

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 noncognitive 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%.

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

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effects, which can even be fatal. Today, exposure leading to clinical symptoms is relatively rare, primarily because of 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 6,000–12,000 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 (Hanna-Attisha et al. 2016). The World Health Organization (WHO) reports that 40% of all children globally have elevated BPb levels.²

Lead exposure is believed to be particularly harmful to infants and toddlers because of their rapidly developing central nervous systems, increased exposure due to hand-to-mouth activities, and, conditional on exposure, 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 link relevant outcomes to exposure and at the same time are 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 United States (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.

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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 The CDC (US Centers for Disease Control and Prevention) established $5 \mu\text{g}/\text{dL}$ blood as the new reference value for elevated BPb levels 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.

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

While a recent literature documents negative causal effects on child outcomes (e.g., Reyes 2007, 2014; Ferrie, Rolf, and Troesken 2012; Billings and Schnepel 2015; Rau, Urzúa, and Reyes 2015; Gazze 2016; Aizer et al. 2018), little is known about the mechanisms through which childhood lead exposure maps into adult outcomes.⁴

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 3 decades later. The data contain measures of both cognitive and noncognitive skills, allowing 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 (EPA). Using several independent data sources, we verify that changes in local moss lead (MPb) levels and 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 the mid-1980s, when, as a result of the phaseout of leaded gasoline, lead levels per liter of gasoline were rapidly reduced by 80%. 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. Because of major differences in the initial lead levels, primarily due to preexisting differences in traffic density, the reforms

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% 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 National Longitudinal Survey of Youth. 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 from 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, North Carolina, and find that behavioral and educational deficits previously associated with high levels of early-life exposure can largely be reversed by intervention. Gazze (2016) exploits similar state mandates and finds that the share of children attending special education decreases. Aizer et al. (2018) use an individual-level data set 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. A series of recent studies have exploited historical data from the late nineteenth and early twentieth centuries to look at the importance of lead in water pipes for infant mortality, cognitive ability, and homicide (Troesken 2008; Ferrie, Rolf, and Troesken 2012; Clay, Troesken, and Haines 2014; Feigenbaum and Muller 2016).

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 the 1920s. Together with a low traffic density, this implies that the BPb levels before the phaseout of leaded gasoline were already much lower than those in the United States at the same time. We are thus able to document the effects of lead in a low-exposure setting. This contextual feature is particularly useful because it helps provide evidence about the effects at exposure levels that are still common in many countries, including the United States, 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 data sets, 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 prereform neighborhood lead exposure is a strong predictor of subsequent changes in neighborhood lead exposure. After the reforms, the exposure is equalized across neighborhoods, and the lead levels decrease drastically in neighborhoods with high prereform levels of exposure, while there are much smaller reductions across the cohorts born in areas with low prereform exposure. We also see that, compared to those of older peers born in the same neighborhoods, the relative academic achievements of children improve among those born in high-prereform exposure neighborhoods but are unchanged in low-prereform exposure neighborhoods. In the paper, we also show that a lower exposure to lead raises compulsory-school grade point average (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., Chetty et al. 2016; Conti, Heckman, and Pinto 2016; Nilsson 2017; Heckman and Karapakula 2019) and in 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 $\mu\text{g}/\text{dL}$. 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 (1) the timing of the changes in outcomes across birth cohorts

