their responsive investments to the behavioral changes induced by for example lead exposure between boys and girls? Or, given the null-effects on cognitive skill, are compensatory investments targeted towards non-cognitive skills more effective in girls than in boys?

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Table 1 Lead exposure and children's BPb level

Panel A: The relationship between MPb and children's BPb levels ($\mu\text{g}/\text{dL}$) in *Landskrona*

<i>Dependent variable:</i>	ln (BPb)	ln (BPb)	ln (BPb)
<i>Specification:</i>	(1)	(2)	(4)
ln(MPb)	.4861*** (.1191)	.4774*** (.1177)	.4957*** (.1196)
ln(MPb) \times Girl			-.0556 (.0642)
Neighb. fixed effects	Yes	Yes	Yes
Year fixed effects	Yes	Yes	Yes
Individual controls	No	Yes	Yes
R-squared	.56	.58	.58
No of children	242	242	242

Panel B: The impact of the 1980/81 reforms on children's BPb levels ($\mu\text{g}/\text{dL}$) in *Trelleborg*

<i>Dependent variable:</i>	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)
<i>Specification:</i>	(1)	(2)	(3)	(4)
Reform \times Urban	-.2036*** (.0568)			-.2278*** (.0867)
Reform (Phase-In) \times Urban		-.1484** (.0661)	-.1994*** (.0749)	
Reform (Full) \times Urban			-.2196*** (.0773)	-.2698*** (.0847)
Reform \times Urban \times Girl				-.0123 (.1181)
R-squared	.29	.30	.30	.31

No of children	651	651	651	651
Individual controls	Yes	Yes	Yes	Yes
Urban area fixed effects	Yes	Yes	Yes	Yes
Period fixed effects		Yes		
Year fixed effects			Yes	Yes

Notes: Panel A reports OLS estimates from equation (B1). BPb (BPb) is measured in (ln) $\mu\text{g}/\text{dL}$ blood and MPb (MPb) exposure in (ln) mg/kg of moss assigned to children's residential address in Landskrona municipality. Individual controls include: gender, age dummies, whether the child is practicing any lead exposure hobbies, and ln(hemoglobin) level. Panel B reports difference-in-differences estimates of the impact of the 1980/81 reforms on children's BPb levels, comparing the differential impact of the reform on children residing in urban areas vs. the rural areas of Trelleborg municipality. The estimation sample consists of 651 primary school age children (rural n=325, urban n=326). Individual controls are gender and age dummies. Period fixed effects are indicator variables for when the BPb levels were sampled (pre, phase-in, and post reform as defined in text) and year fixed effects are indicator variables for the year when BPb was sampled. Robust standard errors are reported in parenthesis. */**/** denote significance at the 10/5/1 percent levels, respectively.

Table 2. Descriptive statistics and definitions for key variables

Variable	Definitions	Mean	Std. Dev
			Full Sample
<i>Outcomes</i>			
GPA	Compulsory school grade point average (%-tile ranked within graduation cohort) [typically at age 16]	50	28
Cognitive Skills	Cognitive test score at military enlistment (Stand. within enlistment year cohort) [age 18; males only]	0	1
Non-Cognitive skills	Non-cognitive test score at military enlistment (Stand. within enlistment year cohort) [age 18; males only]	0	1
High School	=1 if completed high school, 0 otherwise	.79	.31
Ever convicted	=1 if ever convicted in a criminal trial up to age 24, 0 otherwise	.16	.37
Property crime	=1 if ever convicted for a property crime up to age 24, 0 otherwise	.07	.16
Violent crime	=1 if ever convicted for a violent crime up to age 24, 0 otherwise	.02	.26
<i>Lead Exposure</i>			
Moss-Lead Level	Neighborhood of Birth Moss-lead level Mg/Kg (see text)	34	17
<i>Additional Variables</i>			

Share males		.51	.49
Birth order		1.9	.98
Mothers age at birth	Maternal age at birth	27.0	5.0
Birth weight	Birth weight in grams	3482	561
Parents Education	=1 if at least 1 parent completed college, 0 otherwise	.41	.49
Employment rate	Number of employed/total population (in municipality of birth)	.48	.12
Zn Exposure	Neighborhood of Birth Moss-Zink level Mg/Kg (see text)	61.8	15.3
Cu Exposure	Neighborhood of Birth Moss-Copper level Mg/Kg (see text)	8.2	2.4
Cd Exposure	Neighborhood of Birth Moss-Cadmium level Mg/Kg (see text)	.52	.18

Note: Descriptive statistics for N= 699,155 children included in the full sample analysis.

Table 3. The link between predetermined parental characteristics and their children's lead exposure

Panel A:	Parents completed college	Mother completed college	Father completed college	Parents' pre- birth earnings (log)	Father's pre-birth earnings (log)	Mother's pre-birth earnings (log)	Mother's age
Lead exposure (mg/kg)	-.005 (.004)	-.004 (.004)	-.003 (.004)	.004 (.003)	.004 (.003)	-.001 (.006)	-.004 (.029)
Neighborhood FEs	No	No	No	No	No	No	No
Panel B:	Parents completed college	Mother completed college	Father completed college	Parents' pre-birth earnings (log)	Father's pre-birth earnings (log)	Mother's pre-birth earnings (log)	Mother's age
Lead exposure (mg/kg)	.001 (.001)	.001 (.001)	-.0001 (.001)	.003 (.003)	.003 (.002)	.003 (.003)	-.014 (.028)
Neighborhood FEs	Yes	Yes	Yes	Yes	Yes	Yes	Yes

Notes: The table presents the coefficients from OLS regressions of baseline parental characteristics measured two years before birth on their children's early lead exposure. All regressions control for cohort of birth fixed effects. The coefficients and standard errors are scaled by a factor of 10 so that the estimates can be interpreted as the effect of increasing lead exposure by 10 mg/kg. Standard errors (in parenthesis) are clustered at the CZ level. */**/*** denote significance at the 10/5/1 percent levels, respectively.

Table 4. Panel data estimates of the effect of childhood lead exposure for the full sample

<i>Outcomes:</i>	Grade 9 GPA (pct. rank)	P(High school completion)	P(Ever convicted)	P(Property crime)	P(Violent crime)
	(1)	(2)	(3)	(4)	(5)
Baseline	-.1890** (.0961)	-.0034** (.0014)	.0014** (.0007)	.0007 (.0008)	.0004** (.0002)
+ individual & family controls	-.2018*** (.0716)	-.0038** (.0015)	.0017** (.0006)	.0008 (.0007)	.0005** (.0002)
+ municipality characteristics	-.2009** (.0716)	-.0039** (.0015)	.0018** (.0007)	.0008 (.0007)	.0005** (.0002)
+ other traffic pollutants	-.2097** (.0925)	-.0031** (.0014)	.0018** (.0008)	.0009 (.0009)	.0002 (.0003)
Mean of dependent variable	[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. The coefficients and standard errors are scaled by a factor 10 so that the estimates can be interpreted as the effect of increased lead exposure by 10 mg/kg. The sample consists of all children born in Sweden 1973-1974, 1977-1979 and 1982-1984. Crime (in columns 3-5) is defined as having been convicted at least once by age 24. Childhood lead exposure is measured as an average over ages 1-3. The baseline model only controls for cohort and neighborhood fixed effects. Then, we 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, dummies for the quartile of total parental earnings two years before birth (in 1985 for crime outcomes), and 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. The sample means are shown in brackets. The individual data is aggregated into 2,559 neighborhoods of birth that are divided into 74 commuting zones (CZ) defined by Statistics Sweden. All regressions are weighted by the number of children in each cohort-by-neighborhood cell. Cluster robust standard errors (at the CZ level) are shown in parenthesis. */**/*** denote significance at the 10/5/1 percent levels, respectively. The preferred (full) specification is highlighted in bold.

Table 5. Heterogeneity and effects on cognitive and non-cognitive skills, Full sample

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

		<i>income</i>	<i>income</i>	<i>income</i>	
Lead exposure	.0002 (.0024)	-.014** (.0069)	-.016** (.0007)	-.012 (.0007)	.004 (.0035) -.005 (.0005)
Sample mean	.026	.014	.1265	-.114	.1511 -.116

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. 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 includes the full set of controls as specified in the note of Table 4. Cluster robust standard errors (at the CZ level (74 cells)) are shown in parenthesis. */**/** denote significance at the 10/5/1 percent levels, respectively.

Table 6. OLS and 2SLS estimates of the effect of the 1980/81 reforms on males

Dependent variable:									
Panel A:									
	Above-threshold		GPA		P(High school graduation)		Non-cognitive		Cognitive skills
	OLS	2SLS	OLS	2SLS	OLS	2SLS	OLS	2SLS	
Lead exposure (mg/kg)	-.164*** (.030)	-.256*** (.0769)	-.0022*** (.0006)	-.0044*** (.0008)	-.0056** (.0020)	-.0124*** (.0030)	.0003 (.0009)	-.0015 (.0024)	
Commuting zones:	46	46	46	46	46	46	46	46	
Neighborhoods	1,313	1,313	1,313	1,313	1,313	1,313	1,313	1,313	
Children	172,786	172,786	172,786	172,786	172,786	172,786	172,786	172,786	
First-stage <i>F</i> -statistic	N/A	49.1	N/A	49.1	N/A	49.1	N/A	49.1	

Dependent variable:									
Panel B:									
	Below-threshold		GPA		P(High school graduation)		Non-cognitive		Cognitive skills
	OLS	2SLS	OLS	2SLS	OLS	2SLS	OLS	2SLS	
Lead exposure (mg/kg)	-.0109 (.0571)	-.0534 (.1633)	.0009 (.0008)	-.0014 (.0033)	.00251 (.0024)	.01095 (.0085)	.0007 (.0016)	-.0031 (.0037)	
Commuting zones	71	71	71	71	71	71	71	71	

Neighborhoods	1,138	1,138	1,138	1,138	1,138	1,138	1,138	1,138
Number of Children	123,780	123,780	123,780	123,780	123,780	123,780	123,780	123,780
First-stage <i>F</i> -statistic	N/A	19.4	N/A	19.4	N/A	19.4	N/A	19.4

Notes: The table presents OLS and 2SLS estimates from equation (3) for the reform sample. For comparability across outcomes, the estimation sample consists of males for whom all outcomes are non-missing. The instrument is the interaction term (post-cohorts dummy \times pre-reform MPb level). All regressions control for neighborhood fixed effects and the baseline controls in Table 4. The 2SLS model also controls for the phase-in period (1979-1981) interacted with the pre-reform moss-lead level, interpolated pollution measures (Cd, Cu, Zn) for the cohorts 1980-1982, as well as gasoline sales in the municipality of birth. The OLS model nets out all influences from the phase-in period using phase-in dummies interacted with all control variables to provide estimates comparable with the 2SLS estimates. Standard errors are two-way clustered at the neighborhood-by-cohort and commuting zone level. */**/*** denote significance at the 10/5/1 percent levels, respectively.

Figure 1. Pre-reform lead vs. changes in lead and high school completion

Notes: The figure shows raw data changes in the neighborhood MPb level (left-hand y-axis) and changes in the probability of high school completion (right-hand y-axis) for males born between 1977-1979 and 1982-1984 against pre-reform neighborhood lead exposure (average during 1977-1979) as measured in moss (mg/kg). The solid/dashed lines are local linear regressions of changes in lead exposure/high school completion on pre-reform neighborhood lead levels, weighted by the number of children in each neighborhood (using a bandwidth of 10 mg/kg). The histogram shows the density of pre-reform moss-lead levels in the neighborhood of birth (top and bottom 1% excluded).

Figure 2. 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 ~120 elementary school children per year (c.f. Stromberg et al. 1995 and Stromberg et al. 2003). Note that the BPb measurements are taken from children in two different cities in southern Sweden (alternating the city every other year), and hence are included to give an overview of the general trend. See Figure 6 for trends in BPb around the 1980/81 reforms in a more homogenous sample.

Figure 3. Neighborhood of birth lead exposure levels in 1975 (left) 1985 (right), (in color)

Figure 4. Changes in neighborhood moss lead exposure for the cohorts included in the main analysis

Figure 5. Within-neighborhood changes in lead exposure 1975-1985

Figure 6. The impact of the 1980/81 reforms on children's BPb levels

Notes: The figure shows BPb levels among primary school children in Trelleborg before, during, and after the 1980/81 reforms (data from 1977-1985). The solid line shows the average BPb levels of the children residing in the urban areas and the dashed line children in the non-urban areas of Trelleborg municipality using a local mean smoother (bandwidth: 1 year) (see the text for details). In this figure we trim the sample by excluding one observation from the 1983 non-urban sample with an extreme BPb level (4 [2.3] times higher than the 50%-tile [95%-tile] BPb) but keep that observation in the regression sample used in Table 1. The figure also shows the maximum total lead sales in metric tons over the years 1976-1984 (hollow circle line) in Sweden 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 2) in the respective year. For medium grade fuel (which is a blend of regular and premium gasoline) in the year 1980, we use the average max lead content allowed in regular and premium gasoline since the maximum allowed lead content in 1980 differs between premium and regular gasoline.

Figure 7. Semi-parametric fixed effects estimates for non-cognitive and cognitive skills

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 non-cognitive skills and the dependent variable in the bottom graph is cognitive skills, as measured at age 18 at military enlistment. See Table 4 for the full set of controls.

Figure 8. Semi-parametric fixed effects estimates for human capital (Males)

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 the share that completed high school and the dependent variable in the bottom graph is GPA. See Table 4 for the full set of controls.

Figure 9. Semi-parametric fixed effects estimates for crime (Males)

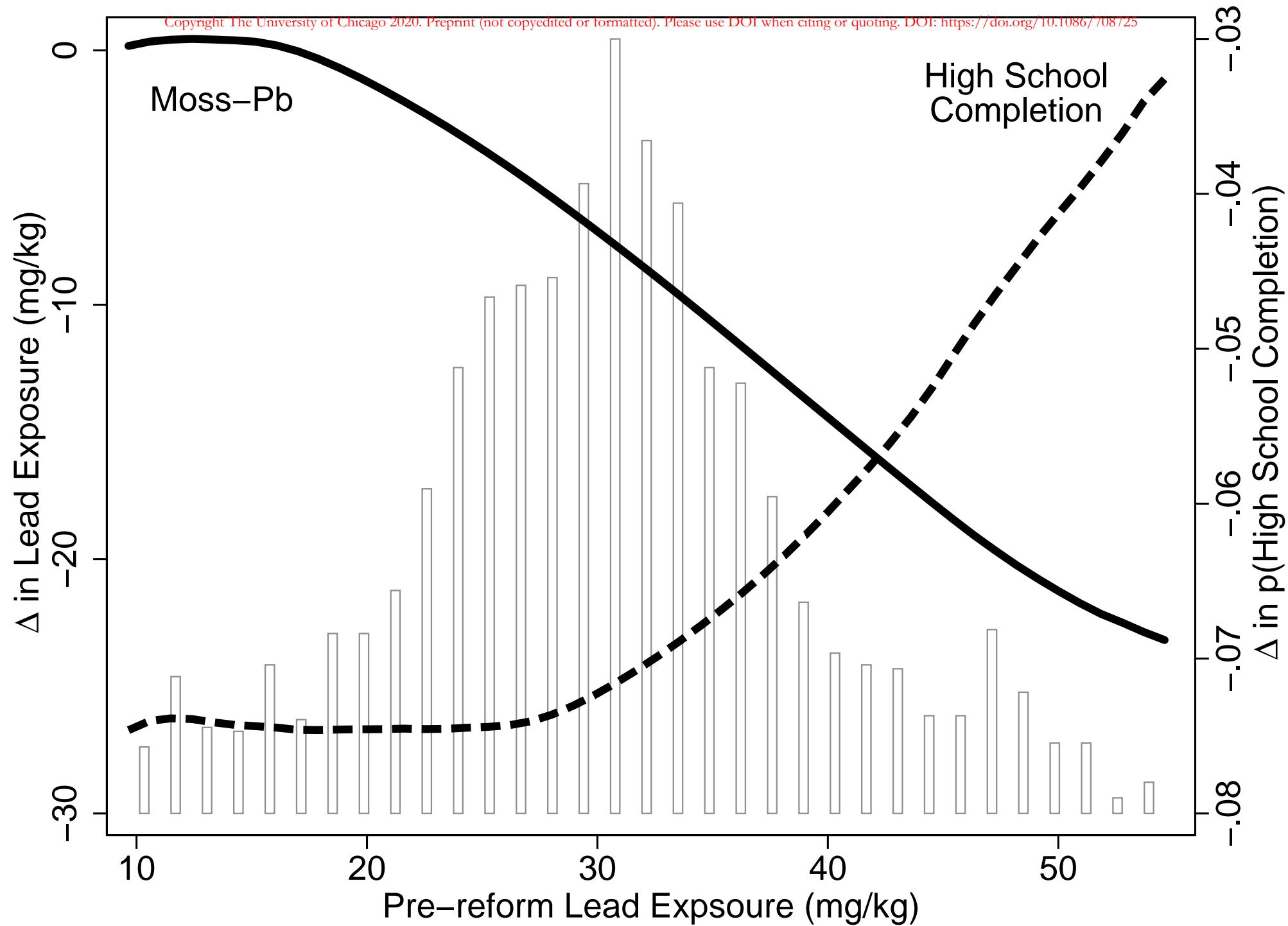
Notes: Semi-parametric fixed effects estimates (see Baltagi and Li 2002) with 95% bootstrapped confidence intervals (1,000 repetitions). The dependent variable in the top graph is P(Ever convicted), in the middle graph P(Property crime) and in the bottom graph P(Violent crime). See Table 4 for the full set of controls.

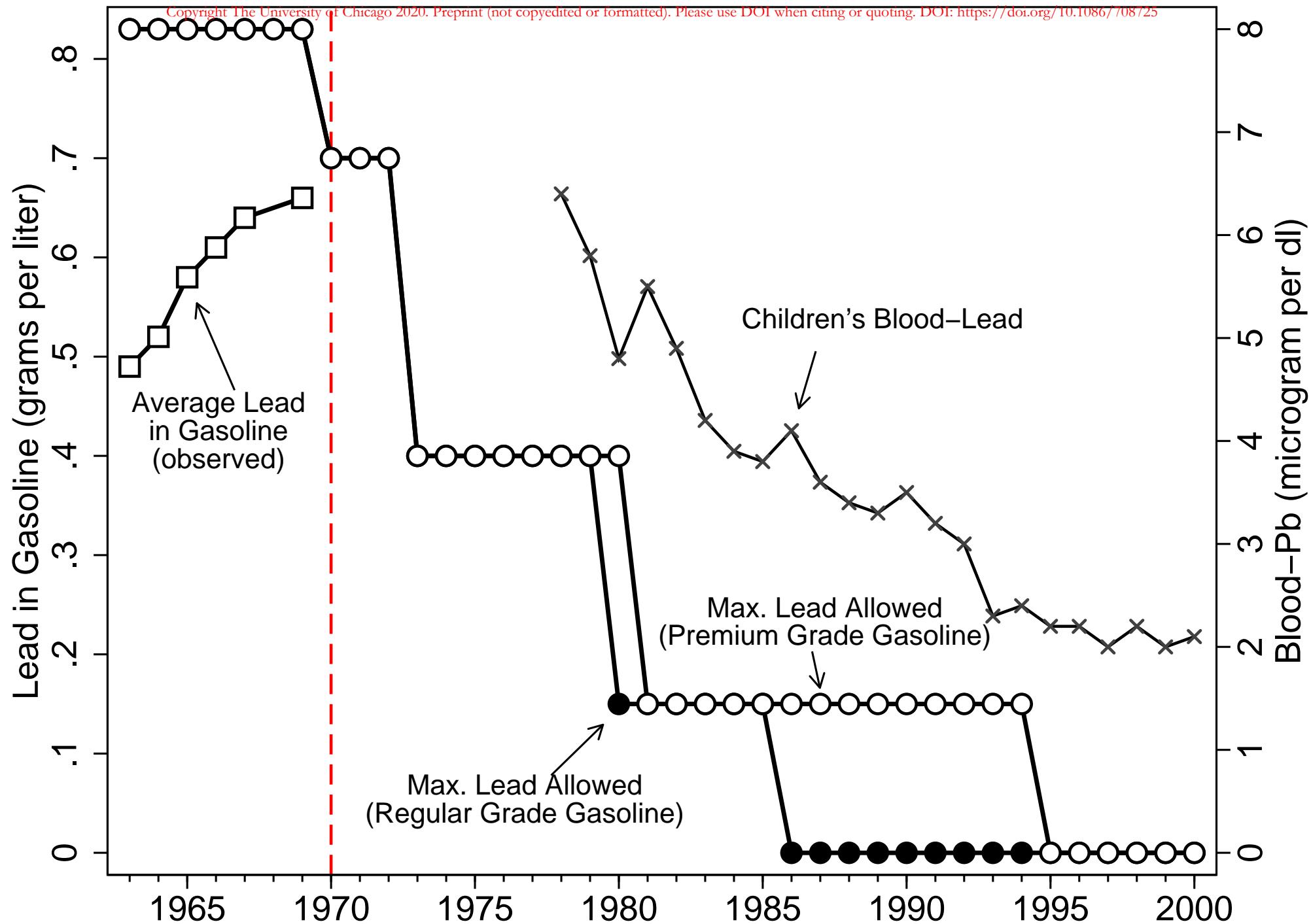
Figure 10. Summary of estimates of the 1980/81 reforms on the long-term outcomes of males

Notes: This figure shows results from four ways of summarizing the effects of the 1980/81 reforms. [Figure A](#) summarizes the results from equation (2). The figure plots the differences in the local-means for the relationship between the cohort-specific effect of the 1980/81 reforms (the parameter γ_c , in equation (2)) against the cohort of birth for the two domains of outcomes. The thick black line shows the differences across neighborhoods above and below the human capital threshold for outcomes predicted to be affected by the reform (* GPA, High school completion, and non-cognitive skills). The thin black line shows the differences across neighborhoods above and below the human capital threshold for outcomes predicted to be unaffected by the reform (** (-)property crime, (-)violent crime, and cognitive skills, rescaled so that positive estimates reflect better outcomes: i.e. *less* crime). Appendix A provides details of how the figure is constructed and reports the full set of results needed to construct it. [Figure B](#) displays point estimates (γ 's) from the DDD version of equation (2) (i.e. the differential impact of an additional unit of pre-reform lead in above threshold neighborhoods vs. below threshold neighborhoods) and shows the reduced form impact of the 1980/81 reforms on an index of the long-term outcomes predicted to be affected by the 1980/81 reforms (GPA, high school, and non-cognitive skills, see the text for details). The index is constructed by first standardizing the outcomes (mean 0, SD 1) and then taking the average of the three standardized outcomes, restricting the sample to individuals for whom all three outcomes are non-missing. The dashed lines represent 90% pointwise confidence intervals. Standard errors are clustered at the CZ and cohort-neighborhood level. [Figure C](#) shows the same estimates as in Figure B after replacing the pre-reform moss-lead level with the actual changes in exposure within neighborhoods, rearranged so that a decrease in exposure is a positive number (i.e. a decrease in exposure is expected to generate a positive effect on outcomes). [Figure D](#) shows the same estimates as in Figure C, but replaces the actual neighborhood change in the exposure variable with a predicted change in neighborhood exposure based on a leave-out estimate of the change in exposure in the neighborhood of birth. The method for constructing the neighborhood-specific predictor of the changes is described in the text.

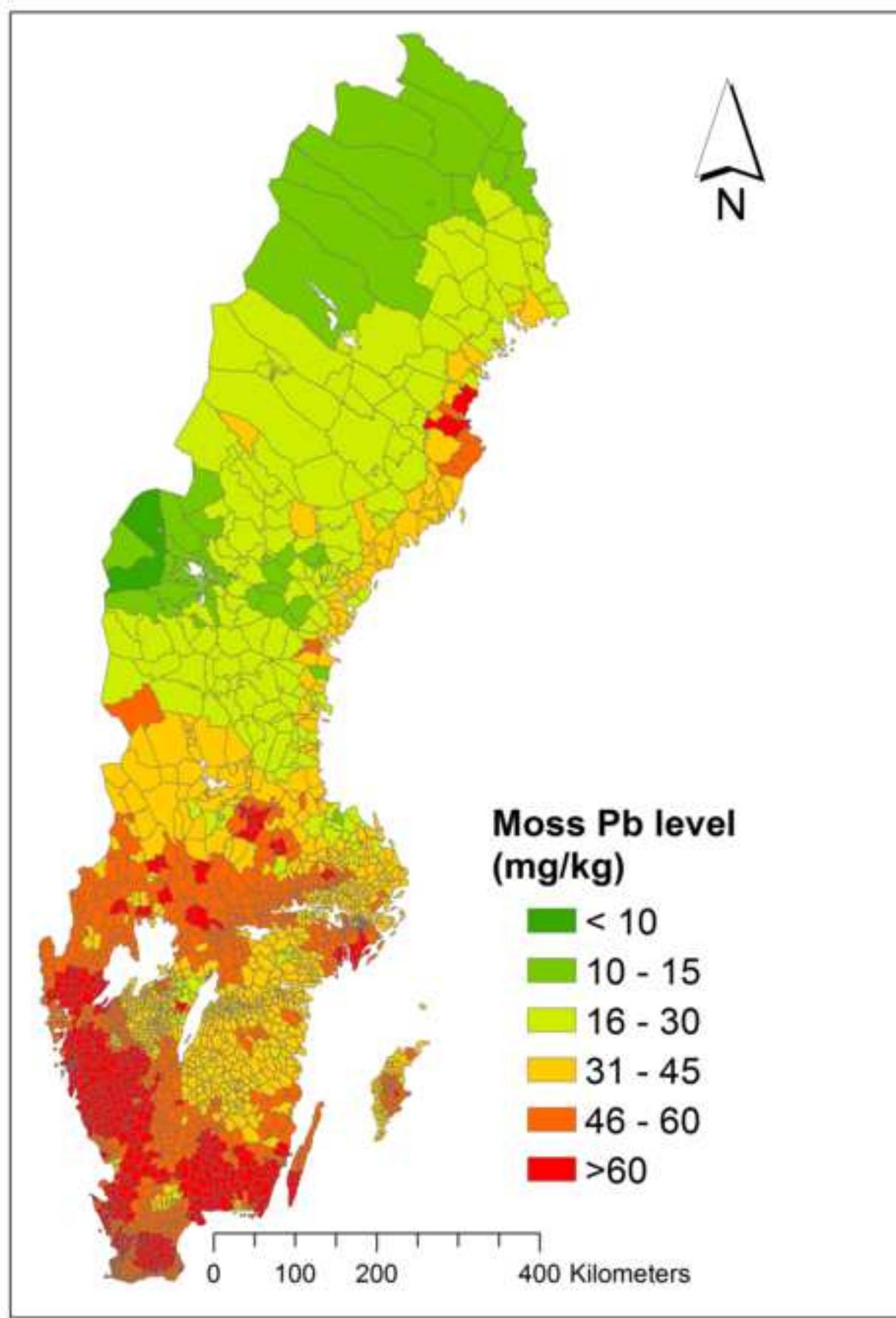
Figure 11. Decomposing the effects of early lead exposure on long-term outcomes by source

Notes: Each bar represents the total treatment effect normalized to 100 percent. The top five bars use the panel data fixed effects model for the full male sample, and the bottom two bars decompose the 2SLS estimates for males in the above threshold neighborhoods. The method and data used to construct the figure are presented in online Appendix F. Following Heckman et al. (2013), we set small and statistically insignificant contributions of the opposite sign to zero. See online Appendix F for information about the simplifications made to produce the figure.* Note that the crime data is not linked to the enlistment data and to construct this figure for the crime outcomes, we use aggregated neighborhood measures of cognitive and non-cognitive skills to calculate the contribution of each skill. For human capital outcomes, we use linked individual data.

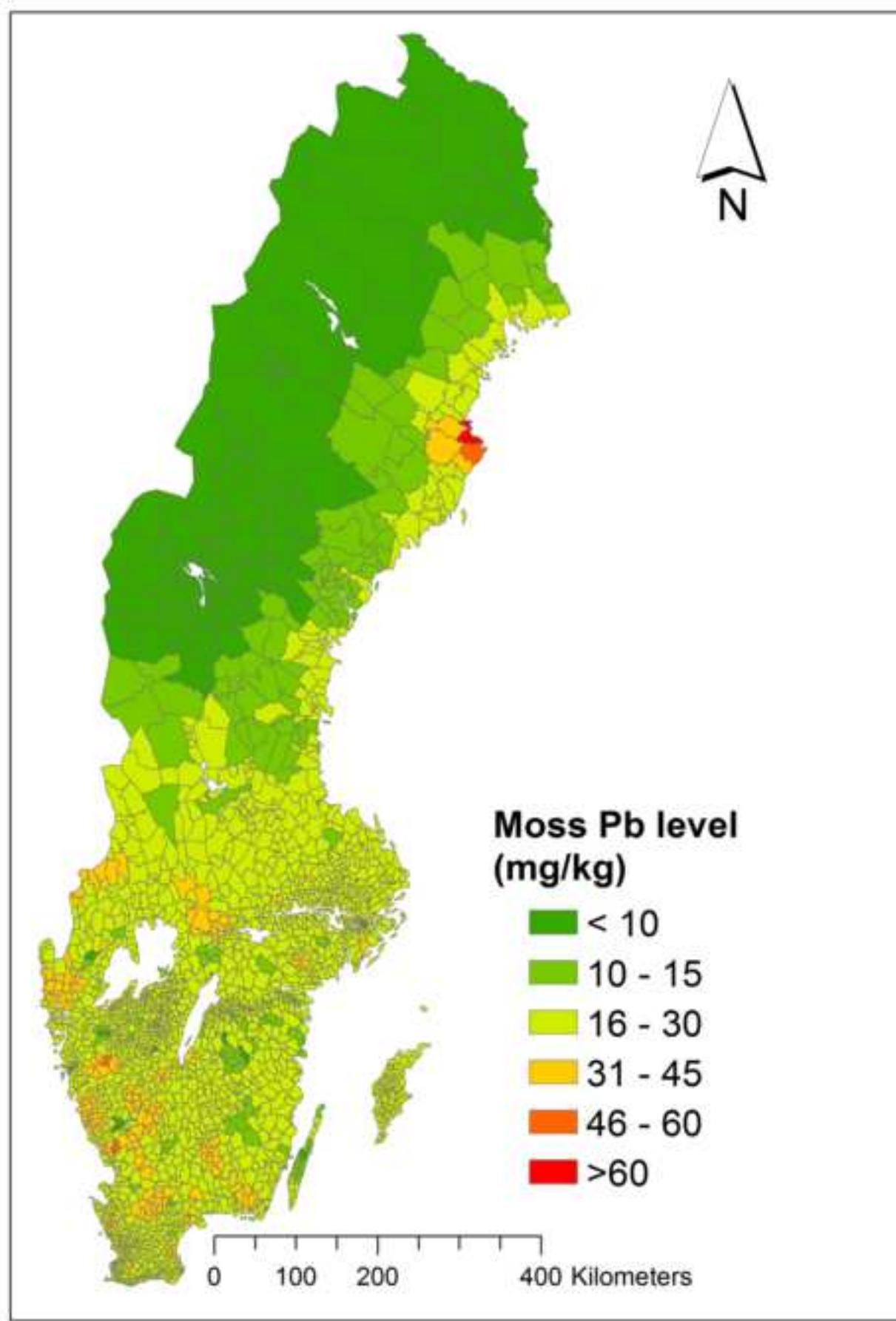




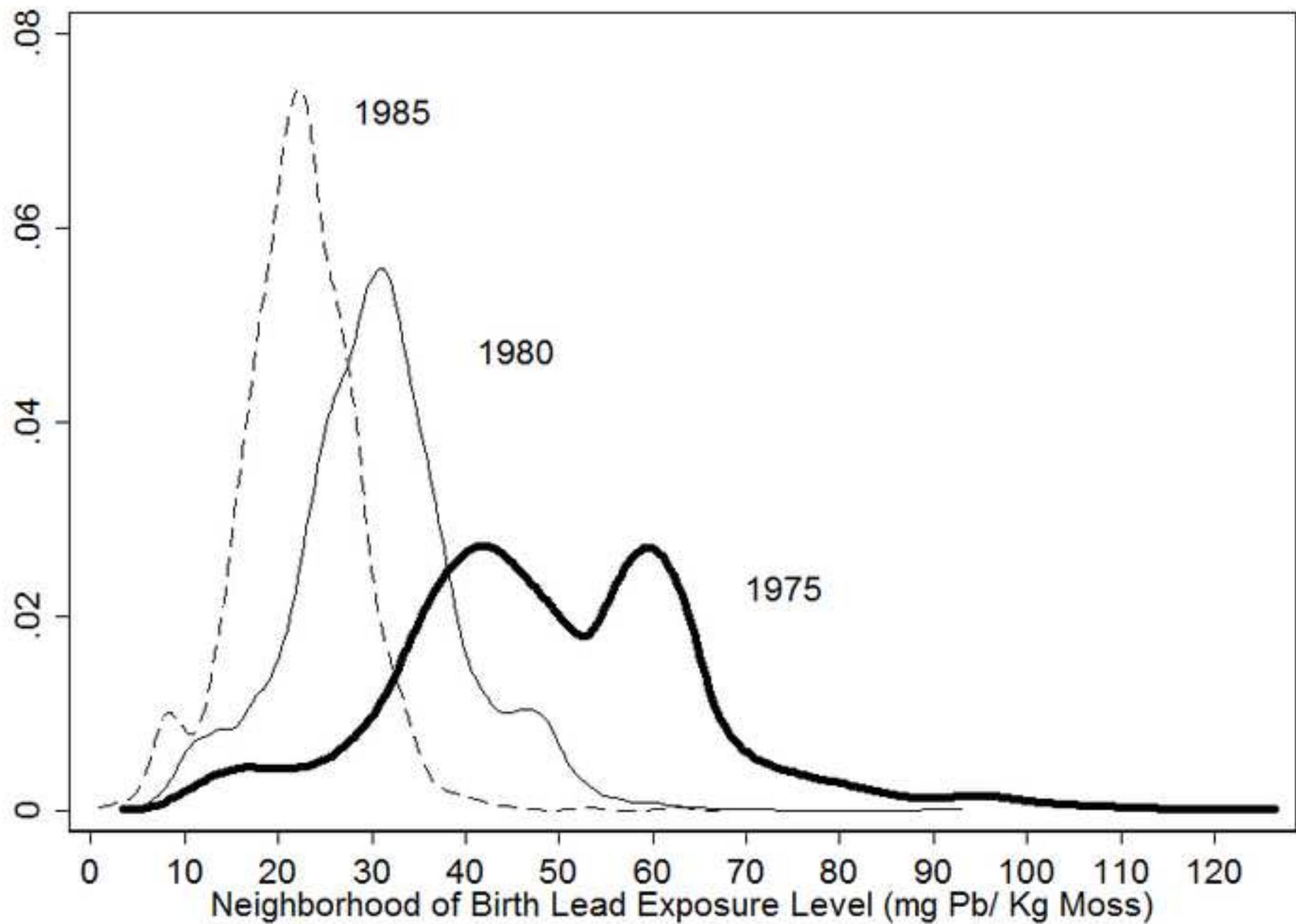
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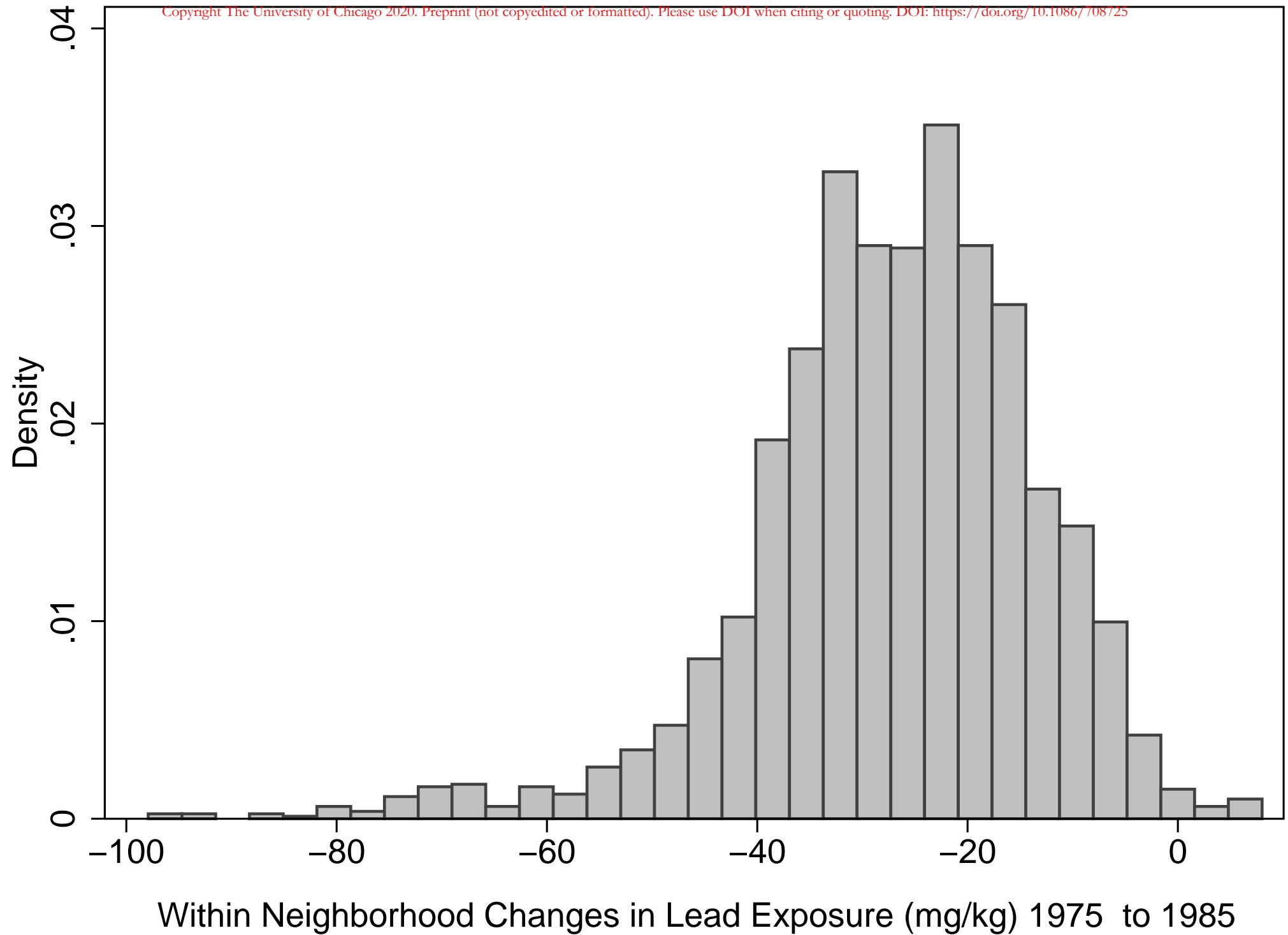


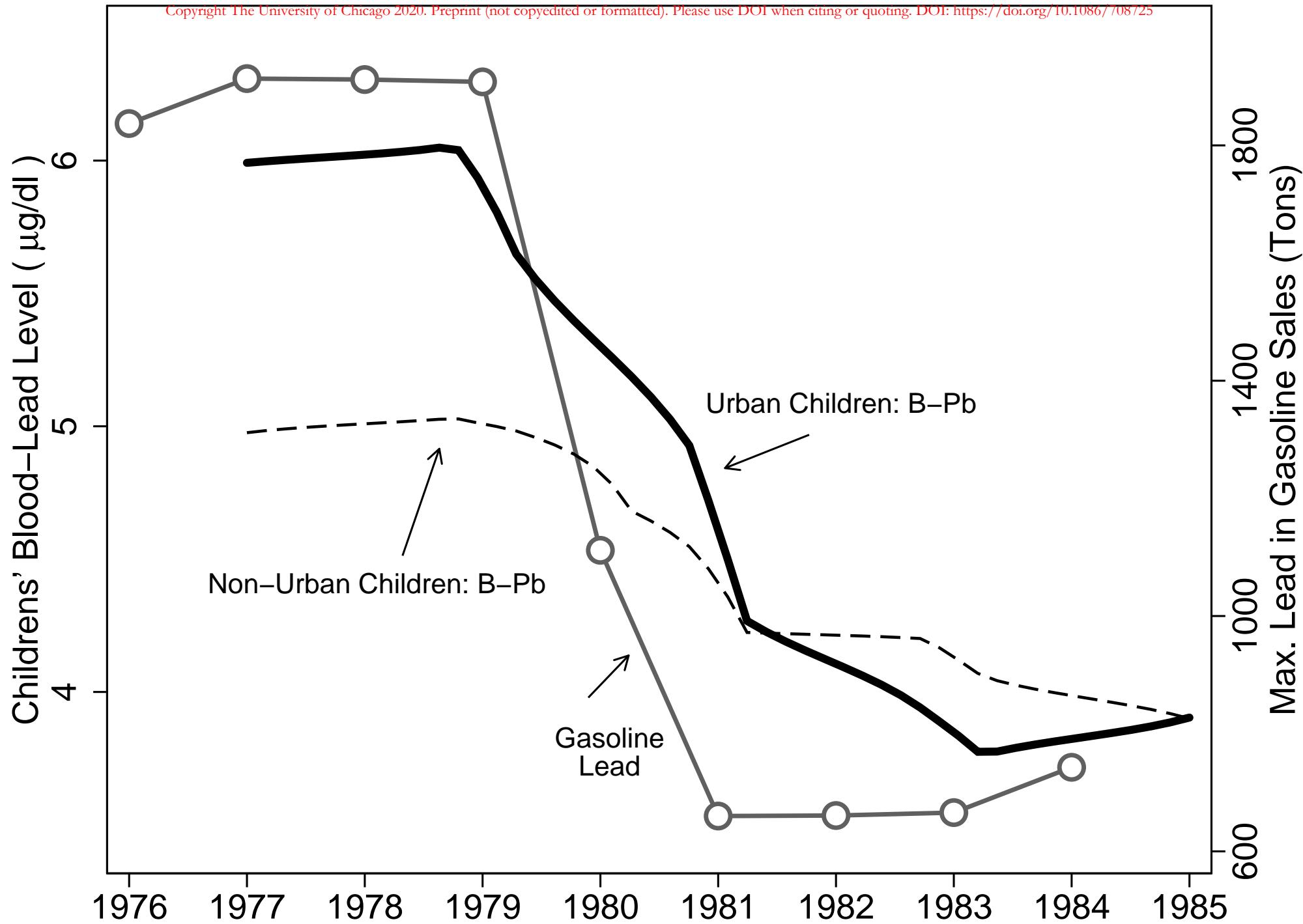
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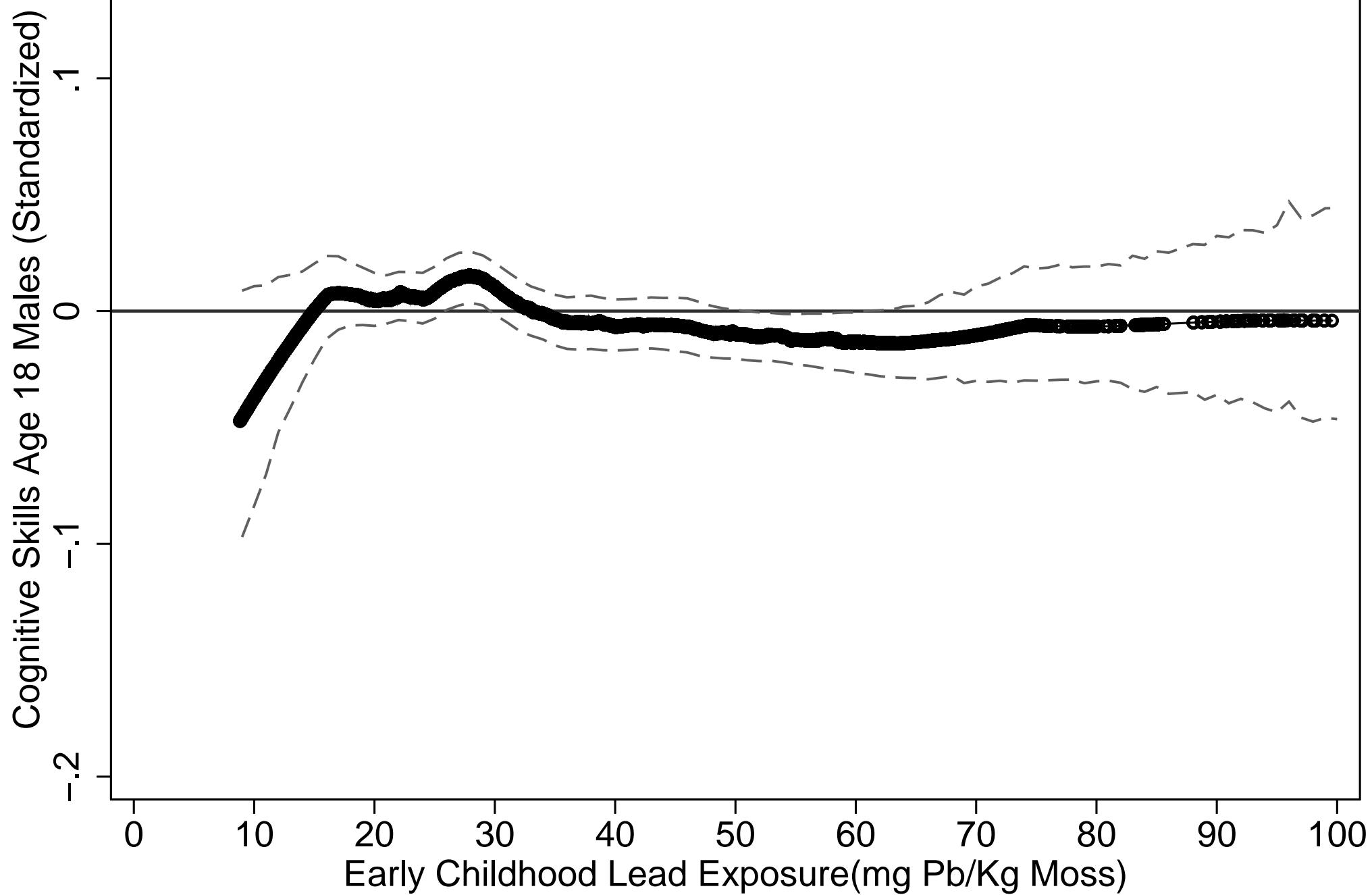
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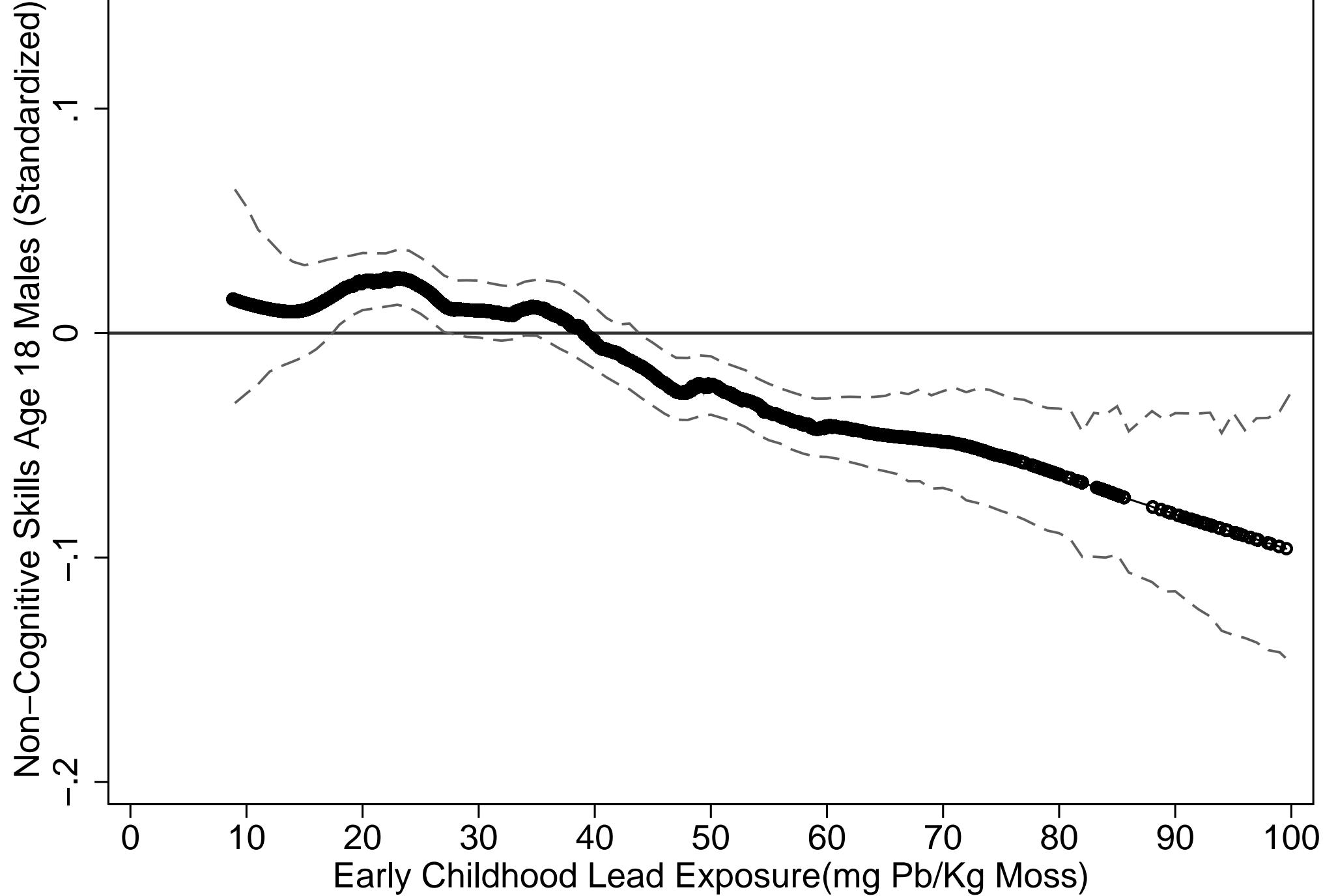