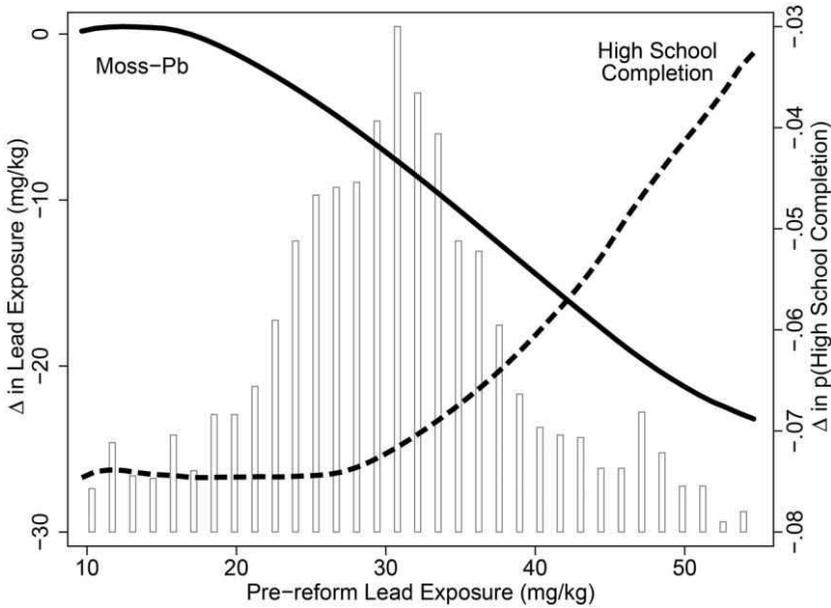


FIG. 1.—Prereform lead exposure versus changes in lead exposure and high school completion. The figure shows raw data changes in the neighborhood MPb level (left-hand y-axis) and changes in the probability of high school completion (right-hand y-axis) for males born between 1977–79 and 1982–84 against prereform neighborhood lead exposure (average during 1977–79) as measured in moss (mg/kg). The solid and dashed lines are local linear regressions of changes in lead exposure and high school completion, respectively, on prereform 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 prereform MPb levels in the neighborhood of birth (top and bottom 1% excluded).

corresponds well to the sharp reduction in lead consumption following the reforms, but (2) children living in neighborhoods below the exposure threshold were not affected by the reforms, whereas (3) the outcomes of children in neighborhoods above the exposure threshold improved significantly. To put our estimates into perspective, a one-unit decrease in BPb above the exposure threshold significantly increases the probability of completing high school by 4% among males.

Interestingly, in our low-exposure setting, we find no significant or consistent pattern with respect to cognitive skills. However, noncognitive 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% of the changes in human capital can be accounted for by the estimated effects of lead exposure on the measured noncognitive 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 noncognitive skills. The specific channels are of obvious importance for efficient remediating investments, for example, because cognitive and noncognitive skills differ in their malleability across a child's life cycle (Francesconi

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 BPb at 27 $\mu\text{g}/\text{dL}$ in the "low-exposure" group. Subsequent epidemiological studies have detected cognitive and behavioral deficits at ever lower levels of exposure (e.g., Banks, Ferretti, and Shucard 1997; Canfield et al. 2003). 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, PA; Dietrich et al. 2001 examine 195 inner-city adolescents from Cincinnati, OH; 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; Hebben 2001; Kaufman 2001a, 2001b; 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 $\mu\text{g}/\text{dL}$. The cohorts studied in Reyes (2007) were estimated to have a BPb level of between 10 and 20 $\mu\text{g}/\text{dL}$. The average predicted childhood blood level in Reyes (2014) is about 7.6 $\mu\text{g}/\text{dL}$. Gazzo (2016) examines the impact on the share of children with special education needs following reductions in the share of children with BPb greater than 10 or 20 $\mu\text{g}/\text{dL}$. Rau, Urzúa, and Reyes (2015) study the effects on academic achievement and earnings where the subjects' BPb levels were, on average, 10 $\mu\text{g}/\text{dL}$, while Aizer et al. (2018) study the effects of low exposure (<10 $\mu\text{g}/\text{dL}$), 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 $\mu\text{g}/\text{dL}$ in 2002 to less than 3 $\mu\text{g}/\text{dL}$ in 2018.

and Heckman 2016).⁶ Our results suggest that policies targeting noncognitive 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 II discusses our data and shows how air lead exposure is linked to children's BPb levels. Section III describes our research design. Section IV presents the empirical results. In section V, we interpret our findings and decompose the effects of early lead exposure by source, and section VI 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 phaseout 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.

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 (apps. A–F are available online) summarizes the evidence in the medical literature, in particular the specific mechanisms that affect development.⁸ The major lessons learned from this review are as follows. (1) 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. (2) BPb levels

⁶ Our finding that noncognitive 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 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. 2011, 2016). Moreover, Almlund et al. (2011) show that neuroticism and conscientiousness are particularly strongly related to externalizing behavior, and Heckman, Pinto, and Savelyev (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; Luechinger 2014; Isen, Rossin-Slater, and Walker 2017; Jans, Johansson, and Nilsson 2018).