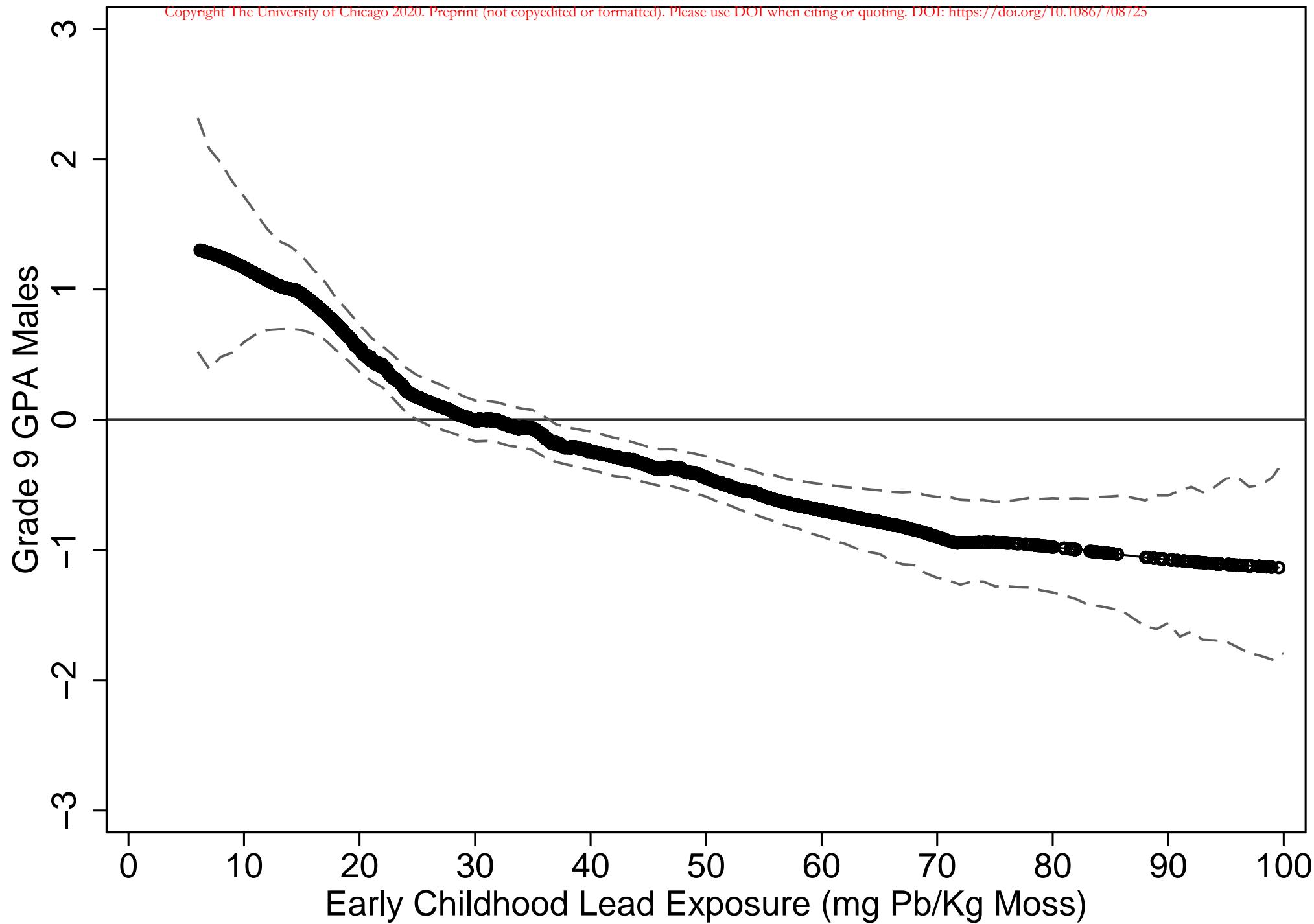


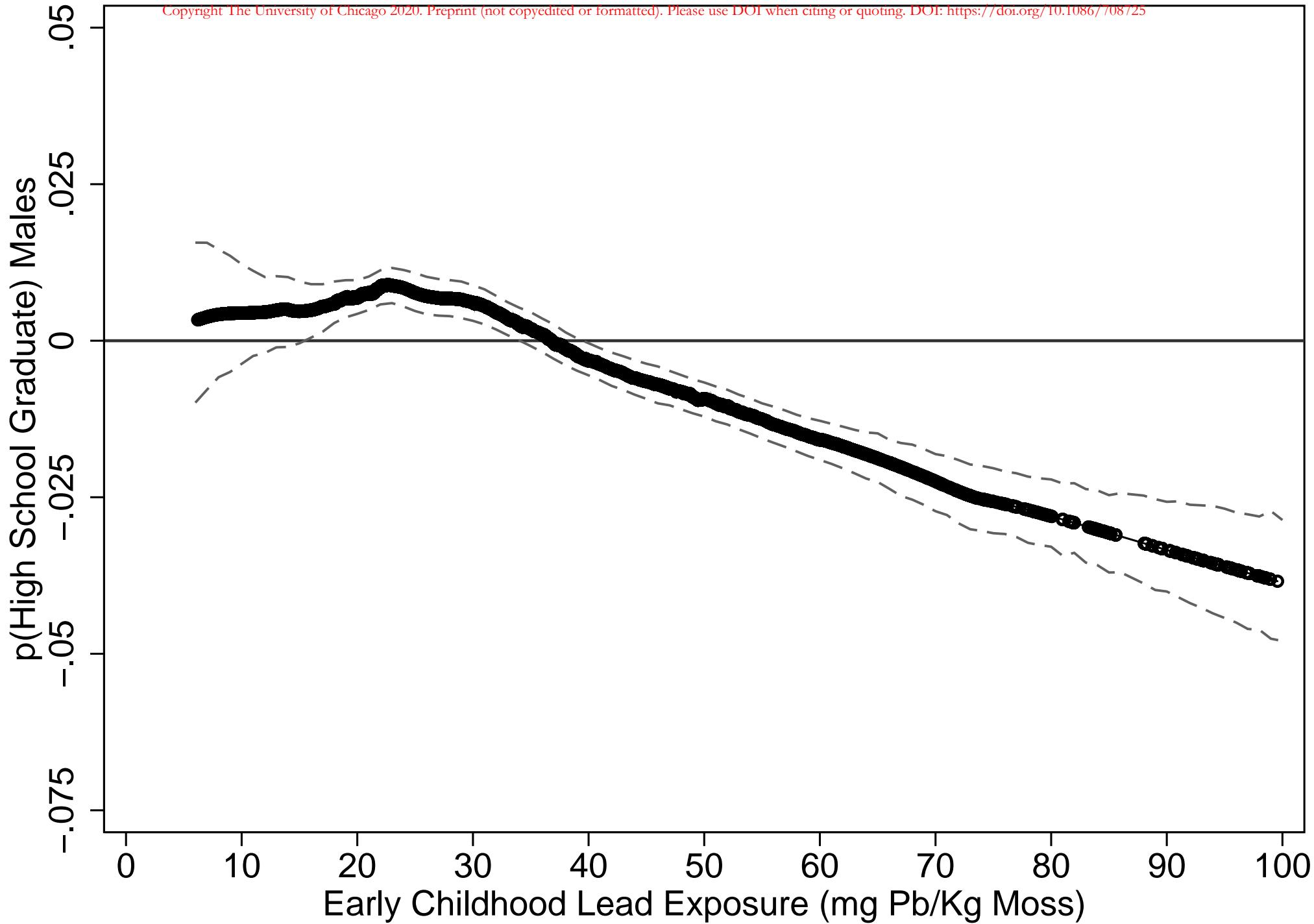
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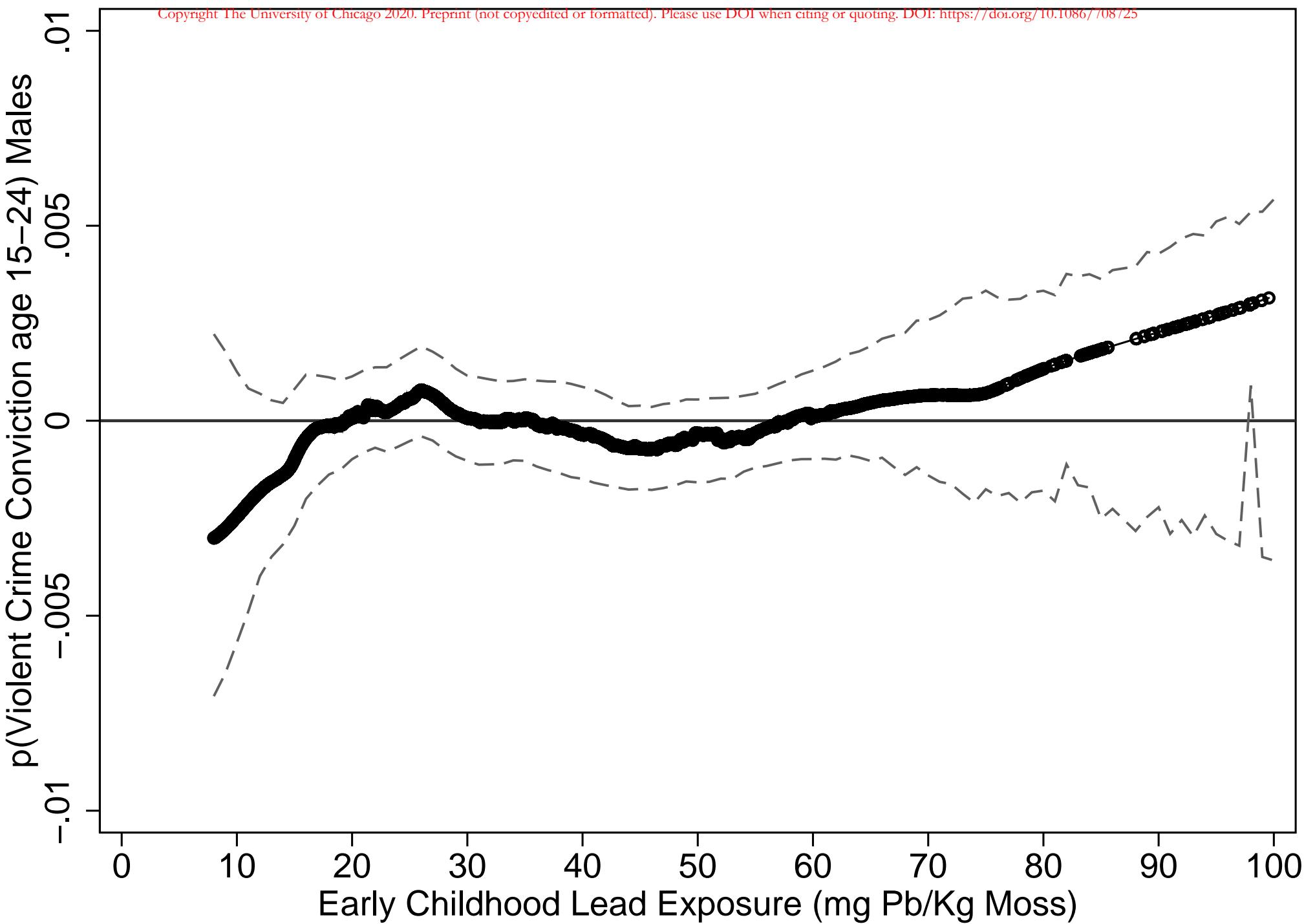


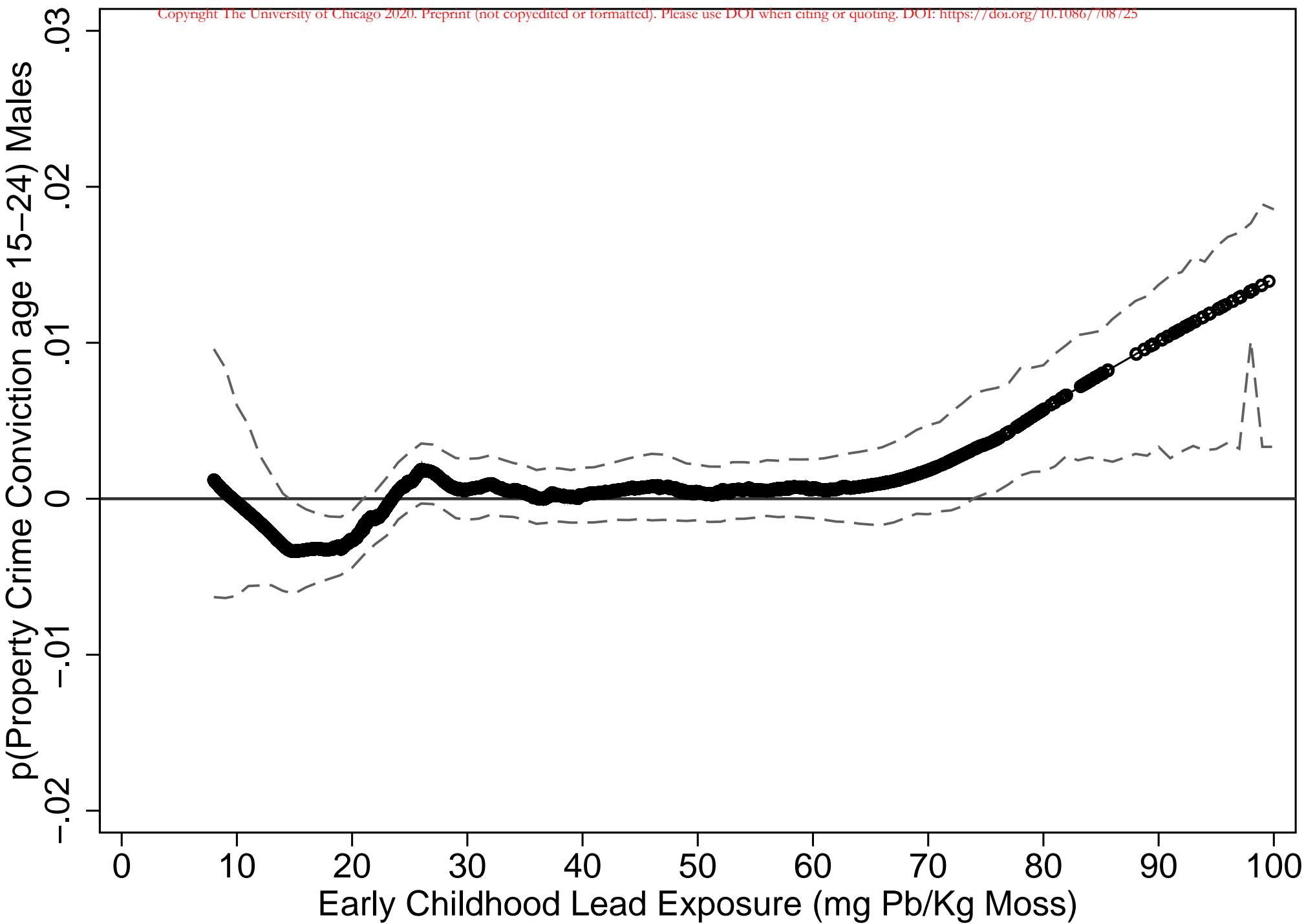
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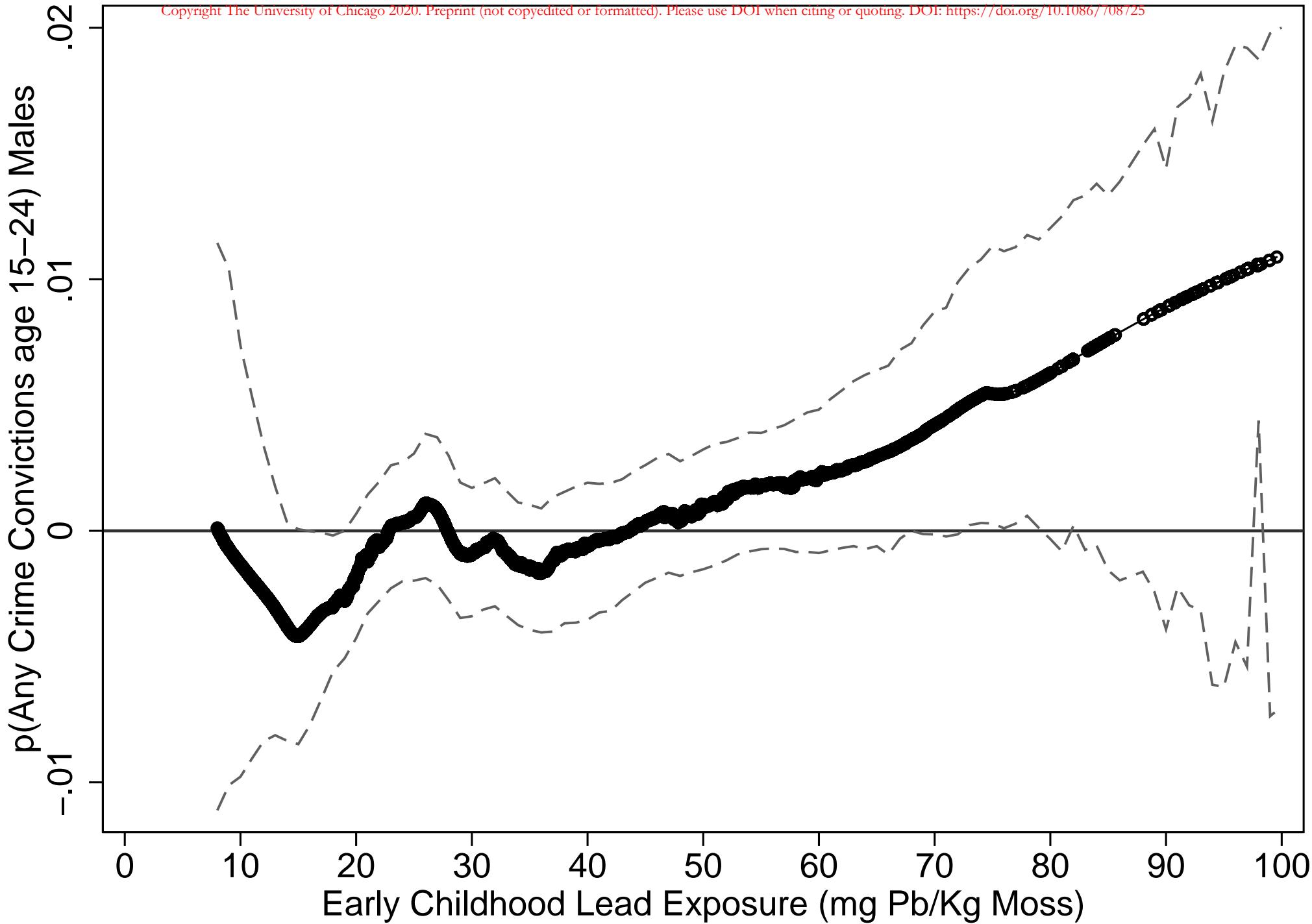


Figure A

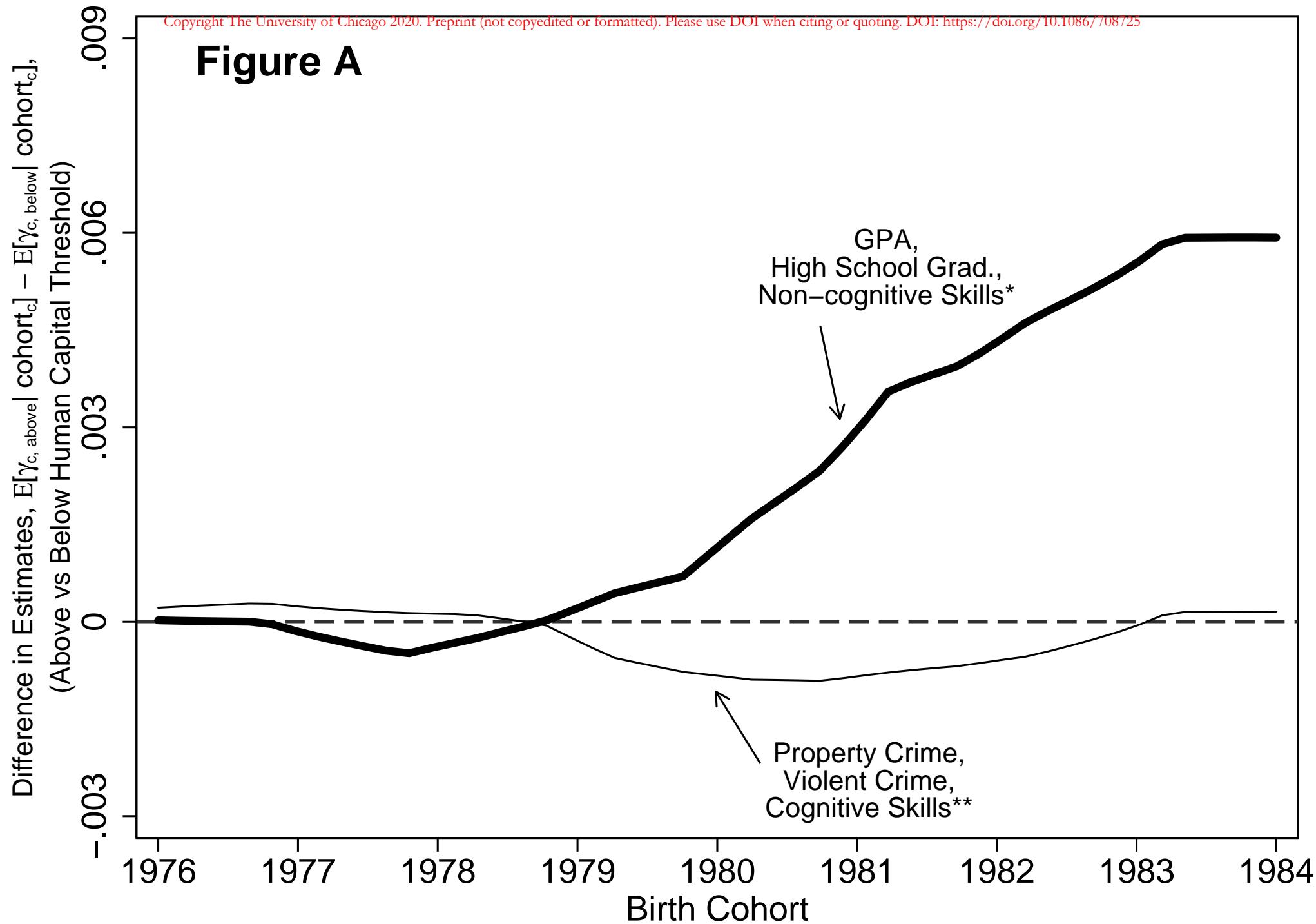


Figure B

DDD-Estimates:
Human Capital Index
(Pre-Reform Level Predictor of Changes)

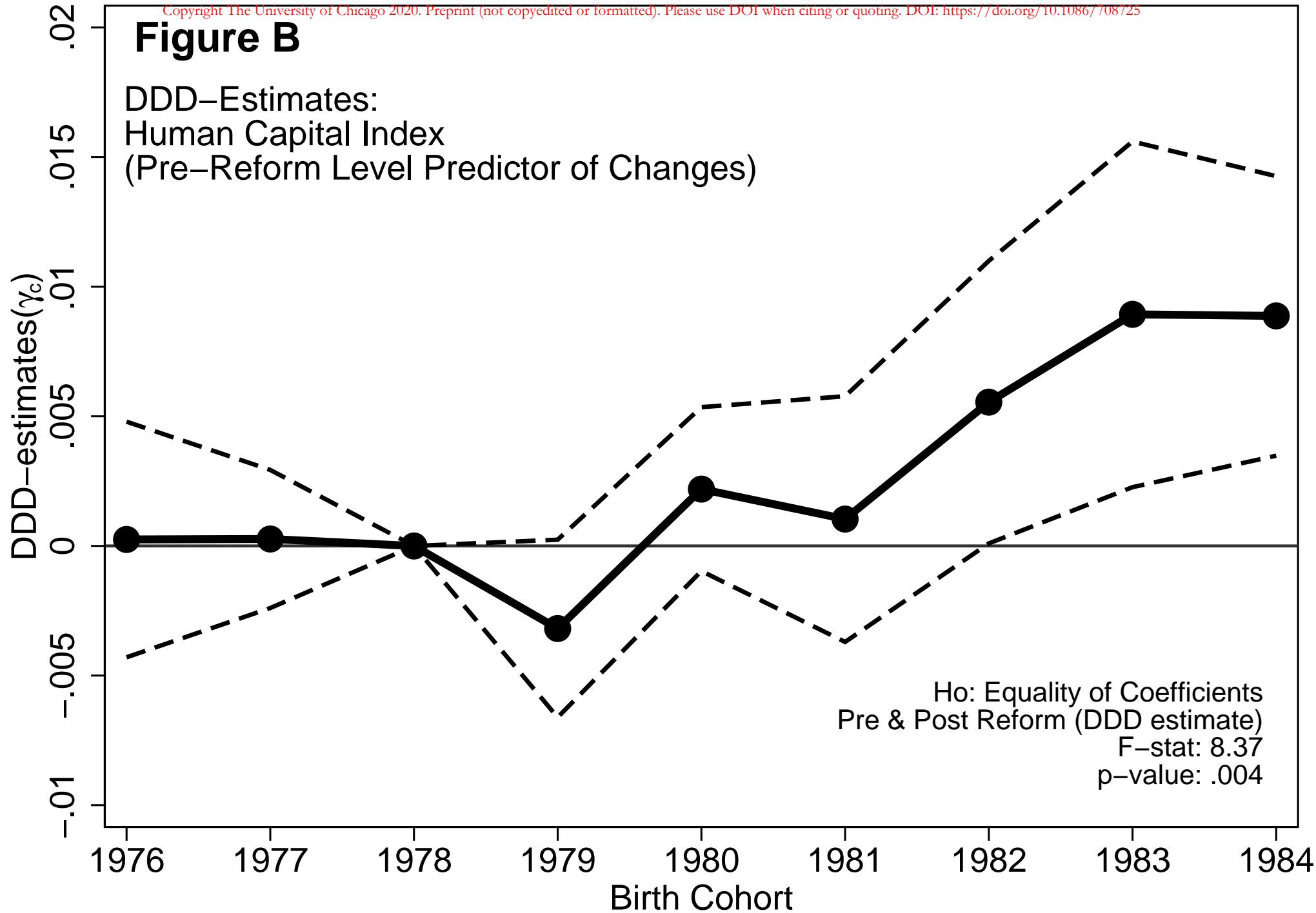
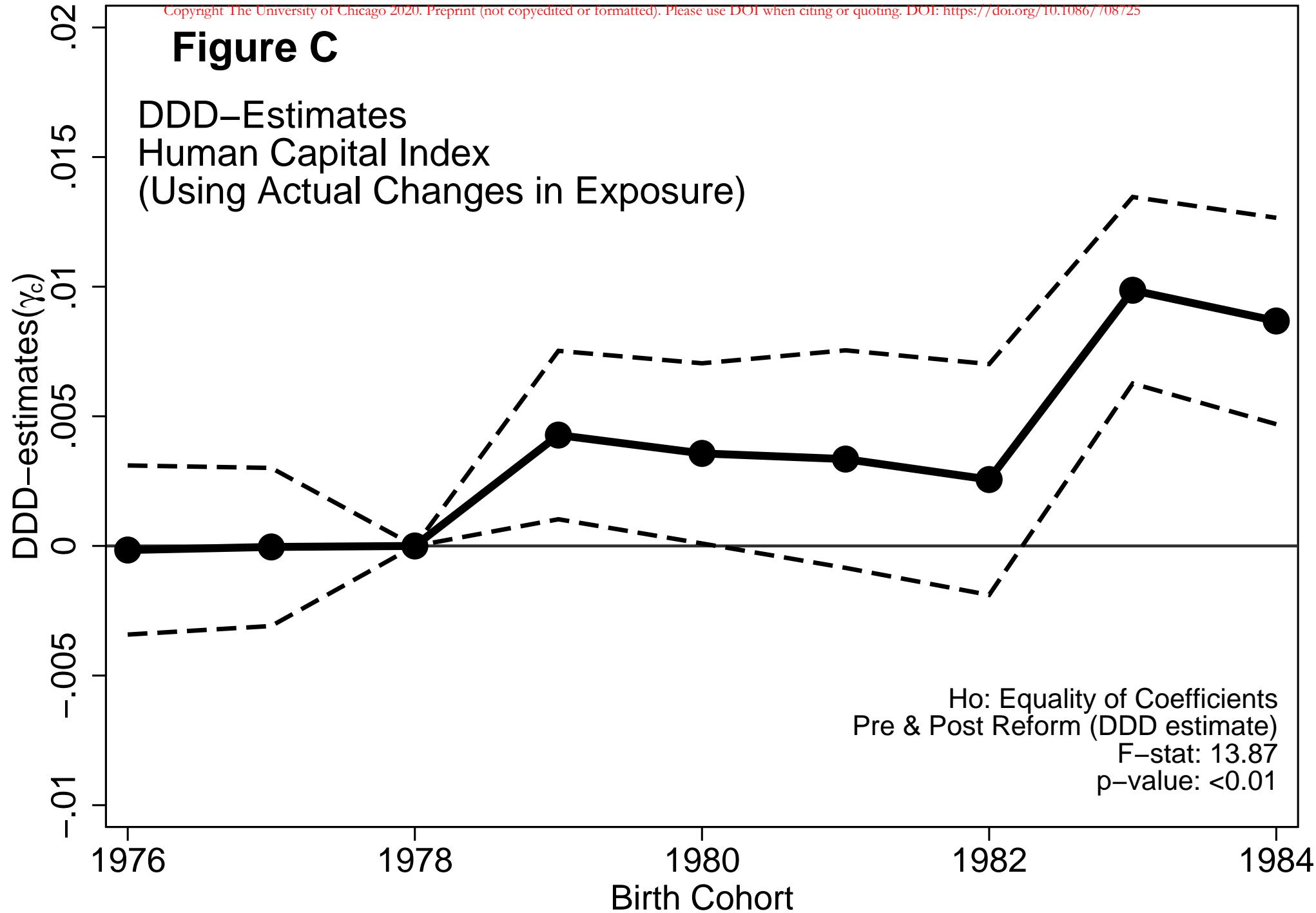