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

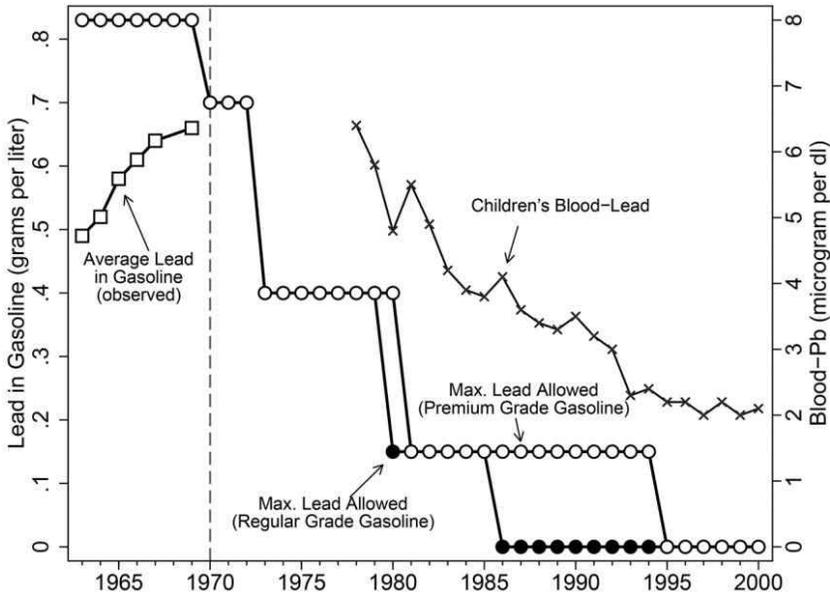


FIG. 2.—Changes in lead levels in Sweden by type of gasoline 1970–2000 and mean BPb levels among elementary school children in Sweden 1978–2000. From left to right, the figure shows the observed average lead levels in gasoline for the period 1963–67 and 1969 (squares; see Danielson 1970), the timing of the first reform (vertical line), the maximum allowed lead content in gasoline for premium-grade (open circles) and regular-grade (filled circles) gasoline; before 1980, the regulation was the same for all grades (data from the Swedish Petroleum Institute). The right-hand-side y-axis shows the BPb level of ~120 elementary school children per year (see Strömberg, Schütz, and Skerfving 1995 and Strömberg 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.

follow an inverted v-shaped pattern between the ages of 6 and 60 months, reaching its peak at the age of 24 months because of the intense hand-to-mouth activity at this age. (3) 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 (functional magnetic resonance imaging) 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

⁹ However, as regards 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 ($>10 \mu\text{g}/\text{dL}$).

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%. 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.

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 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 biomonitors of 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 have participated 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 depends solely 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 Biomonitors have 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.

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 1972–74, 1977–79, and 1982–84. The selection of these years is made for two

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

¹¹ The sampling sites have been chosen with care; they should be located at least 300 m away from major roads and closed residential areas or at least 100 m from smaller roads and single houses. At each sampling site, 5–10 subsamples are collected in an area of 100 m².

reasons. First, the maximum concentration of lead in gasoline permitted decreased sharply in 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 which 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 US Census block groups.

There is a great deal of variation in lead levels between neighborhoods. Figure 3 presents maps for neighborhood lead concentrations in 1975 (*left*) and 1985 (*right*), 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

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.

¹³ The between- and within-neighborhood standard deviations (SDs) are 10.78 and 14.35, respectively.

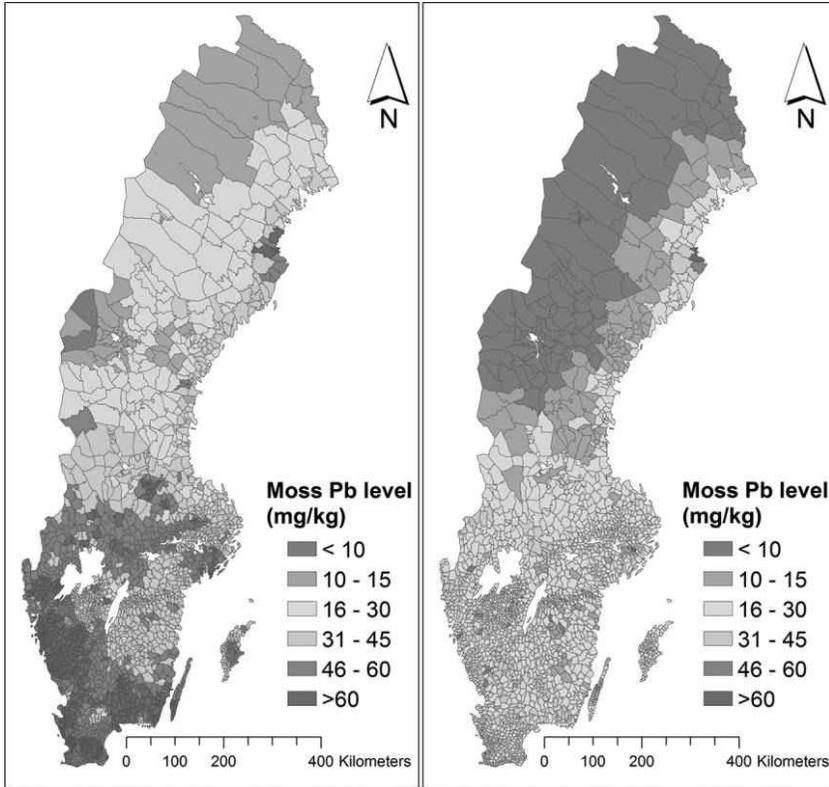


FIG. 3.—Neighborhood-of-birth lead exposure levels in 1975 (*left*) 1985 (*right*). A color version of this figure is available online.

the important link between MPb and children's BPb. Finding that MPb levels are uncorrelated with BPb levels would cast doubt 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 and the estimation procedure and gives additional robustness checks.

As noted above, there are no comprehensive data with measures of BPb available in Sweden. However, since 1978, venous blood samples have been collected from about 120 primary school children (between

¹⁴ That said, the two measures partly capture different aspects of exposure. While BPb level, 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.

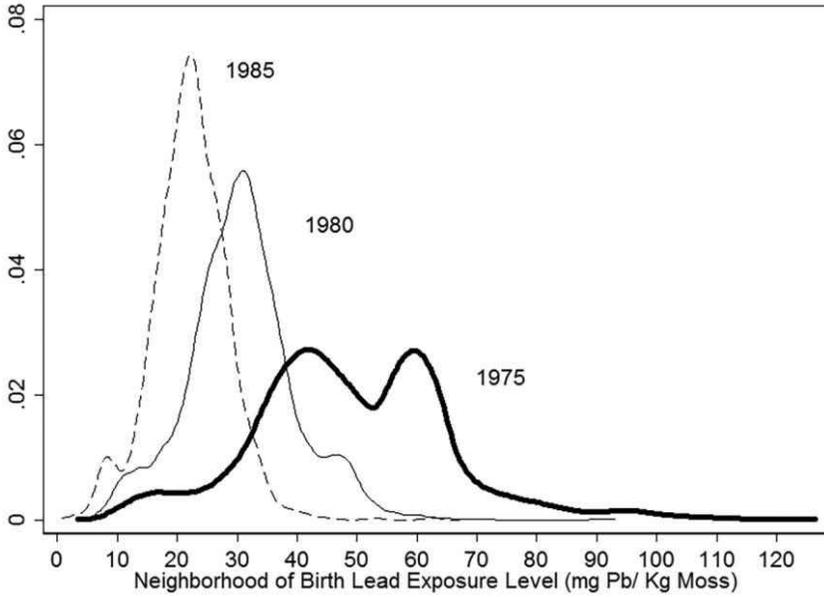


FIG. 4.—Changes in neighborhood MPb exposure for the cohorts included in the main analysis.