Figure C

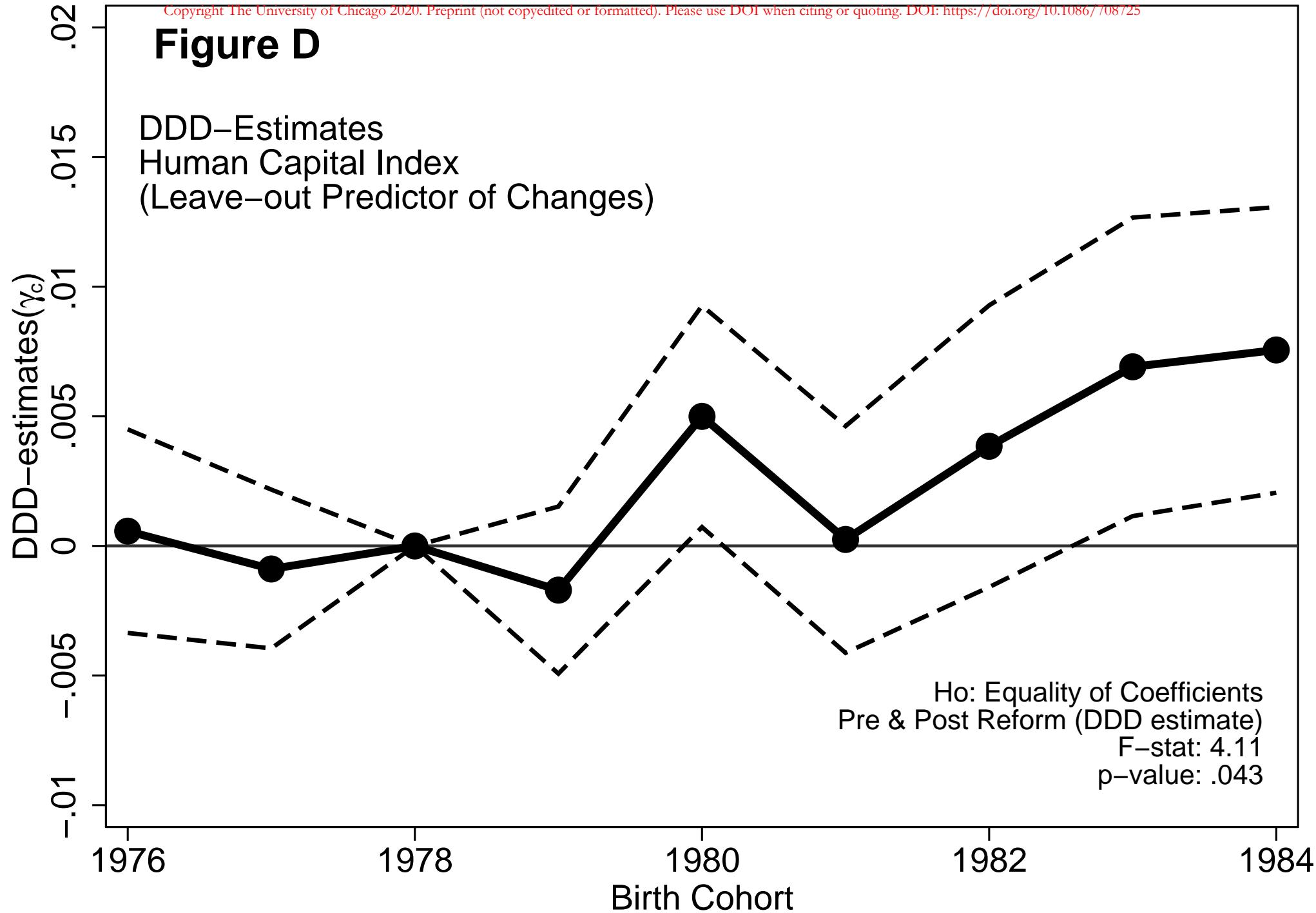
DDD-Estimates
Human Capital Index
(Using Actual Changes in Exposure)

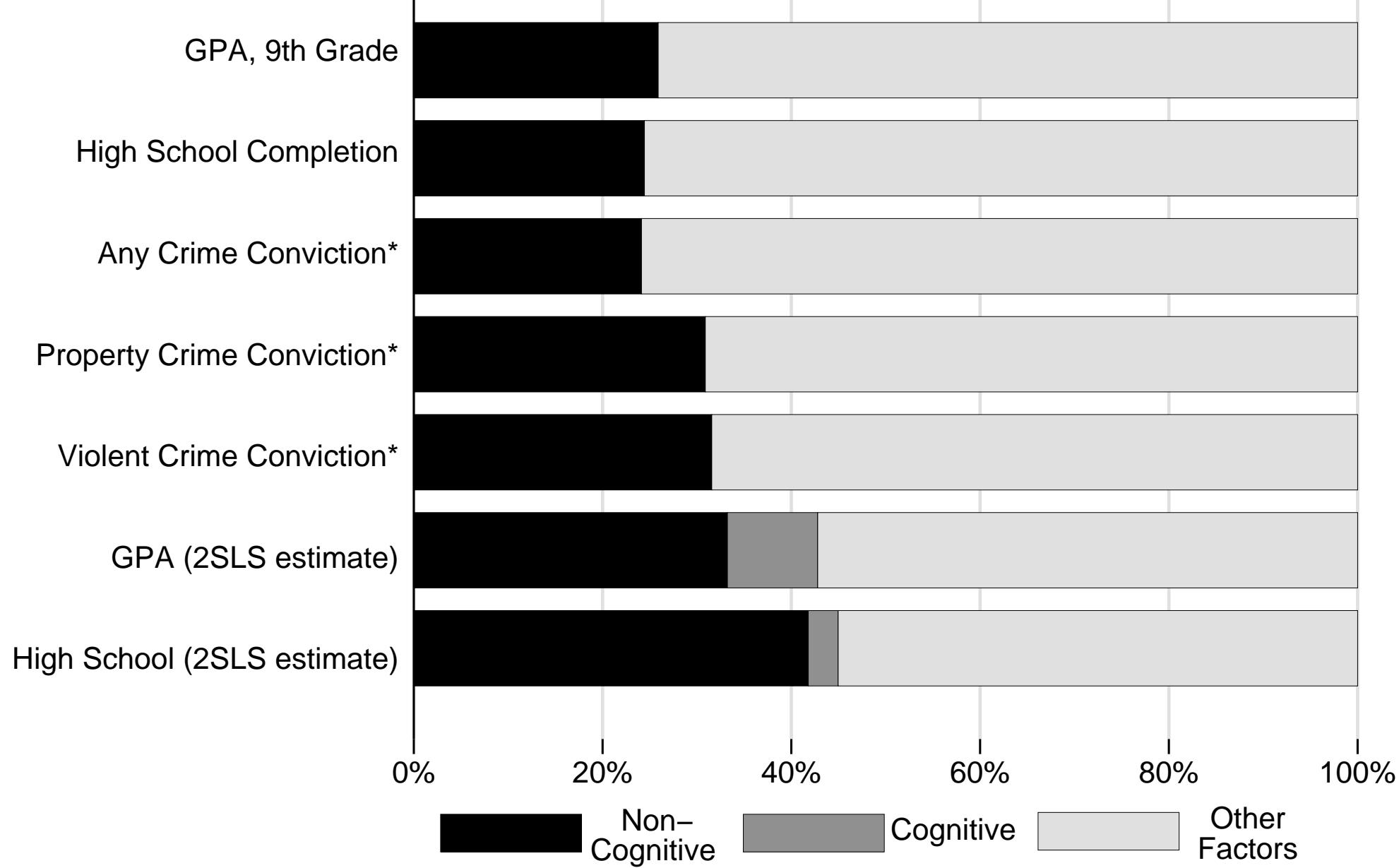


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Figure D

DDD-Estimates
Human Capital Index
(Leave-out Predictor of Changes)





WEB APPENDIX

Understanding How Early Lead Exposure Affects Children's Life-Trajectories

Hans Grönqvist[▼], J Peter Nilsson^{*}, and Per-Olof Robling[▲]

APPENDICES A to F

A: Biological pathways

B: The link between lead exposure and blood lead levels among children

C: Additional results: Panel data analysis

D: Additional results: Reform analysis

E: Additional results: Effects on neonatal health, earnings in 2013, and siblings

F: Decomposing the effects of lead by source

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Web Appendix A:

Biological pathways

A1. How does lead exposure affect development?

Lead is highly toxic to humans, irrespective of how it enters the body, and affects brain development and organ function. Below we review the evidence of the specific mechanisms that affect the development documented in animal studies and outline a conceptual framework, based on the seminal work by Cuhna and Heckman (2007, 2009), which serves to explain how even low levels of early lead exposure may affect outcomes in adulthood.

Biological pathways

After lead has been absorbed from the gastrointestinal tract or the lungs, it enters the blood stream, and is distributed to many tissues and organ systems of the body. The half-life time of lead in blood is around 2-3 months. In small children, around 50 percent of the lead that enters the body via inhalation and ingestion are transported in the blood and are finally deposited in bone. As the child grows, lead stored in bones is remobilizes. 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 properties of calcium, it can pass the blood-brain barrier and affect 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 behavioral problems linked to the prefrontal cortex (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 their 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).¹

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 (see Lidsky and Schneider 2003, for a review). Several epidemiological studies document 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 are done at levels of exposure $<10 \text{ } \mu\text{g/dL}$ (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).

As documented by, for instance, Strömberg et al. (1995) and as we verify empirically in Section 2.2, neither early childhood lead exposure nor take-up differs by gender.² Yet as we show in the paper, early exposure seems to affect boys more than girls. Several recent studies have similarly documented a higher vulnerability in males to adverse prenatal, childhood, family, and neighborhood conditions.³ The direct evidence of gender differences in the effects of early childhood lead exposure is mixed, however. For example, 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 fMRI scans at ages 19-24, 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.⁴ However, as for the more common measures of development, it is unclear whether the gender differences detected by the fMRI scans are due to biological differences in the sensitivity to lead or simply reflect correlated unobservables. Moreover, both Burns et al. and Cecil et al. examine children with high BPb levels ($>10 \text{ } \mu\text{g/dL}$).

¹ Infants and toddlers spend more time on the floor and on the ground than older children, which makes them more likely to ingest lead particles. Xu et al. (2007) show that the child hand-mouth activity decreases with age but detect no gender differences.

² Studies that have investigated gender differences in BPb levels have found that up until around the age of 10, the BPb levels in boys and girls are highly similar (c.f. Strömberg et al. 1995).

³ Nilsson (2017); Chetty et al. (2016); Author et al. (2015); Grönqvist, Niknami and Robling (2016); Golding and Fitzgerald (2016); Garcia, Heckman and Ziff (2017).

⁴ Such non-cognitive traits have previously been linked to e.g., a higher risk of crime (Åkerlund et al. 2016; Heckman Strixrud and Urzua 2006).

Toy model

To better understand how early life lead exposure may influence children's life trajectories, we follow Currie et al. (2014) and Yi et al. (2015) and split the life-cycle into two childhood periods (1 and 2) and adulthood. The production technologies of child skills can be written in the following way

$$(1) \theta_1^k = f_1^k(e_1^H, X, \omega)$$

$$(2) \theta_2^k = f_2^k(\theta_1^k, I_2(\theta_1^k), e_2^H, X, \omega)$$

where θ^k represents skills for $k=\text{Health, Cognitive, Non-cognitive}$; I^k are parental investments in child skills, X are parental and child characteristics; ω are unobserved determinants of skills, such as neighborhood quality; e_t^H is a negative health shock (i.e. lead exposure) in period $t=1,2$ that negatively affects child skills by primarily interfering with the developing central nervous system, i.e. $\frac{\partial \theta^k}{\partial e^H} < 0$.

Adult outcomes are produced through a combination of the different skills, both skills in period 1 and in period 2, with different weights placed on different outcomes so that outcome j is produced by $Y_j = g_j(\theta_1^k, \theta_2^k)$. The total effect of early lead exposure on long-run outcomes can be decomposed into three channels

$$(3) \frac{dY_j}{de_1^H} = \frac{\partial g_j}{\partial \theta_1^k} \frac{\partial \theta_1^k}{\partial e_1^H} + \frac{\partial g_j}{\partial \theta_2^k} \frac{\partial f_2^k}{\partial I_2} \frac{\partial I_2}{\partial \theta_1^k} \frac{\partial \theta_1^k}{\partial e_1^H} + \frac{\partial g_j}{\partial \theta_2^k} \frac{\partial f_2^k}{\partial \theta_1^k} \frac{\partial \theta_1^k}{\partial e_1^H}$$

where the first term on the right-hand side represents the direct biological effect, which operates through the production function. This term is negative when lower skills cause worse outcomes. The second term is a behavioral response that operates through parental investments. The sign of the second term is ambiguous and can vary across different types of skills. The third term captures what Cuhna and Heckman (2007, 2009) denote as self-productivity of skills, which arises when higher stocks of skills in one period create higher stocks of skills in the next period ($\frac{\partial f_2^k}{\partial \theta_1^k} > 0$). In addition, in their model, the stock of skills acquired by the end of the previous period makes investments in the current period more productive.

As discussed above, it is plausible that the lead exposure experienced in period 1 is more harmful due to higher susceptibility, i.e. $\frac{\partial \theta_1^k}{\partial e_1^H} > \frac{\partial \theta_2^k}{\partial e_2^H}$ for $\forall e_1^H = e_2^H$.⁵ Even so, in this dynamic model of skill formation, it is possible that early health shocks are multiplied over the life cycle so that $\frac{\partial \theta_2^k}{\partial e_1^H}$ is large and negative. Moreover, if the technology f_2^k is assumed to be a CES, then one way of allowing for gender differences in the effects of lead is to allow the elasticity of substitution between I_2 and θ_1^k to vary by gender. Early lead exposure could therefore result in greater long-run effects for boys than for girls, even when there are no obvious gender differences in exposure.⁶

Note that our reduced-form estimate $(\frac{dY_j}{de_1^H})$ is an upper (lower) bound of the direct effect if the parents adopt a reinforcing (compensatory) investment strategy, i.e. $\frac{\partial I_2}{\partial \theta_1^k} \leq 0$ ($\frac{\partial I_2}{\partial \theta_1^k} \geq 0$), in the sense that they are investing less (more) in the child as a response to the early health shock.⁷

Empirically, we analyze the effects of early lead exposure on outcomes later in life and on direct measurements of cognitive and non-cognitive skills at the end of childhood. To see how our empirical strategy relates to this model and to better understand how to interpret the estimates, we linearize the production functions (2) as a first-order approximation and add an error term. This gives us the following regression equation:

$$(4) \quad \theta_{2,inc}^k = \alpha^k e_{1,nc}^H + \beta^k X_{nc} + \omega_n + m_{inc}$$

⁵ We find evidence consistent with earlier exposure being more harmful than later exposure, $(dY_j/de_1^H) > (dY_j/de_2^H) = 0$. However, we cannot empirically distinguish whether this “no-effect” of later exposure is due to differences in sensitivity or differences in the take-up of lead over the child’s life-cycle. Based on previous findings, it is likely that both sensitivity and differences in the take-up play a role in explaining this pattern.

⁶ See e.g. Nilsson (2017).

⁷ The strategy that parents employ depends on the parental preferences but also the production technology available to them (c.f. Yi et al. 2015) Early childhood is a sensitive period and the “neurological and behavioral effects of lead are believed to be irreversible” (WHO, 2016). How irreversible the effects of low levels of lead exposure really are remains unclear. A recent study by Billings and Schnepel (2015) finds that some of the effects of lead may be malleable to intervention, even after high levels of exposure. Among rats exposed to the same amount of lead but randomly reared in enriched (large groups) or impoverished (single cage) environments, only those raised in single cages had learning deficits relative to unexposed controls (Schneider et al., 2001). Yi et al. (2015) show that the family acts as a net equalizer in response to early life health shocks across children in China. We do not have any direct measures of parental investment, but to shed some light on the role of responsive investments we provide sibling comparisons estimates which, under certain assumptions (Griliches, 1979), can be informative of the role of responsive investments in accounting for the effects of lead on long-term outcomes.

where i denotes child, n neighborhood, c birth cohort and m_{inc} is the error term. The major methodological challenge is that lead exposure may reflect unobservable characteristics ω_n also predictive of skills. For example, parents may sort so that local lead exposure is correlated with other neighborhood attributes. To net-out neighborhood-specific unobserved heterogeneity, we exploit within-neighborhood changes in early childhood lead exposure induced by the phase-out of leaded gasoline. More formally, it is instructive to think about our empirical strategy as variations in the following within-neighborhood fixed effects estimator

$$(5) \quad \Delta\theta_{2,ic}^k = \alpha^k \Delta e_{1,c}^H + \beta^k \Delta X_c + \Delta m_{ic}$$

where α^k identifies the reduced form effect of lead exposure on child skills, i.e. $(\frac{d\theta_{1,ic}^k}{de_{1,c}^H})$, under the assumption that the within-neighborhood variation in lead exposure is uncorrelated with the error-term.

Web Appendix B:

The link between lead exposure and blood lead levels among children

To what extent is the moss lead level connected to children's blood lead levels? Here we bring evidence to this key question using a range of different samples and specifications. We start by showing the estimated elasticity between MPb levels and BPb levels in children. The following sub-sections present evidence from different validation exercises where we challenge our results using other data sources and research designs. As we will see, the results suggest that the MPb is a strong and robust predictor of children's BPb levels.

B1. The relationship between moss lead and blood lead levels

This sub-section builds on Nilsson, Skerfving, Stroh and Strömberg (2009) who estimate the elasticity between moss-lead levels and blood-lead levels in children. The moss samples were collected at 55 sites each year in the municipality of Landskrona in 1983 and 1995, following the same principles as in the national bio-monitoring program implemented by EPA in 1975 and reflect the average lead levels in the three years prior to the collection date (i.e. in 1980-1982, 1992-1994). We match the MPb data to the (venous) BPb measurements among 242 children aged 7-10 in the year prior to when the moss samples were collected (see Strömberg et al. 2003). 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.⁸ Table B1 reports descriptive statistics for the analysis sample.

Table B2 reports the estimated elasticity between MPb and children's BPb levels using four different versions of the following model

$$(B1) \quad \ln(\text{BPb})_{\text{inc}} = \alpha + \beta \ln(\text{Lead})_{\text{inc}} + \theta X_i + \lambda_n + \lambda_c + \varepsilon_{\text{inc}} .$$

⁸ Following Currie and Neidell (2003), in order to assess the accuracy of the air pollution measure, Nilsson et al. (2009) compare the actual level of pollution at each moss sampling 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 ($r=.73$), suggesting that it is a reasonably 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.

Column (1) shows the estimated elasticity when only controlling for neighborhood (λ_n) and year (λ_c) fixed effects. The coefficient suggests that a 10% increase in the MPb levels raises the average BPb levels by 4.9%. In column (2) we add a vector of individual characteristics (X_i) containing indicator variables for gender, age, lead exposure hobbies, and hemoglobin level. These controls only have a minor impact on the estimated elasticity.

In column (3) we interact female with MPb to test if the take up from air lead levels differs by gender. The main effect is unchanged and the interaction term is not statistically significant. This finding is important since it verifies our prior that the estimated gender differences in the effects of lead exposure on adult outcomes across genders in the main analysis are not likely to be driven by gender differences in the lead take-up. This result is consistent with previous work that finds that the BPb levels in boys and girls are highly similar up until around age 10 (Strömberg et al. 1995).

Non-linearities in the relationship between MPb and children's BPb

In the main analysis, we find that the effect of early lead exposure is non-linear for most outcomes: below certain thresholds (location varies with outcomes) the effect of additional reductions seems to matter much less. This result is consistent with the hockey-stick model predictions of the effects of toxins. However, an alternative hypothesis is that the relationship between long-term outcomes and lead exposure is linear, but that the relationship between MPb and BPb instead follows a hockey-stick shape.

We believe that this alternative explanation is less plausible for two reasons. First, note that the location of the thresholds estimated for the long-term outcomes in the main text varies by outcome. The threshold location is located at a higher level for crime (~50 mg/kg moss) than for human capital outcomes (~30 mg/kg moss). This pattern is inconsistent with the alternative story where the non-linear relationship between moss-lead and long-term outcomes is due to the fact that the relationship between MPb and BPb vanishes at a specific point in the MPb distribution.

Second, we can test this alternative hypothesis directly by replacing the single lead exposure measure in equation (B1) with linear splines in MPb at the estimated crime threshold 50 mg/kg (49% of sample below). The estimated elasticity above vs. below the threshold is indistinguishable (column 4). Since only 20% in the Landskrona sample experienced a moss-lead level below 30mg, the precision of the estimated slope at the lower threshold (column 5) is poor,

and while the point estimate is somewhat smaller, it is not statistically distinguishable from the above threshold estimate or the full sample linear estimates.

Overall, these results do not provide any strong support for the alternative hypothesis.

Table B1. Descriptive statistics for blood-lead sample (Landskrona municipality)

Variable	Mean	Std.dev
Blood-lead ($\mu\text{g}/\text{dL}$)	3.6	1.7
Moss-lead (mg/kg)	87	53
Hemoglobin (g/L)	131	7
Share with lead exposure hobby	.10	
Age	9.12	.68
Share Female	.49	
Distance to motorway E20 (km)	.87	.60
Distance to smelter (km)	4.88	3.75

Notes: The table presents descriptive statistics for the sample used in the estimation of the relationship between BPb and MPb (n=242).

Table B2. Lead exposure and children's BPb level

The relationship between MPb and children's BPb (*Landskrona*)

<i>Dependent variable:</i>	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)
<i>Specification:</i>	(1)	(2)	(3)	(4)	(5)
ln(MPb)	.4861*** (.1190)	.4774*** (.1177)	.4957*** (.1196)		
ln(MPb) × Girl			-.0555 (.0635)		
Spline below crime threshold (49% of sample below)				.4731*** (.289)	
Spline above crime threshold (51% of sample above)				.4796*** (.123)	
Spline below human capital threshold (20 % of sample below)					.4223 (.289)
Spline above human capital threshold (80 % of sample above)					.486*** (.118)
Neighborhood FE	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes
Individual controls	No	Yes	Yes	Yes	Yes
R-squared	0.56	0.58	0.58	0.58	0.58
No of children	242	242	242	242	242

Notes: The table reports OLS estimates based on equation (B1). Blood lead (BPb) level is measured in (ln) µg/dL blood and moss lead (MPb) exposure is in (ln) mg/kg of moss assigned to children's residential address in Landskrona municipality. The full set of individual controls include gender, age dummies, whether the child is practicing any lead exposing hobbies, and the ln(hemoglobin) level. Robust standard errors in parenthesis. */**/*** denote significance at the 10/5/1 percent levels, respectively.

Additional results: moss-lead and local lead pollution sources

The relatively high MPb levels observed in Landskrona are due to the fact that one of Sweden's two secondary lead smelters was in operation in Landskrona during the observation period. A benefit of this is that it generates a great deal of variation in lead exposure across the children in Landskrona. A potential concern is whether air lead emissions from the smelter have a differential impact on BPb levels as compared to lead emissions from vehicles. In this case, it may be difficult to generalize the results to other parts of Sweden without lead emitting industries.

While diffusion differs depending on the emission source, we have found no studies indicating a difference in the lead take up when released into the air from industries or vehicles. However, in Table B3 we address this issue directly by controlling for the distance to the smelter. We geocoded the location of the smelter stack, and re-estimated equation (B1) after including: the linear and quadratic distance to the smelter variables (column 2), and the inverse of the distance to the smelter (column 3). Column (4) shows our preferred way of accounting for the influence of the smelter, where we include linear splines in distance with knots at 5 km from the smelter. This specification is motivated by the negative correlation between moss-lead and the distance to the smelter up to 5 km and the flat relationship thereafter (see discussion below). None of these specifications changes the elasticity between BPb and MPb significantly. If anything adding these additional controls increases the point estimate. The model in column (4) is our preferred specification when relating MPb to BPb in Sweden in general, since very few communities are directly affected by lead emitting heavy industries.

Column (5) shows the relationship between children's BPb levels and the distance to the smelter. Consistent with the relationship between the moss-lead level and the distance to the smelter, there is a significant and negative relationship between children's blood-lead level and the distance to the smelter within 5 km from the smelter, and insignificant thereafter.

The second largest local source of lead exposure in Landskrona is the Freeway European Route E20 which cuts through the municipality, and runs tangent to the two largest communities. Plotting the residuals from a regression of the moss-lead level on the distance to smelter splines and year effects reveals a piecewise-linear pattern in MPb and distance to the E20 with a breakpoint at 1.5 km from the highway (flat and then decreasing). Consistent with this pattern, children's BPb levels and the distance to the E20 are insignificant below 1.5 km, and significant

and decreasing above 1.5 km (column 6). A potential explanation for the flat and then decreasing pattern with respect to the distance to E20 is that the distance measure captures lead exposure not only from E20 but also from busy access roads to the E20.⁹

Column (7) shows the relationship between the distance splines and children's blood-lead levels when the distance splines are included together. While the estimate for the distance to E20 becomes smaller, the point estimates for both the distance to the smelter and the distance to E20 remain sizable. However, as is shown in column (8), when we add the moss-lead measurements, the point estimates for the distance splines for both the smelter and the E20 all become insignificant, and close to zero. The estimates in column (8) show that the moss-lead level data accounts for all information contained in the distance to major local pollution sources variables that is relevant for predicting children's BPb levels. This finding is interesting and important as it suggests that the distance measures are not simply capturing differences in socioeconomic conditions correlated with the distance to these disamenities (e.g. due to sorting based on house prices) and BPb levels.