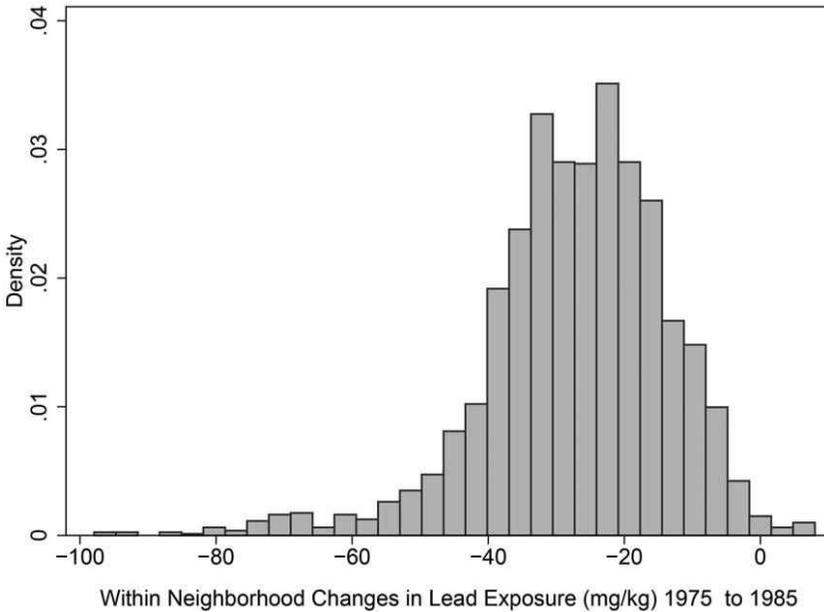


FIG. 5.—Within-neighborhood changes in lead exposure, 1975–85.

8 and 10 years old) 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 data sets from Landskrona provide an opportunity to assess the strength of the relationship between our measure of local air lead exposure and children's BPb. Panel A of table 1 reiterates and extends the findings in Nilsson et al. (2009), which link 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 preban period of 0.47. This implies that a 10% reduction in MPb corresponds to a 4.7% 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% of the change in children's BPb levels.¹⁶

That said, the BPb data have limitations in terms of 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 as a result of 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% higher than that among children aged 6–12. In addition, there are several

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, e.g., the United States at the end of the 1970s and the United Kingdom in the early 1980s). Second, lead water service pipes are a nonissue 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 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 other than airborne lead that may also have contributed to the BPb declines 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 phaseout of leaded gasoline.

TABLE 1
LEAD EXPOSURE AND CHILDREN'S BPb LEVELS

SPECIFICATION	DEPENDENT VARIABLE			
	ln (BPb) (1)	ln (BPb) (2)	ln (BPb) (3)	ln (BPb) (4)
A. Relationship between MPb and Children's BPb Levels ($\mu\text{g}/\text{dL}$) in Landskrona				
ln(MPb)	.4861*** (.1191)	.4774*** (.1177)		.4957*** (.1196)
ln(MPb) \times Girl				-.0556 (.0642)
Neighborhood fixed effects	Yes	Yes		Yes
Year fixed effects	Yes	Yes		Yes
Individual controls	No	Yes		Yes
R^2	.56	.58		.58
No. of children	242	242		242
B. Impact of the 1980–81 Reforms on Children's BPb Levels ($\mu\text{g}/\text{dL}$) in Trelleborg				
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^2	.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

NOTE.—Panel A reports OLS estimates from eq. (B1). BPb is measured in (ln) $\mu\text{g}/\text{dL}$ blood and 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 reforms 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 (prereform, phase-in, and postreform periods, as defined in text), and year fixed effects are indicator variables for the year when BPb was sampled. Robust standard errors are reported in parentheses.

** Significant at the 5% level.

*** Significant at the 1% level.

potentially important layers of measurement problems (mapping moss observations to neighborhoods and 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 MPb levels predict BPb 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 $0.4 \mu\text{g}/\text{dL}$) 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 prereform exposure levels, which to a large extent were caused by preexisting population/traffic density, it is important to examine also whether children's BPb levels were 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 reforms in 1980–81 on children's BPb using data collected from Trelleborg municipality by Strömberg et al. (2003).¹⁷ 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 line with circles in figure 6 shows the total maximum gasoline lead sales (in metric tons) around the time of the reforms.¹⁸ The two other lines show local average BPb levels reported separately for children residing in urban areas and those in rural areas before, during, and after the implementation of the reforms. The three time series in figure 6 follow a strikingly similar pattern.¹⁹ Before the reforms, the BPb levels in urban Trelleborg averaged $6 \mu\text{g}/\text{dL}$. 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 reforms or in the years following the reforms. Among rural children, the BPb levels also decreased following the reforms, but not as sharply. After the full implementation, the BPb levels of urban and rural children leveled off and converged at around $4 \mu\text{g}/\text{dL}$.

¹⁷ Trelleborg is one of the southernmost municipalities in Sweden (i.e., one of the most densely populated regions in the country). It has one of the highest lead exposure levels in the country 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 for the reforms (SOU 1979, 35).

¹⁹ We follow Strömberg, Schütz, and Skerfving (1995) and apply a 2-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.

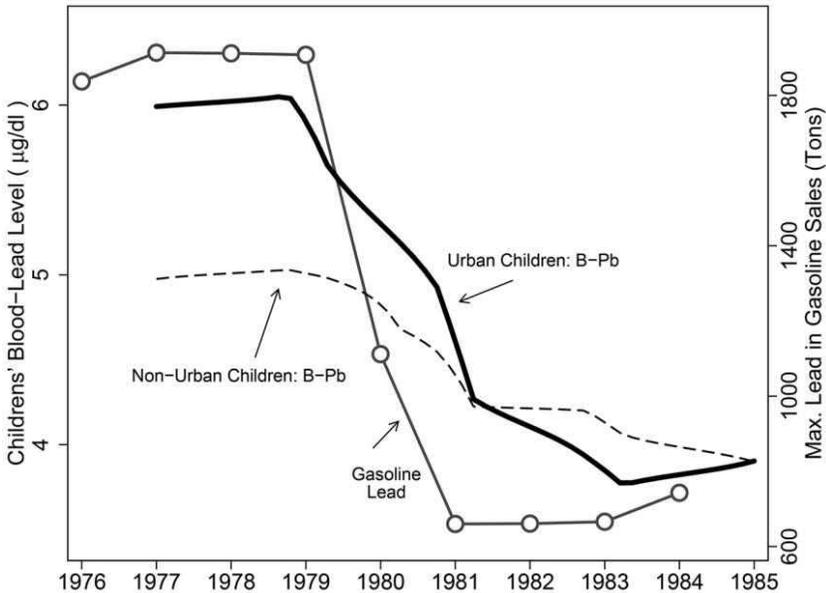


FIG. 6.—Impact of the 1980–81 reforms on children’s BPb levels. The figure shows BPb levels among primary-school children in Trelleborg before, during, and after the 1980–81 reforms (data from 1977–85). The solid line shows the average BPb levels of the children residing in the urban areas and the dashed line those of children in the nonurban 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 nonurban sample with an extreme BPb level (4 [2.3] times higher than the 50th percentile [95th percentile] 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 1976–84 (line with circles) 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 fig. 2) in the respective years. For medium-grade fuel (which is a blend of regular and premium gasoline) in the year 1980, we use the average maximum lead content allowed in regular and premium gasoline, since the maximum allowed lead content in 1980 differs between premium and regular gasoline.

Panel B of table 1 provides difference-in-differences estimates, comparing the impact of the reforms on BPb in urban versus 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 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 (1980–81) and full-reform (1982 and later) periods, and column 3 shows results from the same specification after addition of a year of blood sampling fixed effects. In line with figure 6, the impact of the reforms is significantly stronger among urban children, and the effect increased as the reforms were rolled out (–27%). Column 4

shows that the reforms had 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 before the year in which the moss samples were collected, that is, all those born in 1973–74, 1977–79, and 1982–84.²⁰ The moss samples measure the local lead deposition at ages 1–3 (e.g., mosses collected in 1975 reflect the lead levels in 1972–74), 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 were born in Sweden, so that their neighborhood of birth (and hence, their childhood lead exposure) is known. Sweden is one of the OECD (Organisation for Economic Co-operation and Development) countries with the highest age for university graduates (the average age is 29), and more than 40% of each birth cohort enroll in college (OECD 2014). Hence, we focus on outcomes that we are able to measure at the same age for all cohorts. We measure educational outcomes using the compulsory-school GPA (age 16, percentile ranked within graduation cohorts) and the probability of graduating from high school (age 19).

Criminal behavior is measured using data on criminal convictions available between 1985 and 2008, provided by the National Institute for Crime Prevention (Swedish acronym: BRÅ). The data include 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

²⁰ The data originate from three distinct databases at the Institute for Labor Market Policy Evaluation (IFAU), the Department of Economics at Uppsala University, and the Institute for International Economic Studies (IIES) at Stockholm University. While all the databases draw from the same population-based registers at Statistics Sweden, 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.