⁹ Ideally, we would have used information on the traffic density for each road in the municipality during the observation period to weight the influence of all road segments in the analysis, but the data was not available. See also section B2 below using data from Stockholm, where the traffic density on major roads is known. Here the correlation is more in line with our priors about the diffusion patterns of lead from major roads.

Table B3. Additional results: moss-lead and local lead pollution sources

<u>The Relationship between MPb and Children's BPb (<i>Landskrona</i>)</u>								
<i>Dependent variable:</i>	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)	ln (BPb)
<i>Specification:</i>	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
ln(MPb)	.4774*** (.1177)	.5917*** (.2017)	.5749*** (.1559)	.5652** (.2192)				.5859*** (.2705)
Spline in distance to smelter (<5 km)					-.1247** (.0543)		-.1305* (.0688)	.0266 (.1021)
Spline in distance to smelter (>5 km)					.0198 (.0585)		.0215 (.0663)	.0169 (.0604)
Spline in distance to E20 (<1.5 km)						.0302 (.0639)	.0724 (.0669)	.0122 (.0691)
Spline in distance to E20 (>1.5 km)						-.2324** (.1119)	-.1247 (.1165)	.0511 (.1315)
Linear & quadratic in dist. to smelter	yes							
Inverse of distance to smelter		yes						
Splines in distance to smelter (5 km)			yes					

Notes: All specifications include year and neighborhood fixed effects, and individual controls as specified in Table A2. Column (1) reiterates the results from column (2) in Table A2. Columns 2-4 add alternative controls for distance (km) from residence to smelter. Columns 5 to 7 show estimates between blood-lead and linear splines in distance to the smelter and E20. Column (8) shows that all predictive information for children's BPb levels contained in these splines disappears when we add the moss-lead measurements. Robust standard errors in parenthesis. */**/** denote statistical significance at 10/5/1 percent levels, respectively.

B2. Validation using observed blood-lead levels in pre-school age children in Stockholm

Here we ask if our results for the link between moss lead exposure and blood lead levels among children also hold when studying even younger children living in other Swedish regions. We do this by providing evidence from two independent datasets that cover other cohorts and region. This exercise is relevant because, as discussed in Section 2.1, both exposure and uptake is higher for younger children and exposure also differs across areas depending on pre-existing differences in population size and traffic density. To preview our findings, the model developed in section B1 give an accurate prediction of average blood-lead levels also among preschoolers, even in Sweden's most densely populated city center. This important result gives us confidence in using the moss-lead data to estimate the impact of early childhood lead exposure on children's long-term outcomes. Below we discuss our findings in more detail.

BPb levels in Central Stockholm pre-schoolers in 1979

As a validation exercise we were able to collect data on the BPb measurements of 101 pre-school children measured in April 1979 in Central Stockholm (Hanno et al. 1979). A key benefit with this data is that the sample even includes younger children living in a metropolitan area. The effective response rate in this sample is 77% (131 was asked, 114 agreed, 101 were sampled).

The geometric mean blood-lead level in this sample of 3 to 5 year olds attending 21 different pre-schools in downtown Stockholm is 6.0 µg/dL. However, the survey slightly oversampled children residing close to busy roads (see Figure B1). To get an estimate of the average BPb level among all children in Stockholm, we use residence of birth coordinates (centroid of a 250m*250m grid) of all children born in Central Stockholm in 1977 and calculate the distance from the residence to the closest primary or secondary road. In Central Stockholm, primary and secondary roads (henceforth major roads) correspond well with traffic flow maps from 1977 showing road segments with more than 15 000 vehicles/24hrs.¹⁰ Figure B1 shows that the negative correlation between children's BPb levels and the distance to the nearest major road flattens out after approximately 275 meters. To predict the BPb levels of all children in Central Stockholm, we estimated a linear spline model allowing for a

¹⁰ See SNV (1977).

change in slopes at 275 meters using the individual BPb data.¹¹ Using this model, we predict the BPb level in the full population of Stockholm preschool age children to be 6.0 µg/dL.^{12 13}

To validate the use of the moss data, we proceed by predicting the BPb levels in preschool children in Central Stockholm using the observed MPb levels in Central Stockholm neighborhoods in 1977-1979 and the age-adjusted MPb-BPb estimate presented in Table B3 column (4) above. The observed average moss-lead level in Central Stockholm and the model in Table B3 column (4) evaluated at the mean of the explanatory variables give a precise estimate geometric mean BPb level of Central Stockholm pre-school age children of 5.6 µg/dL (i.e. a prediction error of 0.4 µg/dL).

BPb levels among pre-school children in Stockholm in 1991 and 1992

Berglund et al. (1994) report average blood-lead levels from two additional samples of pre-school age children in the southern part of Central Stockholm (Södermalm, BPb=3.0 µg/dL, ages 1-3, n=90, effective response rate 23%) and in a suburb of Stockholm (Sundbyberg, BPb=2.7 µg/dL, at the 18-month check-up, n=40, effective response rate 38%) measured in 1992 and 1991, respectively. Since these two BPb measurements were sampled in-between two moss sampling rounds, we use the average moss-lead level of the 1990 and the 1995 moss sampling rounds to predict pre-school children's blood-lead levels in the two locations. The moss-lead prediction of the children's blood-lead levels is 2.6 µg/dL for the Sundbyberg children, and 2.6 µg/dL for the Södermalm children, yielding a prediction error of 0.1 and 0.4 µg/dL, respectively.

Considering the several layers of measurement problems for the MPb-BPb predictions, stemming from sources such as the aggregation of moss observations to neighborhoods, linking neighborhood lead to actual exposure, and age adjustments, we consider a prediction error of only 0.4 µg/dL as reassuring. The difference is comparable with the average within-lab standard deviation from tests on reference materials reported in Parsons et al. (2001).

¹¹ We also tested if adding controls for average maternal age and parental income at the grid level and neighborhood fixed effects change the distance estimates. The slope coefficients did not change.

¹² The traffic density on the Stockholm primary and secondary roads is well documented, whereas we have limited knowledge about how well primary and secondary roads capture traffic exposure in general in Sweden. With this caveat in mind we also predicted the blood-lead levels in the full sample of children born in 1979 using the distance from residence to road model for central Stockholm. This gave an average blood-lead level in the country as a whole of 5.78 µg/dL in the 1979 cohort. The national average neighborhood moss-lead level in 1977-1979 predicts an average BPb level of pre-school children of 5.41.

¹³ Setting the knot to 250 or 300 meters, or instead using quadratic splines, or replacing the predicted BPb among children residing more than 275 meters from major roads to 5 µg/dL gives very similar results.

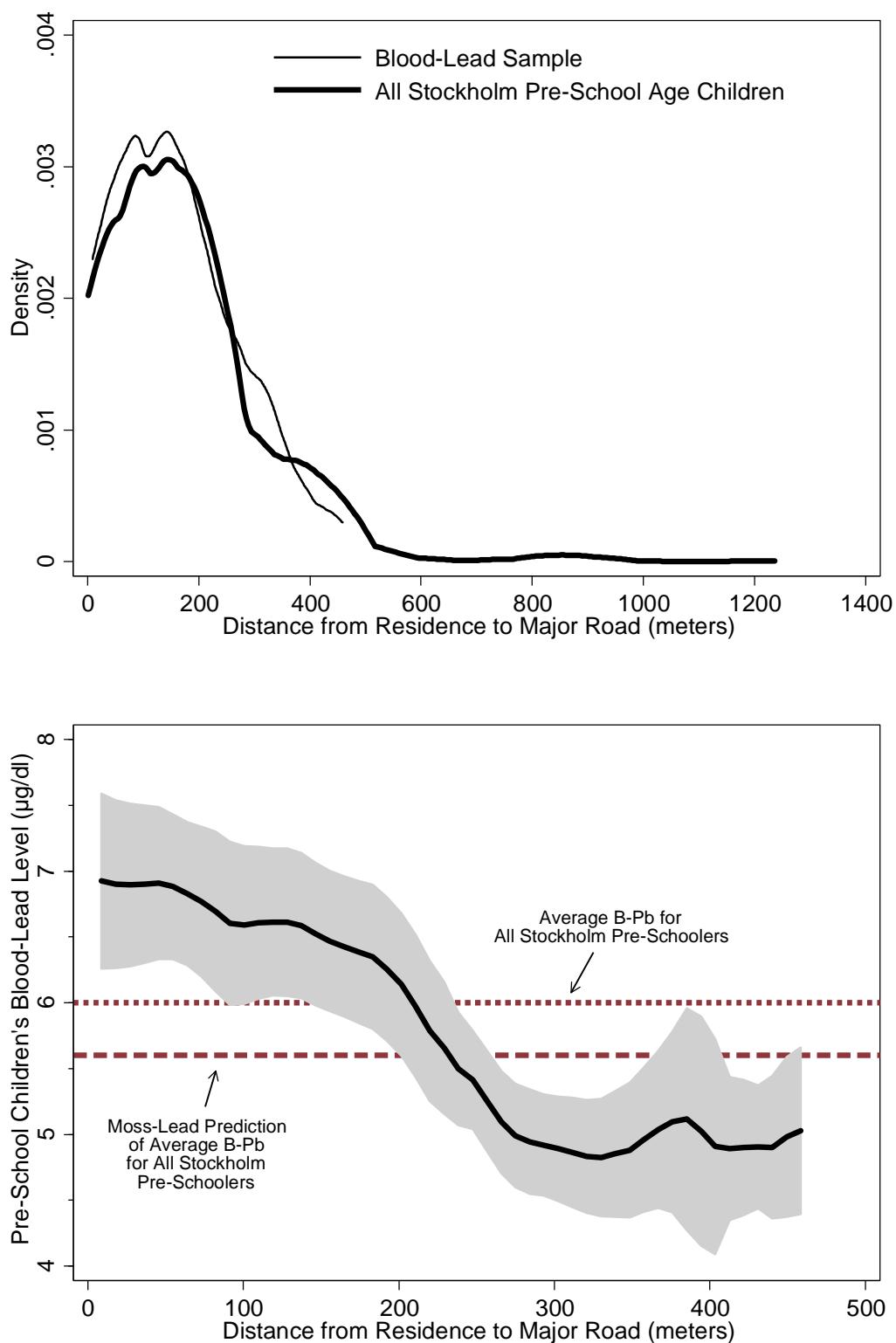


Figure B1. Blood-lead and distance to major roads among Central Stockholm pre-schoolers in 1979

Notes: The top figure plots the kernel density estimate of distance to major roads among the children included in the BPb sample (thin line), and the residential distance to the major roads distribution (thick line) among all children born in Central Stockholm neighborhoods in 1977. The bottom figure plots local average BPb levels (solid thick line), pre-schoolers residing in Central Stockholm in April 1979 vs. the distance to major roads from their address of residence on the x-axis using a bandwidth of 25 meters. The horizontal dotted line shows the mean blood-lead level among all Central Stockholm children, and the horizontal dashed line shows the predicted BPb level of those children using the moss-lead data. See the text for details.

B3. The impact of the 1980/81 reforms on children's BPb levels

So far, we have shown that moss-lead strongly predicts blood-lead in panel data. Because our main empirical strategy focuses on the 1980/81 reforms, we would ideally also want to document the direct effect of the 1980/81 reforms on children's BPb levels. For this purpose, we were able to analyze data on the BPb level using a sample of children residing in Trelleborg municipality. The data was collected by Strömberg et al. (2003) who provided us with data on 651 primary school children's BPb levels, their age and gender, an indicator for whether, at the time of sampling, the child was residing in urban ($n=326$) or more rural ($n=325$) communities, and the year when BPb was sampled. The children are aged 8-12 (mean 9.4), 48% are female, and have an average BPb level of 5.0 $\mu\text{g}/\text{dL}$ (std. 1.8). Since this data is available between 1977 and 1985, we are able to estimate the effects of the 1980/81 reforms on the children's BPb levels. Unlike Landskrona, Trelleborg has no lead emitting industries. Panel A of Table B4 reports difference-in-differences estimates of the effects of the 1980/81 reforms on urban vs. rural children's BPb levels. Following Strömberg et al. (1995), we apply a two-year lag when relating BPb levels to the reform, allowing for lasting environmental contamination and the slow excretion of lead stored in bones. This lag length is consistent with the pattern of the event-study estimates of the 1980/81 reforms on the long-term outcome (see the main text).

Figure 6 in the main text shows how the average BPb levels among rural and urban children evolved around the time of the reform. Before the reform, the trends in BPb levels are parallel and flat, and higher among urban children. Then, around the time of the reform, the BPb levels in both groups start to decrease, with a greater reduction among urban children. After the reform, the average BPb level in the two groups converges, and flattens out at around 4 $\mu\text{g}/\text{dL}$.

Table B4 provides difference-in-differences estimates using this data and compares the impact of the reform on BPb in urban vs. rural areas of Trelleborg using three versions of the following specification.

$$(B2) \quad \ln(\text{BPb})_{\text{inc}} = \alpha + \beta(\text{Urban}_{\text{inc}} \times \text{Reform}_{\text{inc}}) + \theta X_i + \lambda_n + \lambda_c + \varepsilon_{\text{inc}}$$

where BPb is the individual blood-lead level, and Urban \times Reform is an interaction variable equal to 1 if the child is residing in urban neighborhoods and blood-lead was measured in one of the post-reform years. X_i is a vector controlling for age and gender fixed effects, λ_n , is a

dummy equal to 1 if the child is residing an urban area, and λ_c are sampling period fixed effect (post, period, or year of sampling fixed effects depending on the specification).

The reform dummy is an indicator equal to 1 if the BPb levels reflect post-reform levels (1981, 1983, 1985 in the urban and rural sample), and equal to 0 if reflecting pre-reform levels (1977, 1978, 1979 in the rural sample, 1977 and 1979 in the urban sample).

Column 1 reports $\hat{\beta}^{OLS}$ from equation (B2). Consistent with Figure B2, it shows that the reform affected children in urban areas more than children in rural areas (-20%). In column (2), we allow for differential effects in the urban and rural areas between the phase-in period (1981) and the full-reform period (1983, 1985). We see that the estimates are somewhat smaller (in absolute numbers) in the earlier (-15%) than the later period (-22%). Using the same specification but replacing the period fixed effects (pre, phase-in, and post dummies) with year of sampling fixed effects, the estimates increase somewhat (phase-in: -20%, full reform -27%). Finally, column (4) shows no evidence of differential effects of the reform across boys and girls (an insignificant -1.2%).

From Table B4, we conclude that the reform affected children's BPb levels more in densely populated areas (where the pre-reform BPb levels were higher) and there is no evidence that girls' BPb levels were less affected than those of boys. These findings are important for the interpretation of our results in the main text showing that the phase-out of leaded gasoline affected boys in a much clearer way than girls. Moreover, together with Figure 6, Table B4 provides clear evidence that the reform did not only have a differential effect on moss-lead levels but also on blood-lead levels.

Table B4. The impact of the 1980/81 reforms on children's BPb levels (*Trelleborg*)

<i>Dependent variable:</i>	ln (BPb) (1)	ln (BPb) (2)	ln (BPb) (3)	ln (BPb) (4)
Reform × Urban	-.2036*** (.0568)			-.2278*** (.0867)
Reform (Phase-In) × Urban		-.1484** (.0661)	-.1994*** (.0749)	
Reform (Full) × Urban			-.2196*** (.0773)	-.2698*** (.0847)
Reform × Urban× Girl				-.0123 (.1181)
R-squared	.29	.30	.30	.31
No of children	651	651	651	651
Individual controls	Yes	Yes	Yes	Yes
Urban area fixed effects	Yes	Yes	Yes	Yes
Period fixed effects		Yes		
Year fixed effects			Yes	Yes

Notes: The table reports difference-in-differences estimates of the impact of the 1980/81 reforms on children's BPb levels, comparing the differential impact of the reforms on children residing in urban areas vs. rural areas of Trelleborg municipality. The estimation sample consists of 651 primary school age children (rural n=325, urban n=326). Individual controls are gender and age dummies. Period fixed effects are indicator variables for the period when BPb was sampled (pre, phase-in, and post reform as defined in the text) and year fixed effects are indicator variables for the year of BPb sampling. See the text for more details. Robust standard errors in parenthesis. */**/*** denote statistical significance at the 10/5/1 percent levels, respectively.

B4. Initial BPb levels

In this sub-section, we estimate the initial BPb levels for the children included in our main analysis. We use this knowledge to contextualize our findings and make comparisons with the results in previous studies.

We use four different approaches to estimate the initial BPb levels. Before describing these, note that the earliest systematic sampling of BPb among Swedish children started in 1978 (Strömberg et al. 1995) in two municipalities in southern Sweden (where the air lead levels were among the highest in Sweden). At that time, the BPb level was just above 6 µg/dL on average. Needleman et al. (1979) report average BPb levels in the US in the same year of 14 µg/dL.

The first approach to predict the pre-1978 BPb of children born in 1973-1974 is to use the BPb time series displayed in Figure 2 and regress it on the lead content in gasoline during the same period. For the period 1978-2000, the estimated relationship for primary school children is $\text{BPb}=2.406+2.688091 \times \text{grams/gallon}$. Reyes (2007) uses NHANES data and finds that an increase of 1 gram lead per gallon of gasoline increases the BPb level by approximately 3.3 µg/dL in the general population, and that the relationship between gasoline lead content and BPb is 30% higher for children aged 0 to 6 than for children 6-12. Scaling our model with this estimate leaves us with a model for Swedish pre-primary school children's BPb in 1973-1974 as follows:

$$\text{BPb}=2.406+3.4945 \times \text{grams/gallon}$$

This model suggests that in the year of birth of the cohort born in 1973-74, the BPb level would on average have been 9.0 µg/dL.

A second approach is to take the drop in urban children's BPb level around the 1980/81 reforms in Trelleborg before vs. after the full reform (2.04 µg/dL) (see Table B4) and divide it by the drop in grams of lead per gallon of gasoline due to the reform (0.946 grams per gallon). This gives an alternative estimate of the relationship between gasoline lead content and BPb levels ($2.04/0.946=2.158$). Adjusting for age as above and plugging the estimate into the equation above gives the following relationship

$$\text{BPb}=2.406+2.158 \times \text{grams/gallon}$$

This model suggests that in the year of birth of the cohort born in 1973-74, the BPb level would on average have been 7.7 µg/dL among urban children.

A third approach is to combine the estimates in Table B2 column (2) with the average MPb level in 1975 to predict the BPb levels in primary school children at that time. This approach provides a predicted average initial BPb level of around 3 µg/dL. After adjusting for

age and under the additional assumption that the additive separable specification used in the estimation holds for both populations, the relevant BPb level for children aged 0-6 would, on average, correspond to about 5 µg/dL. If using the estimated elasticity when controlling for the influence of the smelter (column (4) of Table A3), the predicted initial average BPb level is 7.8 µg/dL.

A fourth approach to estimating the initial BPb level is to use the estimates in Strömberg et al. (1995) who, based on repeated individual BPb measurements, find that BPb levels in primary school children on average decrease by around 6% per year. Given the average BPb level in 1978, this implies that the average BPb levels among 1-4 year olds in 1973 (since the samples are taken from children aged 7-10 in 1978) would, on average, be 8.5 µg/dL.

All four approaches provide estimates suggesting that the initial BPb levels were on average below 10 µg/dL for the cohorts born between 1973 and 1974. Using the BPb level standard deviation (1.3 µg/dL) of pre-school children in Stockholm suggests that 97.5% of the first cohort of children in our sample had BPb levels below (i) 11.5 µg/dL, (ii) 10.3 µg/dL, (iii) 7.6 µg/dL (10.3 µg/dL if controlling for the effect of the smelter), and (iv) 11.0 µg/dL, depending on the approach used to estimate the early childhood mean blood-lead level in 1973-1974. Note that all except the third of these approaches use data from the two municipalities in southern Sweden. Since these samples were taken in a region with high lead exposure (based on the MPb data), it seems reasonable to assume that these levels represent an upper bound of the BPb level in the general population of children in these birth cohorts.

Web Appendix C:

Additional results: Panel data analysis

C1. Correlations between changes in local lead exposure and changes in municipality characteristics

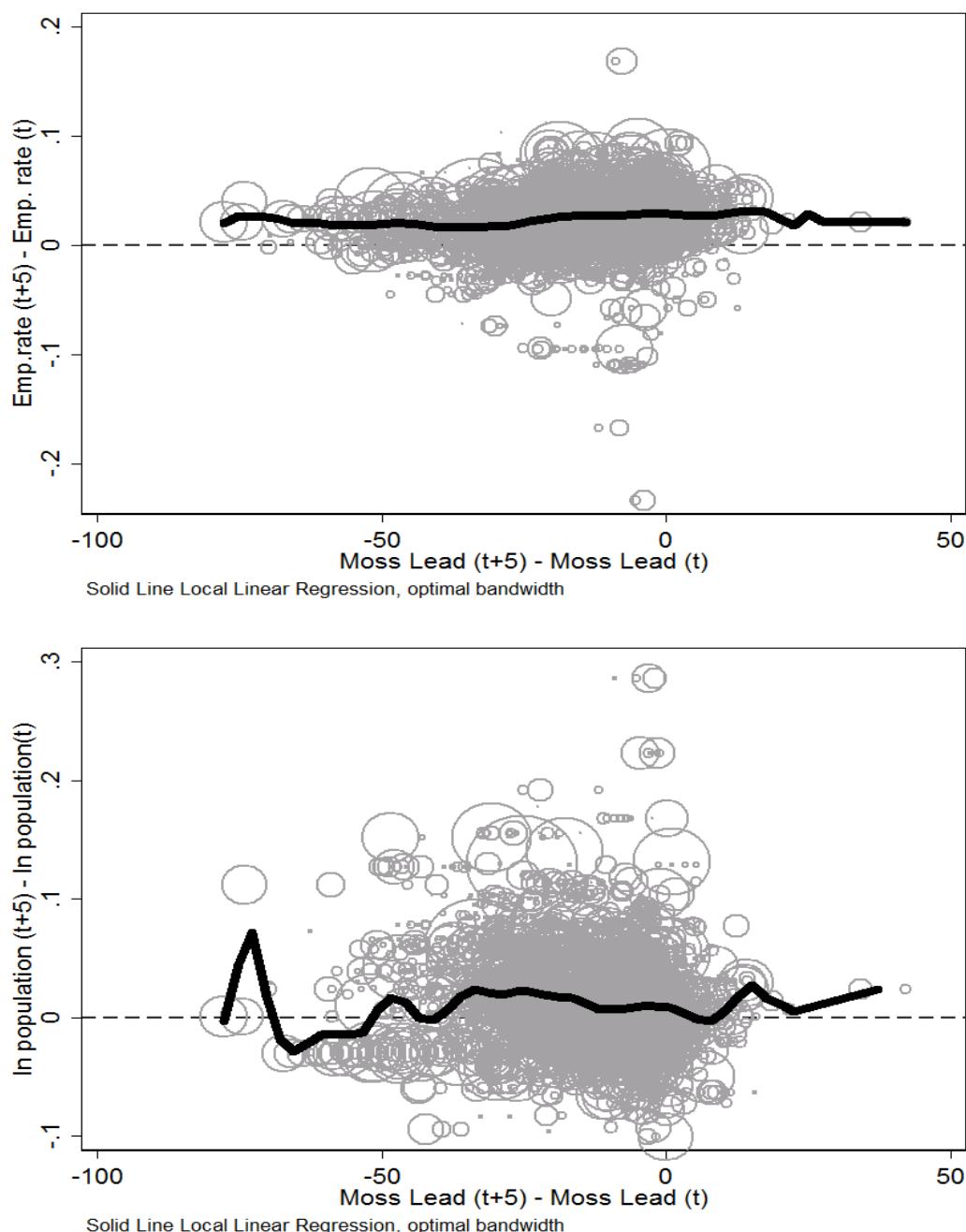


Figure C1. Changes in local MPb levels vs. changes in employment (top figure) and population density (bottom figure).

Notes: Scatter plot and local linear regression weighted by number of children born in the neighborhood during the observation period.

C2. Baseline robustness checks: differential trends and different ways of clustering

Table C1 provides additional baseline robustness checks, addressing the key concern of differential trends in between rural and urban areas as well as sorting and alternative ways of accounting for spatially correlated errors.

To assess the importance of differential trends, we estimated models where we split the country into a Northern/South sector or a North/Central/South Sector (Norrland/Svealand/Götaland), and interacted these dummies with the cohort of birth dummies, thus allowing for differential trends across the more rural north and the more densely populated Central and Southern regions. We also tested a specification where we excluded the entire Stockholm CZ (22% of the sample), and a specification excluding the three most populated municipalities (Stockholm, Göteborg, and Malmö, accounting for 14% of the sample). Finally, we added initial population density specific trends. Overall, neither of these specification checks change the results in any significant or systematic ways, giving a first indication that unobserved differential rural/urban trends do not seem to explain the association between early lead exposure and long-term outcomes.

If sorting within the neighborhoods is correlated with changes in lead exposure, this could also bias our estimates. Over the observation period, new residential areas were established all across Sweden, often in areas where traffic density was lower. To check whether within-neighborhood sorting is correlated with the changes in lead exposure, we tested if the average distance (in meters) to major roads within neighborhoods changed at the same time as the changes in lead by using (log) distance as the dependent variable in the full specification.¹⁴ The point estimate was small, negative, and not significant (estimate -.00026 std. err: .00102), suggesting that a 30 mg/kg increase in lead exposure is associated with less than a 1% decrease in the distance to roads. Changes in the distance to roads due to gentrification or the establishment of new residential areas are therefore unlikely to drive the main results.

¹⁴ The distance to major roads (highways and primary roads) from the residence at birth is calculated by using place of residence (available at the 200m×200m (1km×1km) grids in urban(rural) areas. We calculated the distance to major roads from each residential grid centroid using data from OpenStreetMap.org, and then used the (log) average distance within the neighborhoods as the outcome variable in these regressions.