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

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 noncognitive skills measured at military enlistment. All Swedish men were obliged by law to participate in the military draft. Around 90% 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 similar to the Armed Forces Qualification Test in the United States. The test assessed the subject's cognitive ability on the basis of 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 1–9 scale. We standardize the score within each cohort of draftees to account for minor changes in the tests over time.²³

The noncognitive score is based on a 20–25-minute interview with a psychologist resulting in four different scales, all ranging from 1 to 5, here displayed with their respective subcategories and the Big Five personality traits²⁴ to which they are related: (1) psychological energy (perseverance [C], ability to fulfill plans [C], and ability to remain focused [C]); (2) emotional stability (ability to control and channel nervousness [N], tolerance of stress [N], and disposition to anxiety [N]); (3) social maturity (extraversion [E], having friends [E], taking responsibility [C], and independence [O]); and (4) intensity (capacity to activate oneself without external pressure [C] and intensity and frequency of leisure activities [O]). Using principal component analysis (PCA), we combine the subscores into a general measure of noncognitive ability and use it as our main measure of noncognitive ability.²⁵

²² In principle, only those with a physical or intellectual disability were exempt from the enlistment tests. As a result of 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 noncognitive skills in the analysis of the 1980–81 reforms.

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

²⁴ Openness to experience (O), conscientiousness (C), extraversion (E), agreeableness (A), and neuroticism (N).

²⁵ The correlation between the noncognitive index and the noncognitive 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 noncognitive skills. The PCA gives approximately the same weight to the different noncognitive subskills. The correlation between the noncognitive skills index and cognitive skills is 0.3. The correlation between cognitive (noncognitive) skills and ninth-grade GPA is 0.64 (0.4). Nilsson (2017) provides an account of how the military noncognitive subscores relate to the Big Five traits of personality (see Bihagen, Nermo, and Stern 2012). Lindqvist and Vestman (2011) relate the cognitive and noncognitive skills to labor market outcomes.

Table 2 provides summary statistics for the main analysis sample. For example, we can see that 79% of the children in our sample completed high school and that a large proportion of the children, 16.4%, have been convicted at least once by the age of 24. The number is well in line with other studies using data from the same sources (e.g., Grönqvist, Öckert, and Vlachos 2017). The average child grew up in a neighborhood with MPb exposure equal to 34 mg/kg, and around 40% 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 semiparametric fixed effects 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 that reduced the maximum lead content from 0.4 to 0.15 g/L. We describe the different approaches below.

A. Analyses

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:

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

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 milligrams per kilogram of 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

TABLE 2
DESCRIPTIVE STATISTICS AND DEFINITIONS FOR KEY VARIABLES

Variable	Definition	Mean	Standard Deviation
Outcomes:			
GPA	Compulsory-school grade point average ^a	50	28
Cognitive skills	Cognitive test score at military enlistment ^b	0	1
Noncognitive skills	Noncognitive test score at military enlistment ^b	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
Lead exposure:			
MPb level	Neighborhood-of-birth MPb (mg/kg; see text)	34	17
Additional variables:			
Share males		.51	.49
Birth order		1.9	.98
Mother's age at birth (years)		27.0	5.0
Birth weight (g)		3,482	561
Parents' education	1 if at least 1 parent completed college, 0 otherwise	.41	.49
Employment rate	No. of employed/total population (in municipality of birth)	.48	.12
Zn exposure	Neighborhood-of-birth moss zinc 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 children ($N = 699,155$) included in the full-sample analysis.

^a Percentile ranked within graduation cohort; typically at age 16.

^b Standardized within enlistment year cohort; age 18, males only.

for both spatially and serially correlated errors within the 74 CZs.²⁶ The quantity $\hat{\beta}^{\text{OLS}}$ (where OLS = ordinary least squares) provides the effect on adult outcomes of an additional unit of lead exposure (mg/kg) in early childhood.

²⁶ There are 2,559 neighborhoods in our data. Statistics Sweden divided these into 74 CZs. 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 because clustering at the CZ level also accounts for an arbitrary correlation in the errors across neighborhoods within the same CZ.