Table C1. Robustness checks: rural and urban trends and alternative ways of clustering

<i>Outcomes:</i>	Grade 9 GPA (pct. rank)	P(High school completion)	P(Ever convicted)	P(Property crime)	P(Violent crime)
<i>Baseline results</i>	(1)	(2)	(3)	(4)	(5)
A: Full specification (for reference)	-2097** (.0925)	-.0031** (.0014)	.0018** (.0008)	.0008 (.0009)	.0002 (.0003)
B: Add (North×Cohort) FE	-.2411** (.0945)	-.0027* (.0014)	.0013 (.0010)	.0005 (.0007)	-.0002 (.0004)
C: Add (North/Central/South) by Cohort FE	-.2256** (.0882)	-.0019 (.0016)	.0021*** (.0007)	.0011 (.0008)	.0003 (.0003)
D: Excluding Stockholm CZ	-.2187** (.1025)	-.0032* (.0017)	.0016* (.0009)	.0012 (.0009)	.0006*** (.0002)
E: Excluding the three largest municipalities.	-.131* (.0780)	-.0031* (.0017)	.0018* (.0009)	.0005 (.0009)	.0002 (.0004)
<i>Additional results wrt Trends</i>					
F: Adding baseline population density specific trends	-.225** (.091)	-.0035** (.0014)	.0020** (.0009)	.0007 (.0009)	.0002 (.0003)
G: Adding baseline pop. density specific quadratic trends	-.207** (.082)	-.0032** (.0015)	.0019** (.0008)	.0006 (.0008)	.0002 (.0003)
<i>Alternative ways of clustering</i>					
H: Clustering at the Nhbrhd level	-.2097*** (.063)	-.0031*** (.0010)	.0018** (.0009)	.0008 (.0006)	.0003 (.0003)
I: Clustering at Nbrhd-by-cohort level	-.2097*** (.0529)	-.0031*** (.0008)	.0018*** (.0006)	.0008* (.0005)	.0003 (.0003)
J: Clustering at Closest Pb Measurement point level	-.2097*** (.065)	-.0031*** (.0011)	.0018** (.0009)	.0008 (.0006)	.0003 (.0003)
Mean of Outcome Variables	[50.72]	[.79]	[.164]	[.07]	[.024]
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. The full specification is highlighted in bold for reference. 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 by age 24. Childhood lead exposure is measured as an average over ages 1-3. All regressions are weighted by the number of children in each neighborhood-cohort cell. Clustered robust standard errors (at the commuting zone level, 74 cells) are shown in parenthesis unless otherwise stated. */**/** denote statistical significance at the 10/5/1 percent levels, respectively.

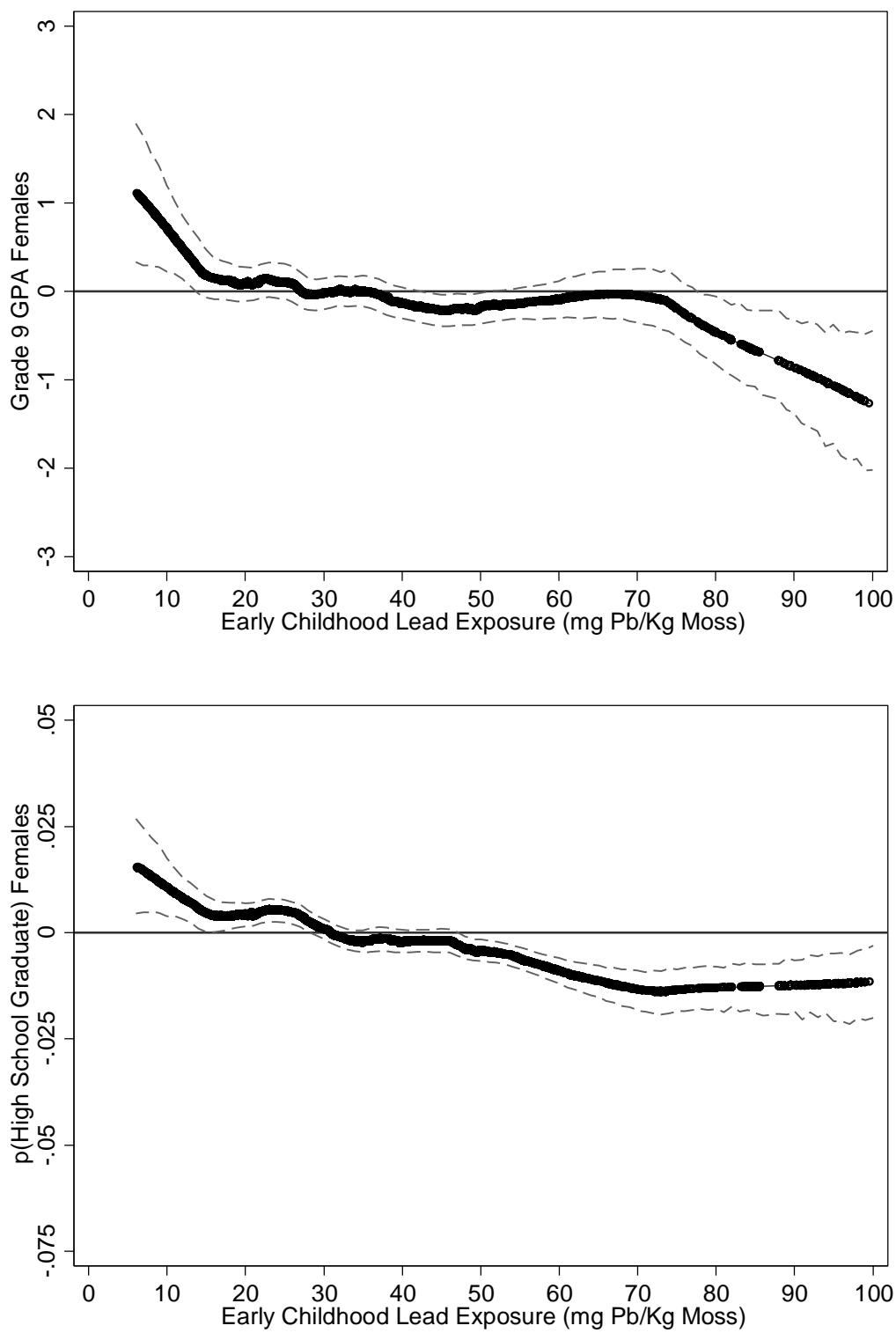


Figure C2. Semi-parametric estimates human capital (FEMALES)

Notes: The graphs present estimates along 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). Childhood lead exposure is measured as an average over ages 1-3. See the notes of table 4 for details regarding the full set of controls. The confidence interval accounts for clustering at the CZ level.

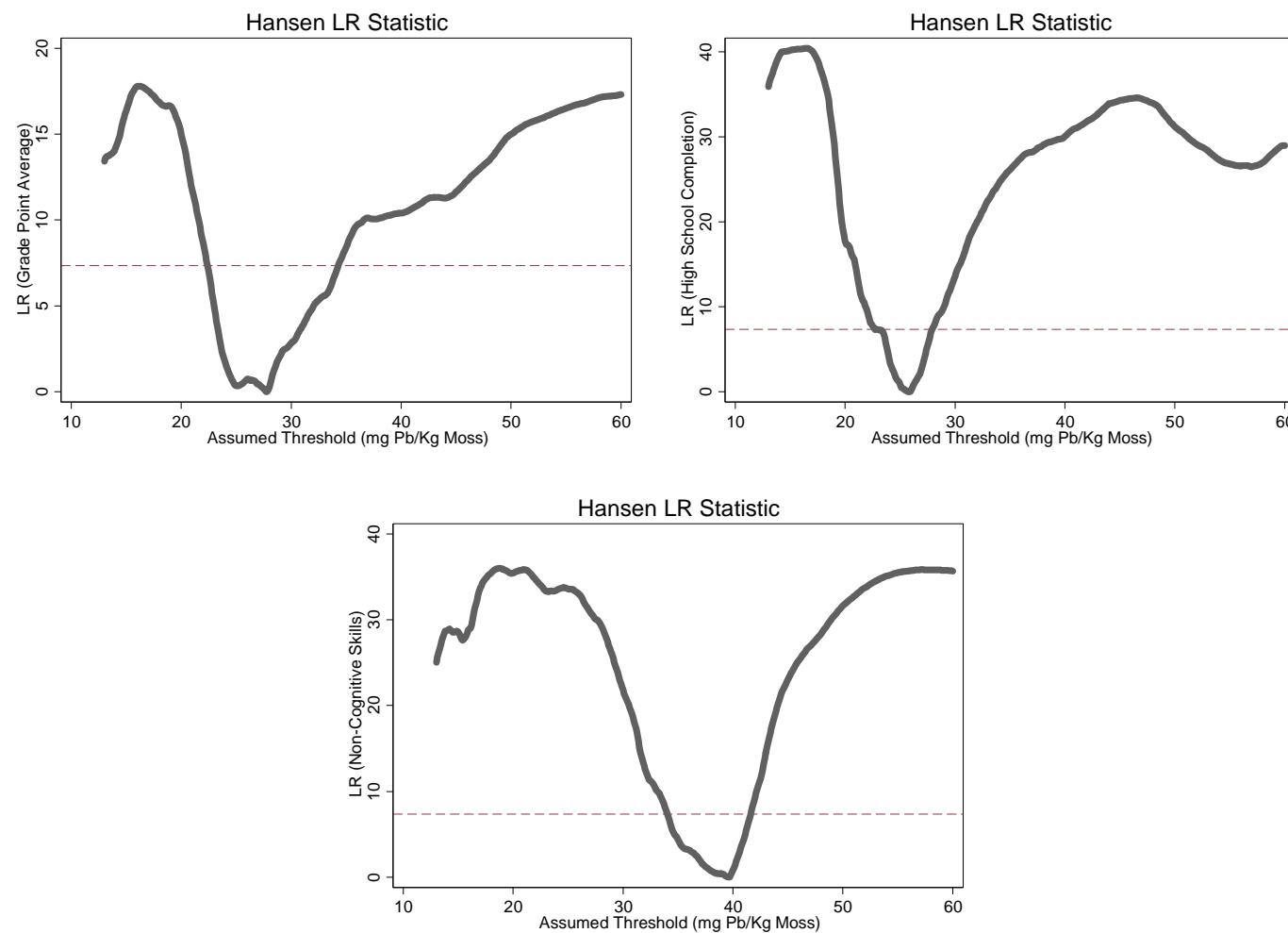


Figure C3. Hansen's LR test for the threshold for human capital outcomes (MALES).

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 estimated most likely location of 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 and LR's below the vertical dashed lines are locations which are within the "no rejection" regions, within which we cannot reject that the true threshold is actually located. We do not search for thresholds for cognitive skills since Figure 7 shows no indications of meaningful thresholds for cognitive skills within the ranges of exposure observed in our sample.

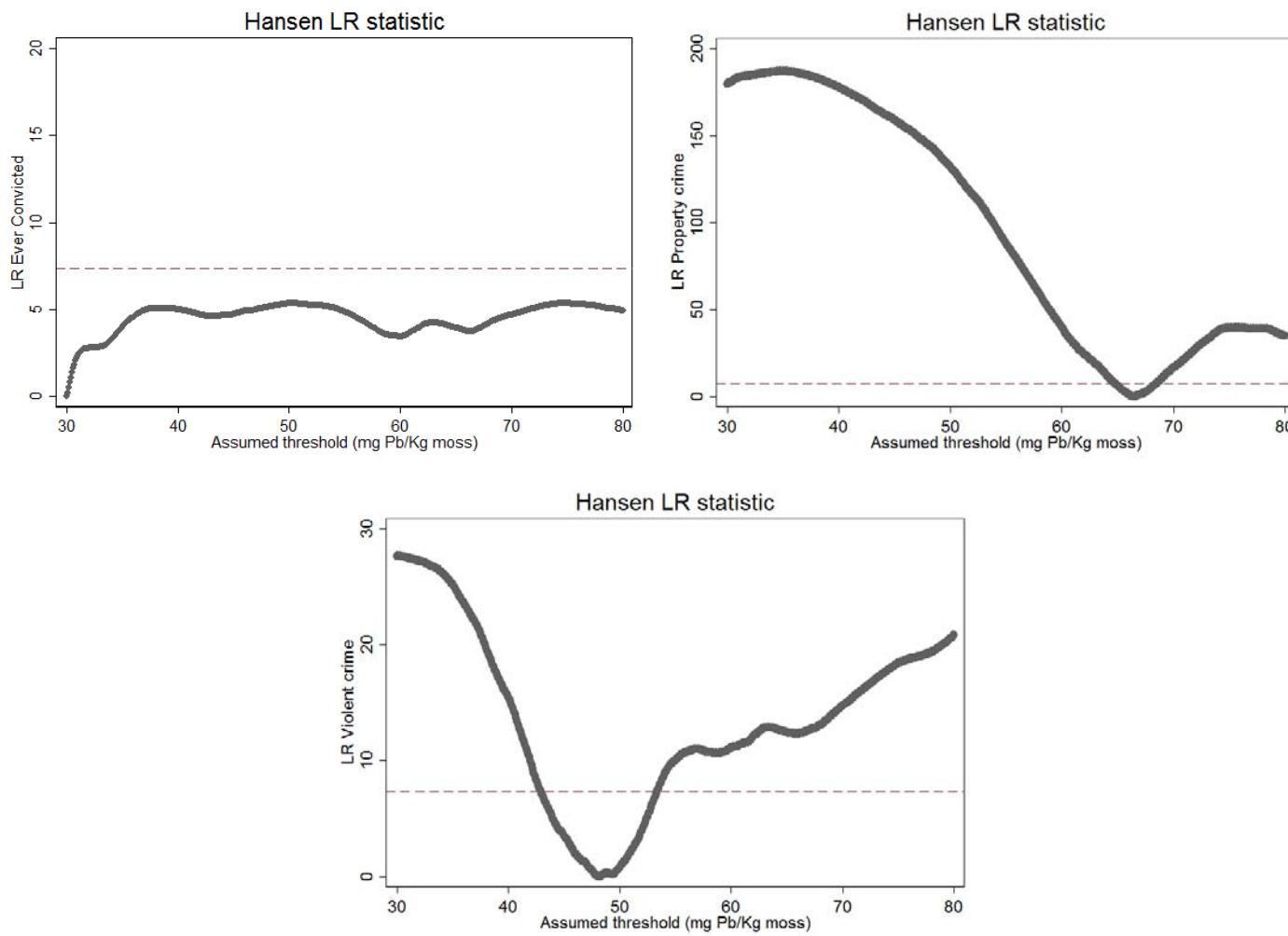


Figure C4. Hansen's LR test for the threshold effect for crime outcomes (MALES)

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). Note that for violent crime and property crime, we identify threshold ranges; however, for the ever convicted outcome, there is no location that is significant. The 5% critical value of the likelihood ratio is 7.35.

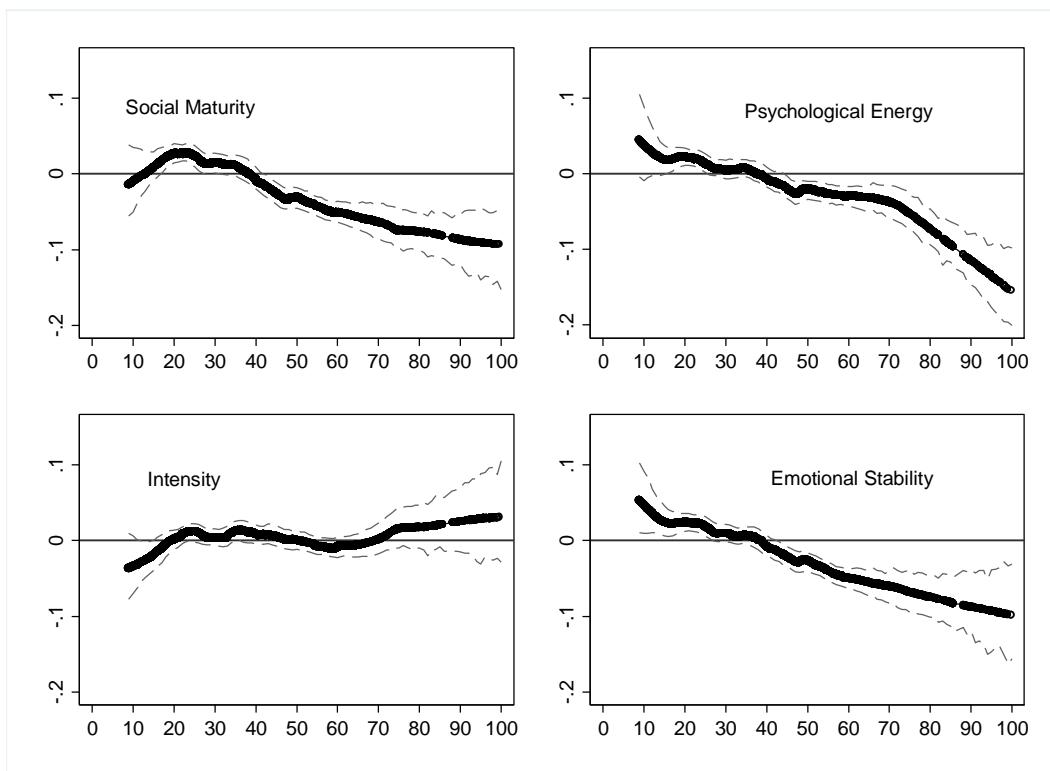


Figure C5. Semi-parametric estimates for sub-components of non-cognitive skills (Males)

Notes: The graphs present estimates along the 95% confidence interval (bootstrapped using 1,000 repetitions) from semi-parametric fixed effects models (see Baltagi and Li 2002). The non-cognitive score is from the psychologist evaluation 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)penness 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** (the 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). Besides neighborhood and birth cohort fixed effects, the model controls for the full set of controls as specified in Table 4.

Appendix D:

Additional results: The analysis of the 1980/81 reforms

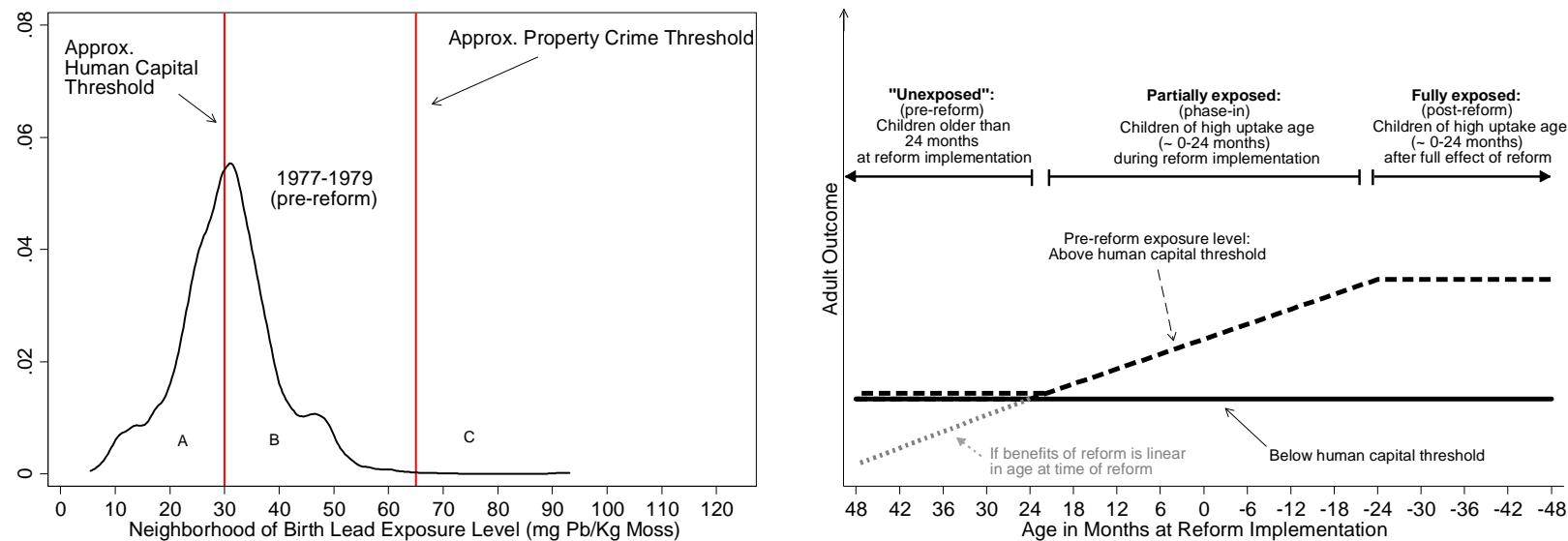


Figure D1. Illustration of the location of the human capital and crime thresholds in the pre-reform (1977-1979) distribution of exposure and the hypothetical impact of the reform

Notes: The left-hand side figure plots the average neighborhood of the birth lead exposure level as measured in moss in 1977-1979. The figure highlight the predictions described in section 4.3. If born in a neighborhood with a pre-reform exposure level below the estimated human capital threshold (for example at location A in the figure), then we predict that the reform induced changes in lead exposure should not affect the outcome of the child. If, on the other hand, born in a neighborhood at location B (i.e. above the human capital threshold) then we could expect to see improvements in the child's human capital outcomes following the reform. For the crime outcomes we do not expect to see any improvements in the outcomes if born in locations A or B following the reform since this is below the estimated crime threshold. In the event-study analysis we examine these predictions as outlined in section 4.3 by splitting the data at the human capital threshold and estimating equation (2) separately. We do not estimate separate models for those above the *crime threshold* (e.g. at location C) since, as can be seen in the figure above, few children grow up in neighborhoods still above the threshold in 1977-1979. The right-hand side figure provides a hypothetical example of an exogenous decrease in lead exposure across children's age and their neighborhood of birth (above or below human capital threshold neighborhoods). This hypothetical example shows the transition path following an exogenous decrease in air lead levels in below (solid line) and above (dashed line) threshold neighborhoods when the adult outcomes of children are affected by decreases in exposure before the age of 24 months (due to the well-documented differences in the lead take-up and sensitivity) and it takes 24 months before an immediate reduction in air lead fully translates into a reduction in actual lead exposure (due to lingering environmental contamination). The gray dotted line shows the expected transition path in above threshold neighborhoods when there is no difference in the lead take-up/sensitivity among children aged above or below 24 months at the time of the implementation of the reform.

Constructing Figure 10A

Below we provide the step by step estimates needed to create the figure summarizing the estimates shown in Figure 10A.

- 1) To construct the figure we first standardize all outcomes (mean 0, SD 1). We then estimate equation (2) for each standardized outcome separately for children born in neighborhoods above and below the human capital threshold, retaining the γ estimates.
- 2) We then group the outcomes into two domains. The first domain of outcomes (GPA, High School Completion, and Non-cognitive skills) contains outcomes that, based on the findings from the semi-parametric estimates, we expect to be affected by the 1980/81 reforms, at least among children born in neighborhoods above the estimated human capital threshold (30 mg/kg). The second domain (property crime, violent crime, and cognitive skills) contains outcomes which are expected not to be affected by the 1980/81 reforms, either because the estimated threshold was located at a much higher level of exposure (i.e. property crime, violent crime), which was out of reach at the time of the 1980/81 reforms (see the main text, and Figure D1), or because we found no clear or significant pattern in the semi-parametric panel data analysis (i.e. cognitive skills).
- 3) We orient all estimates so that a positive sign implies a more beneficial outcome (i.e. positive signs for less crime). For the two domains of outcomes, we then calculated a mean of the γ estimates using a local mean smoother (bandwidth 1 year). We use the inverse of the standard errors of the γ point estimates as weights, giving more weight to more precise estimates. We plot the local domain means (solid line for above threshold neighborhoods, and dashed line for below threshold neighborhoods) together with the γ estimates (solid circle for above threshold neighborhood point estimates, and hollow circles for below threshold neighborhood estimates) below in Figure D6.
- 4) We difference the local means of the above threshold neighborhoods and below threshold neighborhoods (i.e. the difference between the solid and the dashed line) for each outcome group and plot the differenced values in the same graph (Figure 10A). That is, Figure 10A summarizes the findings in the fourteen figures shown below.

Below we present the estimates on the original (unstandardized) outcomes individually (figures D2 to D5, grouped as in Figure 10A), together with 95% point-wise confidence intervals, and F-tests of equality of coefficients between the pre-vs-post reform period. Figure D6 reports the estimates for the standardized outcomes, and Figure D7 shows the main text graph.

STEP 1: DID-event study estimates by (unstandardized) outcomes for outcomes predicted to be affected by the reform (Domain 1)

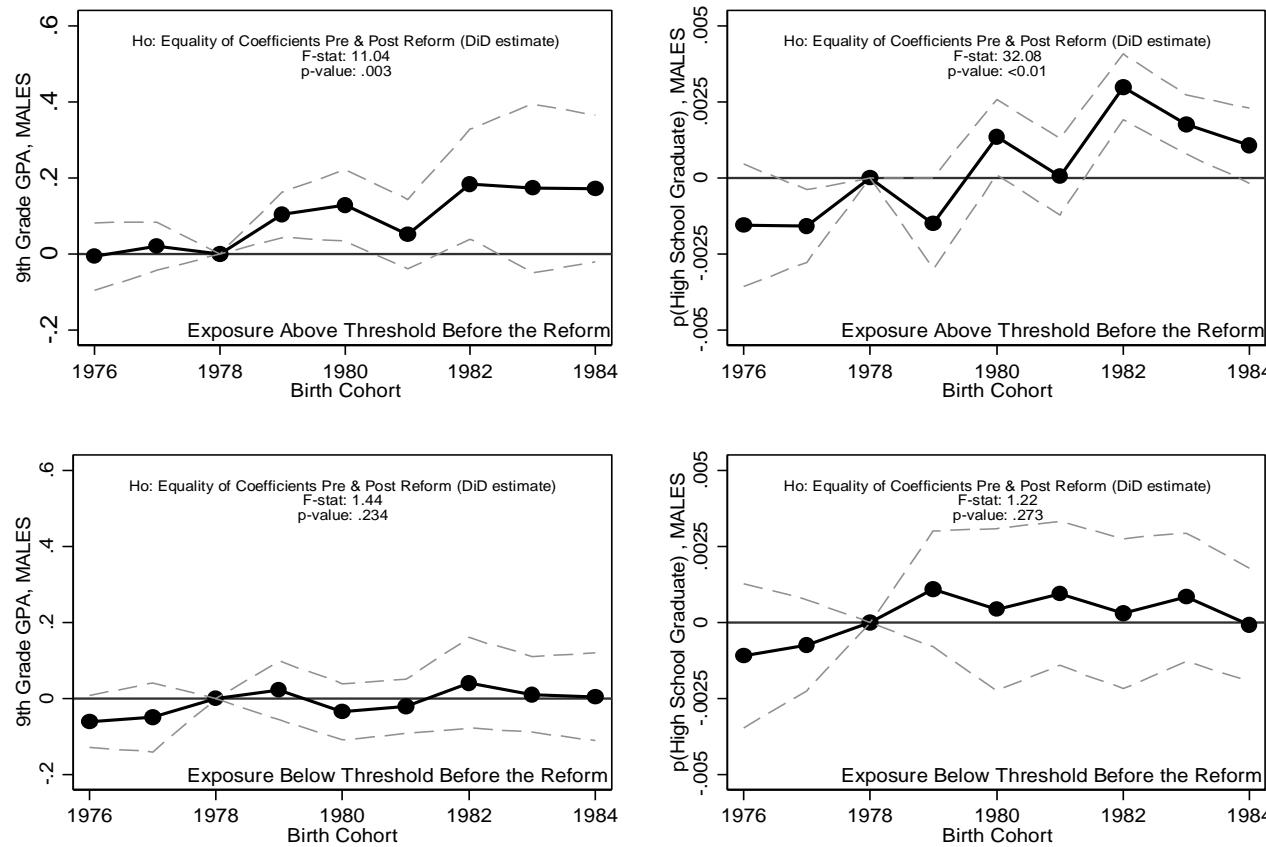


Figure D2. GPA (percentile-ranked) and high school completion

Notes: These figures show the point estimates and pointwise 95% confidence bands from the estimation of the cohort-specific γ_c from equation (2) for the male sample together with the F-stats and p-values for a test of equality of the γ_c coefficients before and after the reform (i.e. difference-in-differences for 1976-1978 vs. 1979-1984 cohort coefficients). The top (bottom) left-hand figure provides the estimates for 9th grade GPA among children born in neighborhoods with a lead level above (below) 30 mg/kg just before the reform. The top (bottom) right-hand figure provides the estimates for high-school completion among children born in neighborhoods with a lead level above (below) 30 mg/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 10. See Figure 10 for further details.

STEP 1: DID-event study estimates by (unstandardized) outcomes for outcomes predicted to be **affected by the reform (Domain 1). -**
Continued...

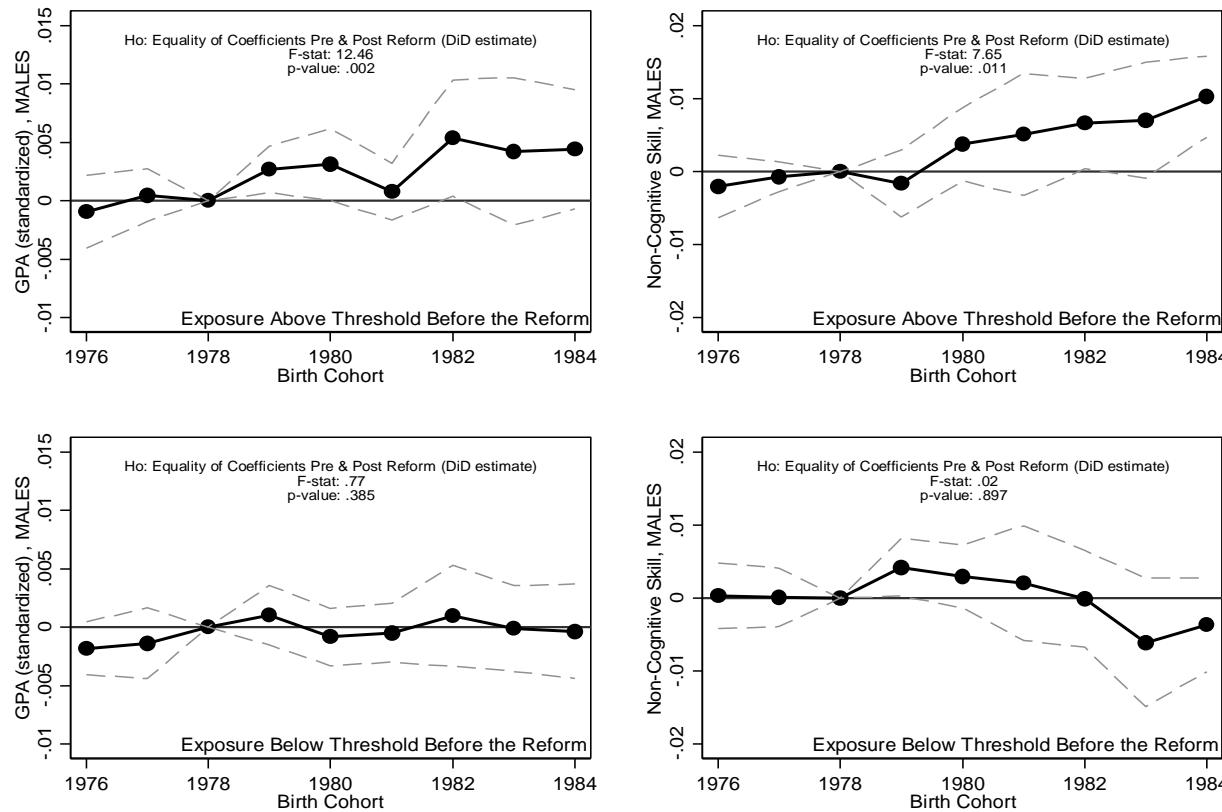


Figure D3. GPA (z-score) and non-cognitive skills

Notes: These figures show the point estimates and pointwise 95% confidence bands from the estimation of the cohort-specific γ_c from equation (2) for the male sample together with the F-stats and p-values for a test of equality of the γ_c coefficients before and after the reform (i.e. difference-in-differences for 1976-1978 vs. 1979-1984 cohort coefficients). The top (bottom) left-hand figure provides the estimates for 9th grade GPA among children born in neighborhoods with a lead level above (below) 30 mg/kg just before the reform. The top (bottom) right-hand figure provides the estimates for high-school completion among children born in neighborhoods with a lead level above (below) 30 mg/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 10. See Figure 10 for further details.

STEP 1: DID-event study estimates by (unstandardized) outcomes for outcomes predicted to be **unaffected by the reform (Domain 2)**

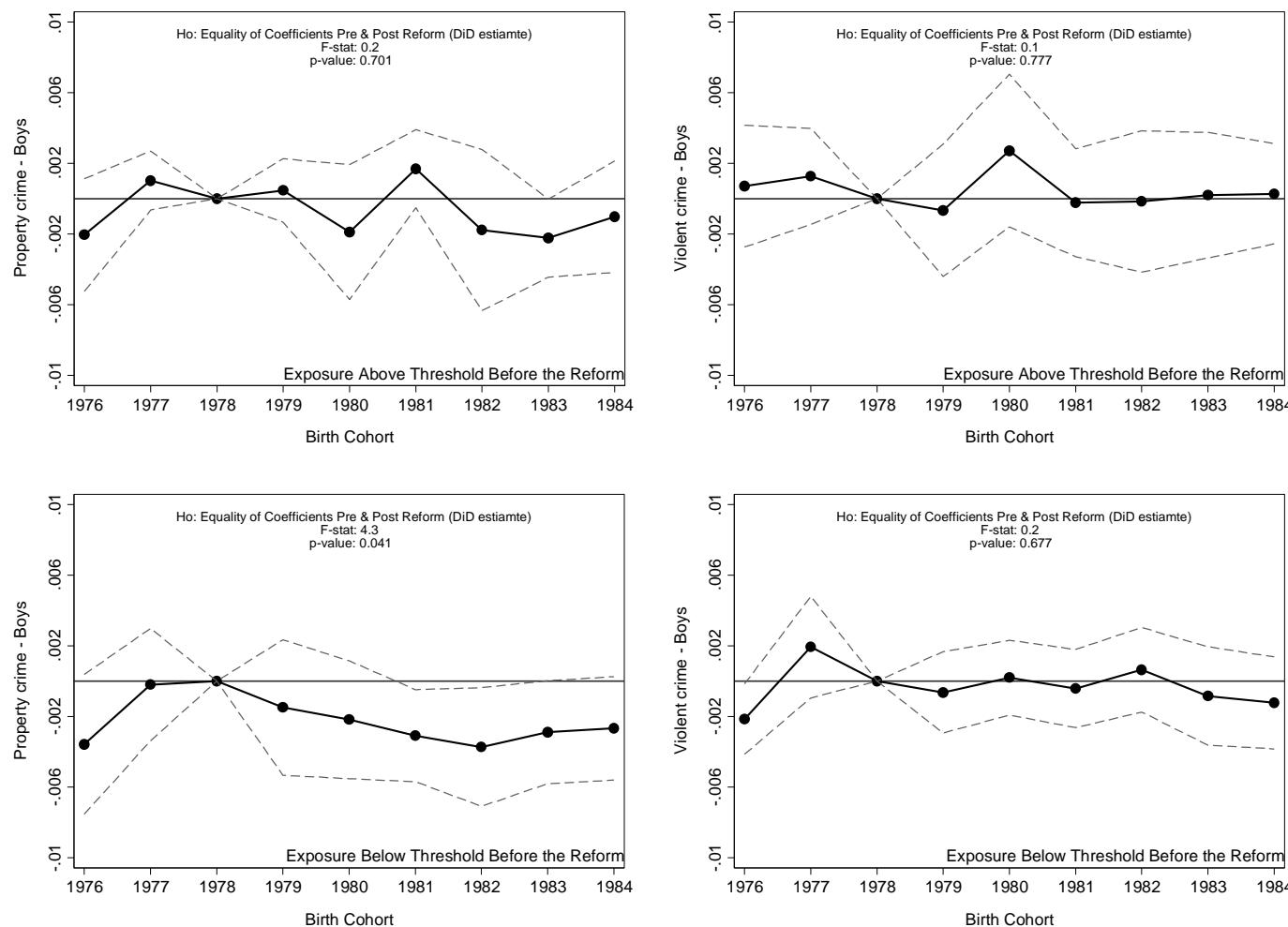


Figure D4. Property crime and violent crime

Notes: These figures show the point estimates and pointwise 95% confidence bands from the estimation of the cohort-specific γ_c from equation (2) on the male sample together with the F-stats and p-values for a test of equality of the γ_c coefficients before and after the reform (i.e. difference-in-differences for 1976-1978 vs. 1979-1984 cohort coefficients). The top panel provides the estimates of children born in neighborhoods with a lead level above (below) 30 mg/kg just before the reform. The bottom panel provides the estimates for children born in neighborhoods with a lead level above (below) 30 mg/kg just before the reform.

STEP 1: DID-event study estimates by (unstandardized) outcomes for outcomes predicted to be unaffected by the reform (Domain 2), Continued...

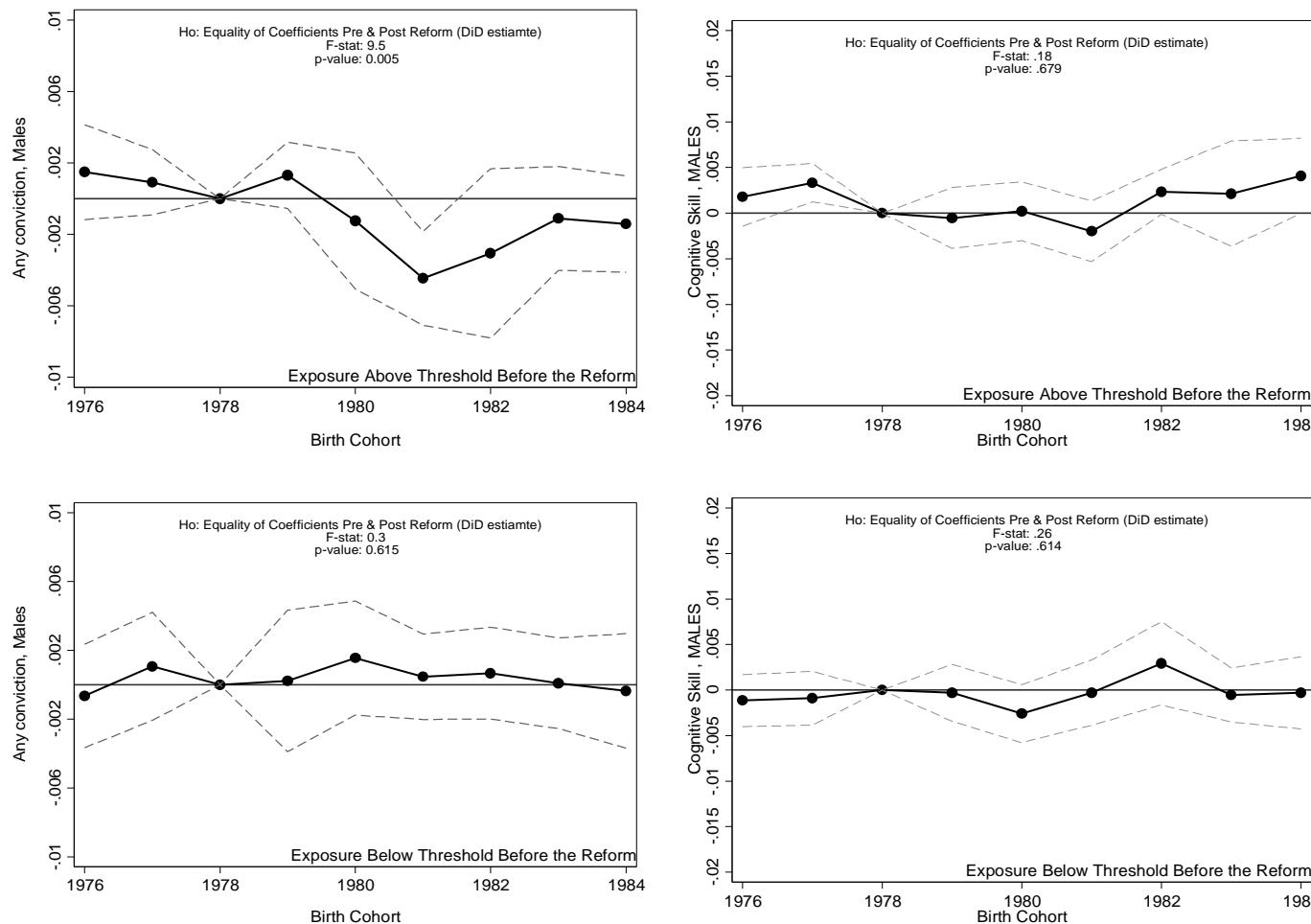
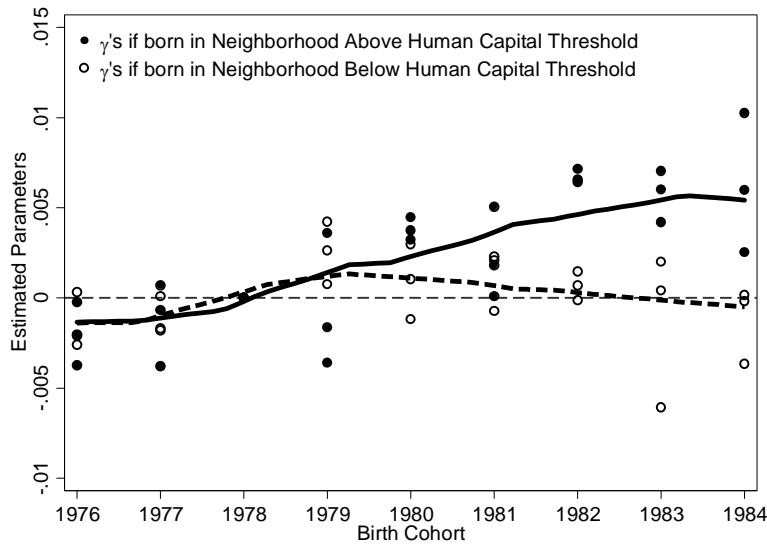


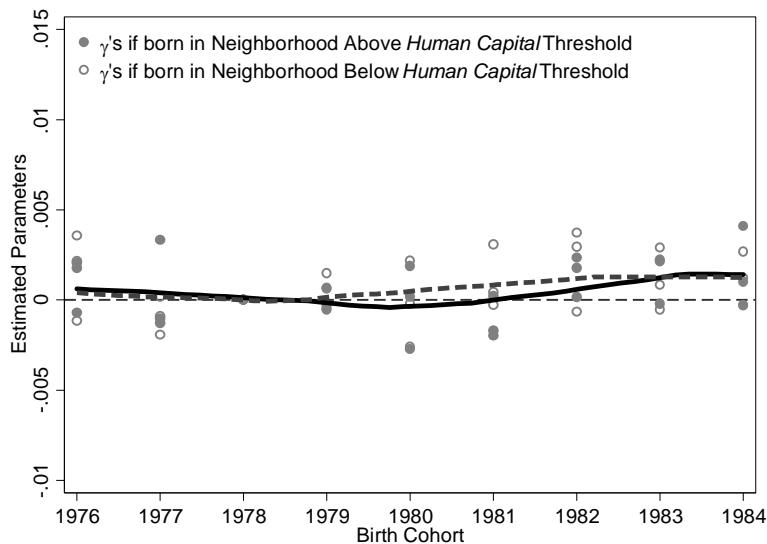
Figure D5. Any crime and cognitive skills

Notes: These figures show the point estimates and pointwise 95% confidence bands from the estimation of the cohort-specific γ_c from equation (2) on the male sample together with the F-stats and p-values for a test of equality of the γ_c coefficients before and after the reform (i.e. difference-in-differences for 1976-1978 vs. 1979-1984 cohort coefficients). The top panel provides the estimates of children born in neighborhoods with a lead level above (below) 30 mg/kg just before the reform. The bottom panel provides the estimates for children born in neighborhoods with a lead level above (below) 30 mg/kg just before the reform.

Step 2 Local means of the estimated coefficients across domains



Panel A: Local means of Domain 1 coefficients



Panel B: Local means of Domain 2 coefficients

Figure D6. DiD estimates of the 1980/81 reforms on human capital and crime (MALES)

Notes: The figures provide the point estimates using the standardized outcomes. The outcomes are standardized before the estimation (mean 0, SD 1). 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 are the estimates for children in low exposure neighborhoods. The solid 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. Panel A shows the results for outcomes predicted to be affected by the reform (GPA, high school completion, and non-cognitive skills), and Panel B shows the results for outcomes predicted to be unaffected by the reform (-property crime, -violent crime, and cognitive skills). The sample consists of males born in 1976-1984. Pre-reform lead exposure reflects exposure in 1977-1979. Children born in 1978 are above the age of 24 months when the take up generally starts to decline (c.f. Canfield et al. 2003) at the time of the full reform, and is therefore used as a reference cohort.

Step 3 Differences in the local means of the estimated coefficients across domains

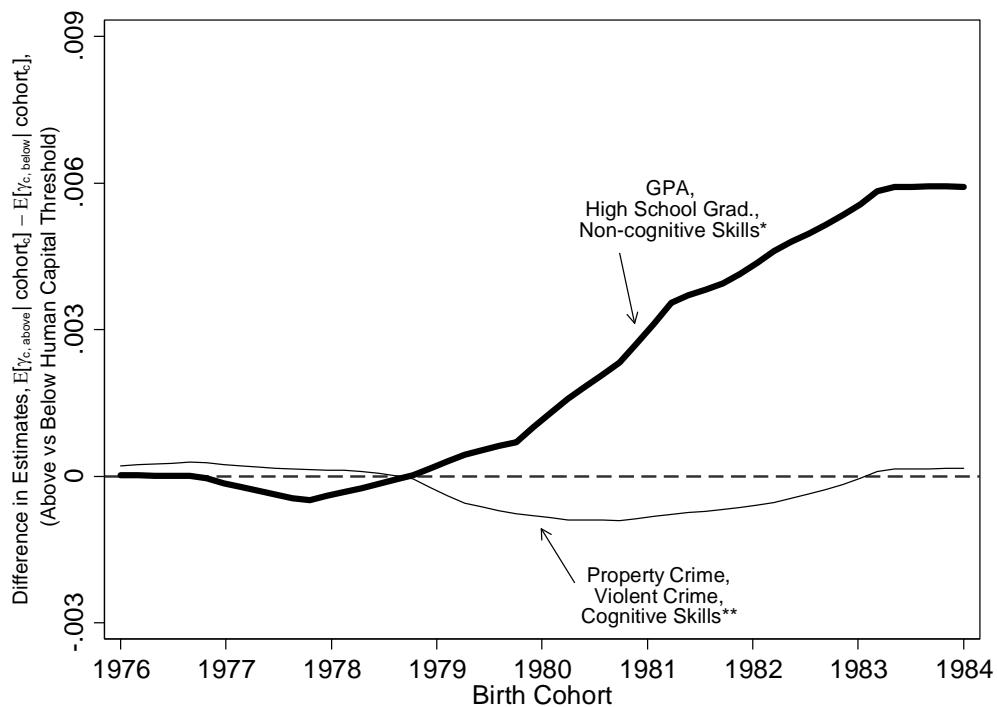


Figure D7. Summary of event study estimates of the 1980/81 reforms on long-term outcomes (Males)
Notes: This figure shows the differences in the fitted values of the local mean smoother in Step 2.

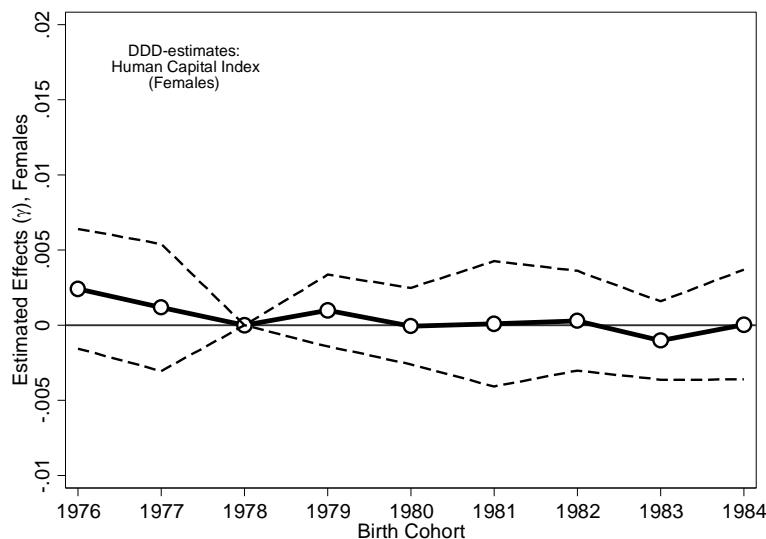


Figure D8. DDD estimates for the summary index of long-term outcomes (Females)

Notes: The figure displays point estimates (γ 's) from the triple-difference version of equation (2) for females. It shows the impact of the 1980/81 reforms on an index of the long-term outcomes predicted to be affected by the reform. The dashed lines represent 90% pointwise confidence intervals. Standard errors are clustered at the CZ level. See Figure 10 for further details on the estimation. We do not have any measures on non-cognitive skills (enlistment data) for women. The index of human capital skills for women is therefore instead based on GPA and high school completion data only. The corresponding figure for males (i.e. without using non-cognitive skills to create the human capital index) yields a similar pattern to that of Figure 10B, and is available upon request.

Table D1. Effect of the 1980/81 reforms on FEMALEs

<i>Panel A: Above-threshold</i>	Dependent variable:			
	GPA		P(High School Graduation)	
	OLS	2SLS	OLS	2SLS
Lead exposure	-.04363 (.0434)	-.1096 (.0729)	-.0008 (.0006)	-.00046 (.00111)
Commuting zones	46	46	46	46
Neighborhoods	1,313	1,313	1,313	1,313
Children	172,786	172,786	172,786	172,786
First-stage <i>F</i> -statistic	N/A	49.1	N/A	49.1

<i>Panel B: Below-threshold</i>	Dependent variable:			
	GPA		P(High School Graduation)	
	OLS	2SLS	OLS	2SLS
Lead exposure	-.06458** (.0301)	-.1931 (.13071)	.00002 (.0004)	-.00346 (.00226)
Commuting zones	71	71	71	71
Neighborhoods	1,138	1,138	1,138	1,138
Number of Children	123,780	123,780	123,780	123,780
First-stage <i>F</i> -statistic	N/A	19.4	N/A	19.4

Notes: The table presents OLS and 2SLS estimates from equation (3) for the reform cohort sample. For comparability across outcomes, the estimation sample consists of females for whom all outcomes are non-missing. The excluded instrument for the second stage is the interaction term (post-cohorts dummy×pre-reform MPb level). All regressions control for neighborhood fixed effects and the baseline control variables (see note in Table 4). The 2SLS model also controls for the phase-in period (1979-1981) interacted with the pre-reform moss-lead level, interpolated pollution measures (Cd, Cu, Zn) for cohorts 1980-1982, as well as gasoline sales in the municipality of birth. The OLS model nets out all influences from the phase-in period using phase-in dummies interacted with all controls to provide estimates comparable with the 2SLS estimates. Standard errors are two-way clustered at the neighborhood-by-cohort and commuting zone level.

Web Appendix E:

Additional results: Effects on neonatal health, earnings in 2013, and comparing siblings

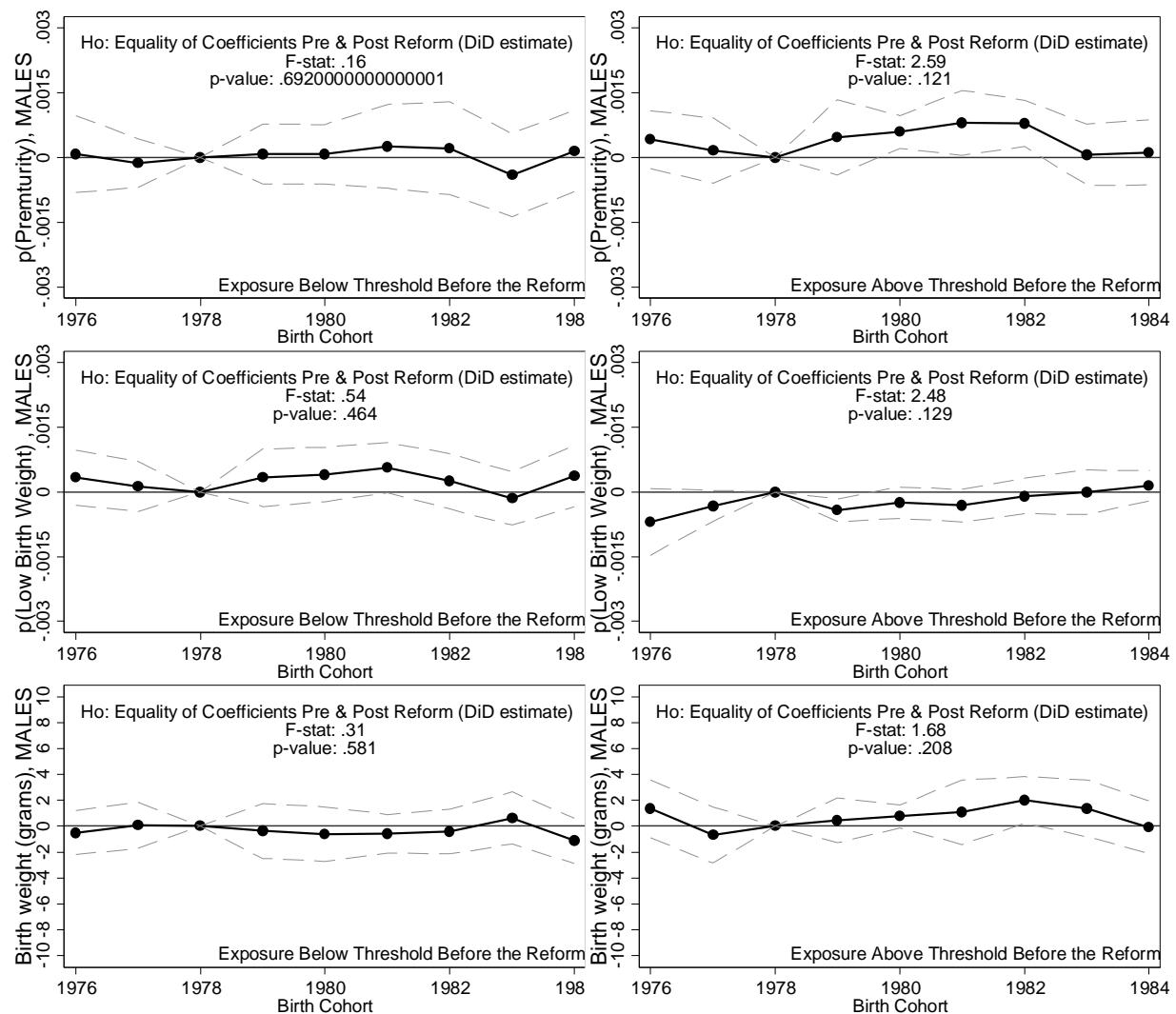


Figure E1. Reform effect on birth-weight, low-birth weight and prematurity (MALES)

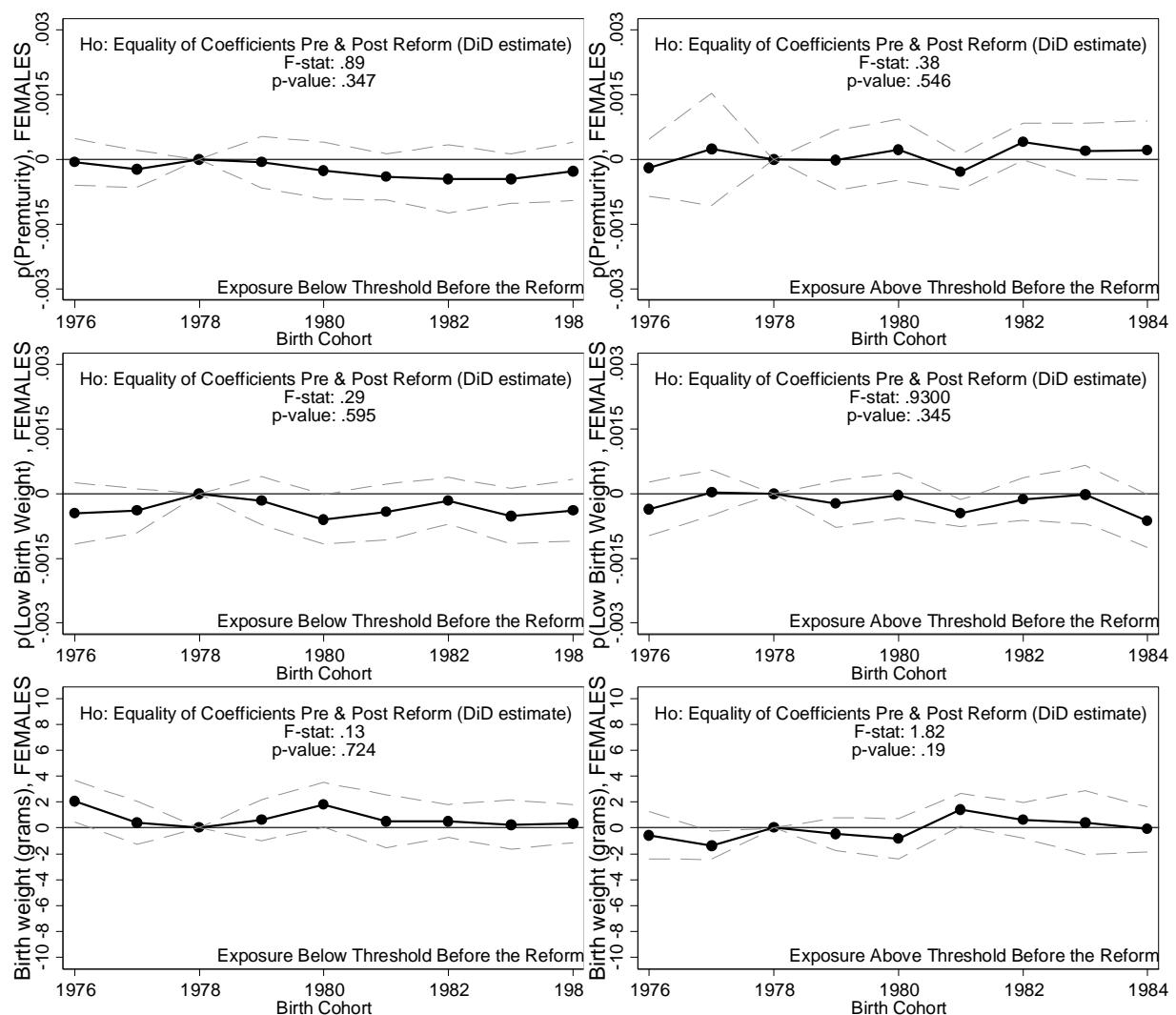


Figure E2. Reform effect on birth-weight, low-birth weight and prematurity (FEMALES)

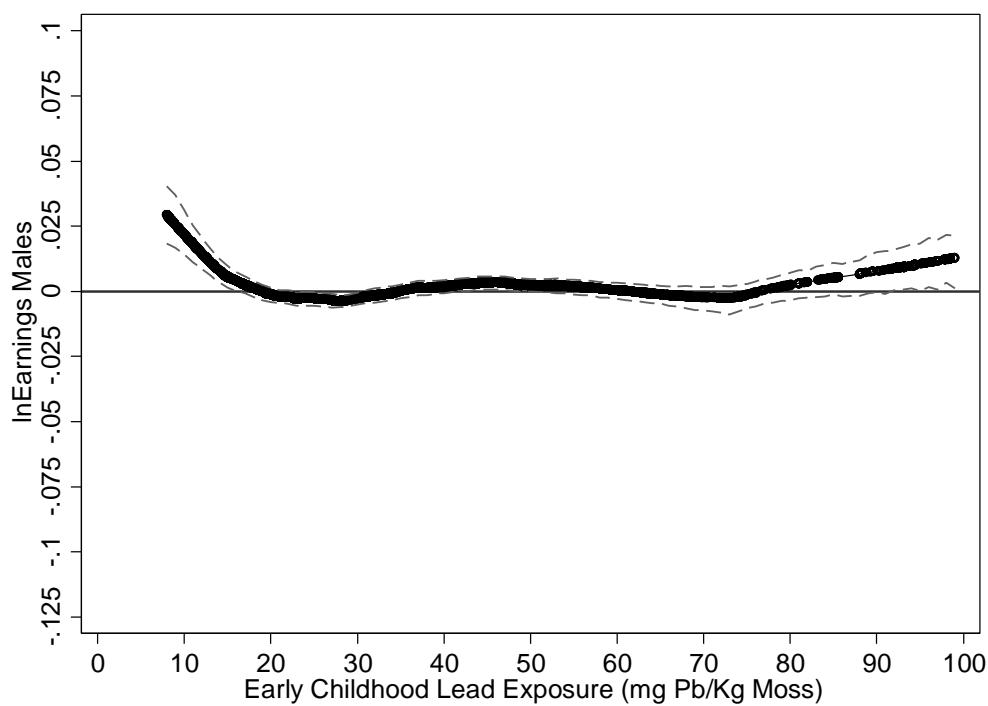


Figure E3. Semiparametric estimates on males $\ln(\text{Earnings in 2013})$

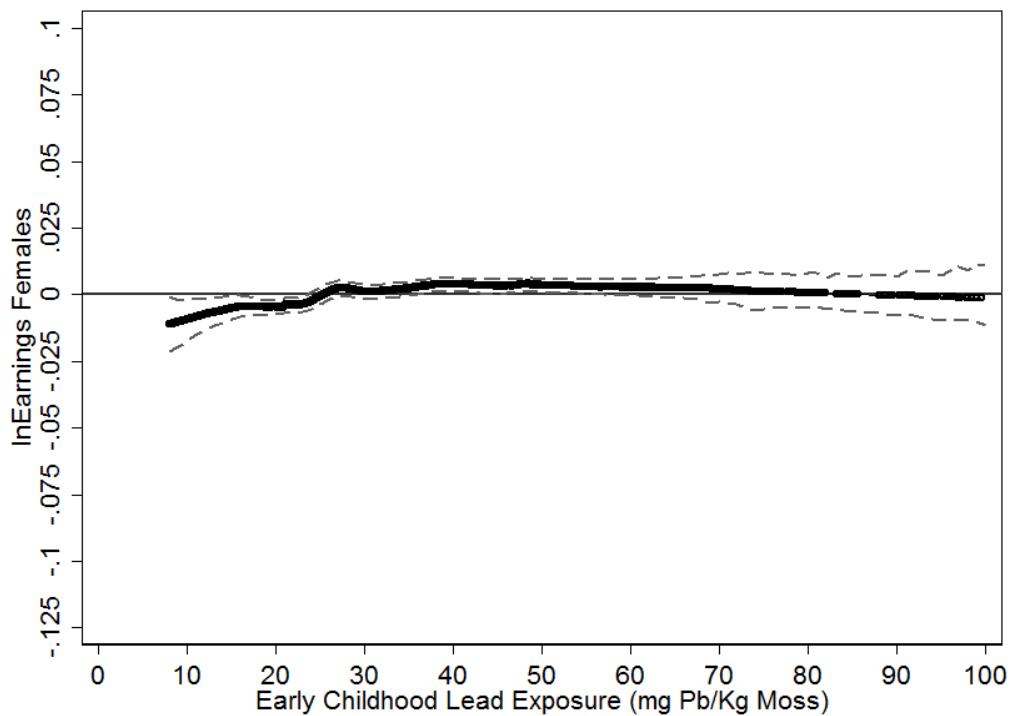


Figure E4. Semiparametric estimates on females $\ln(\text{earnings in 2013})$

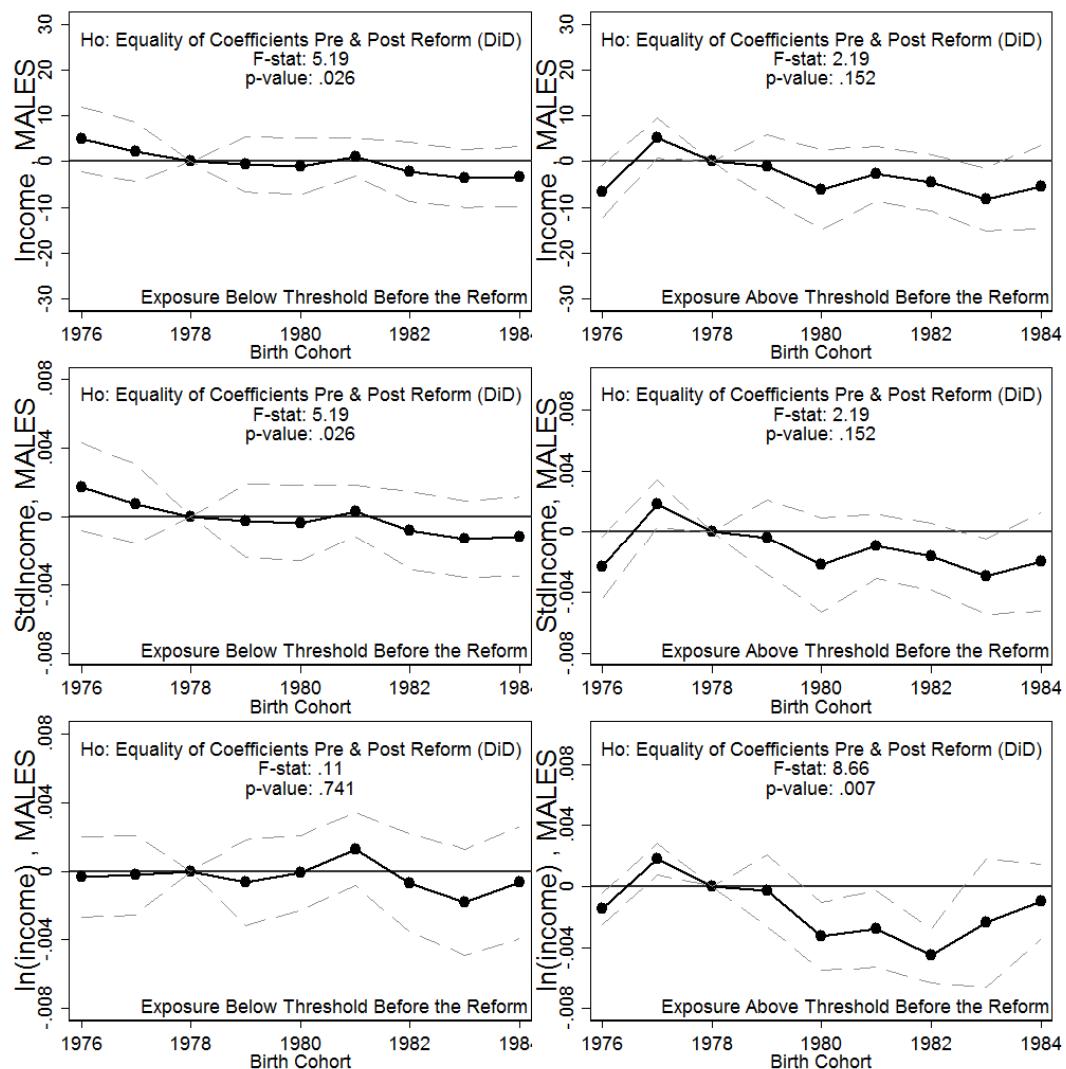


Figure E5. The estimated impact of the 1980/81 reforms on earnings in 2013 (MALES)

Notes: These figures show the point estimates, pointwise 95% confidence bands from the estimation of the cohort-specific γ 's from equation (2) 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-hand figure provides the estimates for Earnings/Standardized Earnings/ln(Earnings) among children born in neighborhoods with a lead level below 30 mg/kg just before the reform. The right-hand figure provides the same estimates for children born in neighborhoods with a lead level above 30 mg/kg just before the reform.

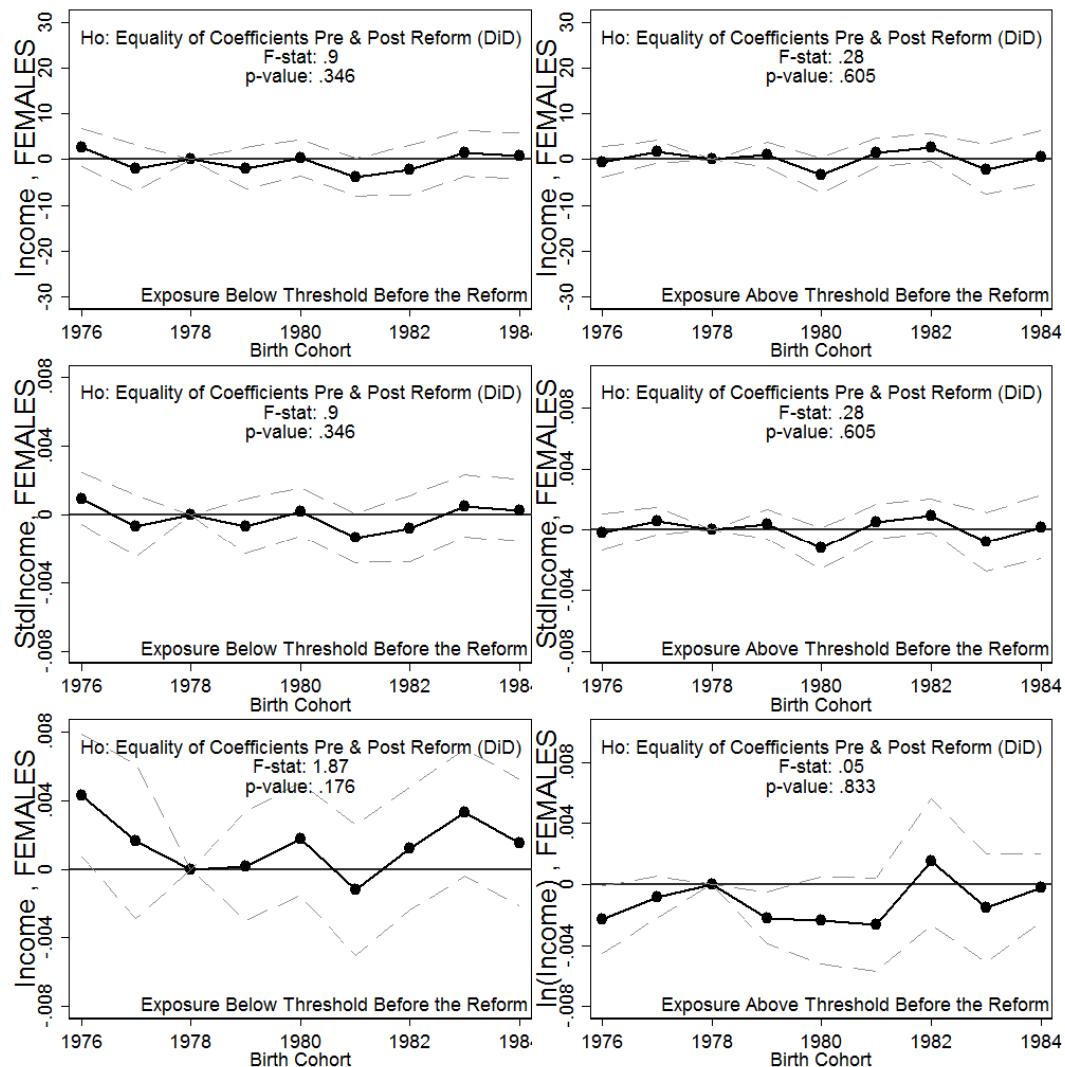


Figure E6. The estimated impact of the 1980/81 reforms on earnings in 2013 (FEMALES)

Notes: These figures show the point estimates, pointwise 95% confidence bands from the estimation of the cohort-specific γ 's from equation (2) 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-hand figure provides the estimates for Earnings/Standardized Earnings/ $\ln(\text{Earnings})$ among children born in neighborhoods with a lead level below 30 mg/kg just before the reform. The right-hand figure provides the same estimates for children born in neighborhoods with a lead level above 30 mg/kg just before the reform.

Table E1. The effect of earnings in 2013 (ages 29-40)

	ln (Earnings)	Earnings	Standardized earnings
<i>Panel A: Girls</i>			
Early lead exposure	.00036 (.00022)	.5202 (.3888)	.00019 (.00014)
Sample mean	7.54	2303.68	.824
<i>Panel B: Boys</i>			
Early lead exposure	.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. The coefficients reflect the effect of increasing lead by 1 unit. The sample consists of children born in 1973-1974, 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. */**/*** denote statistical significance at the 10/5/1 percent levels, respectively. See Table 4 for a full set of controls.

Table E2. Sibling fixed effect estimates

		Dependent variable:							
<i>Panel A:</i> <i>Above-threshold</i>	GPA	P(High school graduation)				Non-cognitive		Cognitive skills	
		OLS	Sibling FE	OLS	Sibling FE	OLS	Sibling FE	OLS	Sibling FE
Lead exposure	-.1558** (.064)	-.1361 (.1308)	-.0023*** (.0008)	-.0044*** (.0013)	-.0054* (.0030)	-.0062* (.0035)	.00071 (.0017)	.00084 (.0034)	

		Dependent variable:							
<i>Panel B:</i> <i>Below-threshold</i>	GPA	P(High school graduation)				Non-cognitive		Cognitive skills	
		OLS	Sibling FE	OLS	Sibling FE	OLS	Sibling FE	OLS	Sibling FE
Lead exposure	-.0127 (.0766)	-.0452 (.1477)	-.0005 (.0016)	-.0006 (.0021)	0.0022 (0.003)	.00344 (.0055)	-.00002 (.0031)	-.00015 (.0061)	

Notes: The table shows OLS-neighborhood fixed effects estimates and sibling fixed effect estimates of the effects of early lead exposure (mg/kg moss) for the sibling reform sample. The sample is restricted to males in families with at least two and at most three sons born in the observations window (1976-1984 cohorts) without missing values for any outcome. In the above human capital threshold sample, the number of sibships is 19,247 and the number of children is 39,370, in the below cut-off neighborhoods there are 13,606 sibships and 27,877 children. The standard errors are twoway-clustered at the CZ and maternal level. See notes to Table 5 for additional details of the specification used.

Web Appendix F:

Decomposing the effects of early lead exposure by source

Here we describe the mediation analysis used to construct Figure 11 in the main text.

Figure 11 provides a decomposition similar in spirit to that of Heckman, Pinto, and Savelyev (2013) for GPA, high school completion and the three crime outcomes. Using the enlistment data, we decompose the effects of lead exposure on later outcomes into the effects via the measured cognitive and non-cognitive skills and the effect via unmeasured skills. For GPA and high school completion data, we have individual measures of both outcomes and skills. In the crime data we do not have any access to data on individual skills, for the crime outcomes we do instead rely on the impact on average skills among males in the neighborhood-cohort of birth.

The bars in Figure 11 are produced in three steps:

- (1) For the top five bars, we regress each outcome on lead exposure and the standardized cognitive and non-cognitive tests from the military enlistment using the full sample linear specification. From this regression, we retain the β and the estimated coefficients for the cognitive (α_C) and the non-cognitive (α_{nc}) tests. For the two bottom bars, we use the reform framework and estimate an augmented version of the baseline event-study specification in the above threshold neighborhoods where we split the post-reform cohorts into a phase-in group and a full reform-group and also include the measures of cognitive and non-cognitive skills. The lower two bars use the full-reform (γ_{Full}) estimates of the reform. Using the reliability ratios estimated by Grönqvist, Öckert, and Vlachos (2010) (0.73 for cognitive, and 0.5 for non-cognitive skills), we then rescale the coefficients for cognitive and non-cognitive skills.
- (2) Second, we take the product of the rescaled estimates and the change in cognitive and non-cognitive skills induced by lead exposure/the policy, which provides the (measurement error

corrected) induced change in the outcome for these two inputs under the assumption that the measured skills are exogenous to the unmeasured skills (the “other factors”).

(3) We calculate the total effect as the sum of β (γ_{Full}) and the lead induced changes in the outcomes via the cognitive and non-cognitive skills using the panel data (reform analysis) as follows:

$$\begin{aligned}\beta + \frac{\widehat{\alpha}_c}{0.73} \Delta \text{Cognitive skill} + \frac{\widehat{\alpha}_{nc}}{0.5} \Delta \text{Noncognitive skill} &= \pi . \\ \gamma_{FULL} + \frac{\widehat{\alpha}_c}{0.73} \Delta \text{Cognitive skill} + \frac{\widehat{\alpha}_{nc}}{0.5} \Delta \text{Noncognitive skill} &= \pi .\end{aligned}$$

The respective bars for the contribution of e.g. the measured non-cognitive skills are then constructed by taking $\frac{\widehat{\alpha}_{nc}}{0.5} \Delta \text{NonCognitive skill}/\pi$.

For the linear panel-data estimates, the effects on cognitive skills estimated in step 2 are insignificant, small and with an unexpected sign. In these cases we set this coefficient to zero. For the reform estimates, the estimated effects on cognitive skills go in the right direction (see Heckman et al. 2013). For all specifications, the estimated α_i 's enter with the expected sign.

Table F1 on the next page provides the results necessary to produce Figure 11.

Table F1. Estimates used to construct Figure 11

		Outcome regressions when adding cognitive and non-cognitive skill measures					The estimates used to construct Figure 11				
Impact on cognitive	Impact on non- cognitive	Impact on outcome	Cognitive	Non cog.	Cognitive part (1×4)	Non cog part (2×5)	Total (3+6+7)	Share non cog (7/8)	Share cog (6/8)	Share residual (3/8)	
Outcomes:	1	2	3	4	5	6	7	8	9	10	11
<i>Panel Data Est.</i>	β_{OLS-FE}	β_{OLS-FE}	β_{OLS-FE}	$(\frac{\alpha_c}{0.73})$	$(\frac{\alpha_{nc}}{0.50})$						
GPA	.0002	-.0014	-.0279	17.04	7.018	0	-.0097	-.0376	.2589	0	.74
High School	.0002	-.0014	-.0005	.0775	.1085	0	-.00015	-.0006	.2444	0	.76
Any Crime	.00005	-.0016	.0003	-.0378	-.0475	0	.00008	.0003	.2413	0	.76
Property Crime	.00005	-.0016	.00014	-.0030	-.0376	0	.00006	.0002	.3089	0	.69
Violent Crime	.00005	-.0016	.00004	-.0085	-.0109	0	.00002	.00006	.3157	0	.68
<i>Reform Estimate</i>	β_{2SLS}	β_{2SLS}	β_{2SLS}	$(\frac{\alpha_c}{0.73})$	$(\frac{\alpha_{nc}}{0.50})$						
GPA	-.0015	-.0124	-.1789	19.59	8.382	-.029	-.1039	-.313	.3323	.096	.57
High School	-.0015	-.0124	-.0024	.0883	.144	-.0001	-.0018	-.004	.4180	.031	.55

Notes: Columns (1-2) are the estimated impact of the policy on cognitive and non-cognitive outcomes presented in Table 4. In Columns (3-5) each row represents separate regressions using Equation (1) for the top 5 rows, and equation (2) for the bottom two rows, augmented with the cognitive and non-cognitive test scores. The cognitive and non-cognitive coefficients from these regressions are then measurement error corrected using the reliability ratio estimated by Grönqvist, Öckert, and Vlachos (2013); 0.73 for cognitive and 0.5 for non-cognitive skills. Following Heckman et al. (2013), we set the contributions to zero when insignificant, small and of the opposite sign than what was expected. Note that the crime data is not linked to the enlistment data, instead we use the neighborhood averages of the skill measures to decompose the effects for the crime outcomes.