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 that 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 neurocognitive development or behavior.²⁸ The literature that attempts to address the 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 semiparametric fixed effects models (see Baltagi and Li 2002; Libois and Verardi 2013) that allow the exposure-response function $f(\text{Lead}_{ic})$ to be estimated in a fully flexible way. The semiparametric 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

²⁷ For instance, encephalopathy, seizures, and coma ($>70\text{--}80\ \mu\text{g}/\text{dL}$), renal (kidney) failure ($50\ \mu\text{g}/\text{dL}$), and hemoglobin disruption ($25\ \mu\text{g}/\text{dL}$; ATSDR 2007).

²⁸ The extent to which this is due to the limitations of the cross-sectional research designs, the fact 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 (see 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, 118). A potential explanation is that the composition of children exposed to low 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. Since the phaseout, only a share of the children have been exposed, namely, children living in low-standard housing with deteriorating water pipes and/or 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 and 20 $\mu\text{g}/\text{dL}$.

³⁰ The nonparametric exposure-response function, $\hat{f}(\cdot)$, is estimated using local linear regression, and we present the nonparametric slope together with bootstrapped 95% confidence intervals (1,000 repetitions). Specifically, we estimate a narrow-bandwidth (0.3), robust local linear regression model (LOWESS), using Cleveland’s tricube 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.

Both the linear and semiparametric fixed effects models rest on the same key underlying assumptions: unobserved determinants of long-run outcomes should not covary 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 II highlights a potentially important factor: the 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 (see 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 before the 1980–81 reforms.³² Hereafter, we sometimes simply refer to this as the “reform” analysis. The model, estimated by ordinary least squares (OLS), is specified as follows:

$$Y_{\text{inc}} = \sum_{c=1976}^{1984} (\lambda_c \times \text{Lead}_n^{\text{Prereform}}) \cdot \gamma_c + \theta X_{\text{inc}} + \delta Z_{\text{inc}} + \lambda_c + \lambda_n + \varepsilon_{\text{inc}}, \quad (2)$$

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 effects 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 understate 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 prereform period, which we use to examine prereform 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 prereform measure of exposure (1977–79). The 1973 reform is not as useful, since the closest MPb exposure measure captures a mix of pre- and postreform levels of exposure (1972–74). The 1986 reform is less useful because it targeted only regular-grade gasoline, and the changes in lead exposure are much more gradual than those following the 1980–81 reforms, which targeted all grades.

where $\text{Lead}_n^{\text{Prereform}}$ is the (predetermined) MPb level just before the reforms in neighborhood n . To map out the changes in outcomes for cohorts born around the time of the reforms, we interact prereform 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 Again, 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 MPb exposure data, and we estimate this model using data on all cohorts 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 first-stage effects of the reforms. To implement the 2SLS analysis, we therefore divide the children into three groups based on cohort of birth: the prereform (1976–78), phase-in (1979–81), and full-reform (1982–84) cohorts. We then interact the phase-in and full-reform dummies with the prereform MPb level in the neighborhood of birth and estimate a system of equations of the following form:

$$\begin{aligned} \text{Lead}_{\text{nc}} &= \pi_1 (\text{FullReform}_c \times \text{Lead}_n^{\text{Prereform}}) \\ &\quad + \omega_1 X_{\text{inc}} + \omega_2 Z_{\text{mc}} + \rho_c + \rho_n + \tau_{\text{inc}}, \quad (3) \\ Y_{\text{inc}} &= \delta \cdot \widehat{\text{Lead}}_{\text{nc}} + \theta X_{\text{inc}} + \delta Z_{\text{mc}} + \lambda_c + \lambda_n + \varepsilon_{\text{inc}}, \end{aligned}$$

where the excluded instrument is the interaction term between the full-reform cohort dummy, FullReform_c , and the prereform MPb level in the

³³ Gasoline lead sales are useful, since we do not have any direct measures of the other traffic-related pollutants (Cu, Zn, and Cd) for the 1980–82 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 CZ level aligns the analysis with the baseline (cohort-by-neighborhood) panel data specification.

neighborhood of birth, $\text{Lead}_n^{\text{Prereform}}$.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 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.

B. Basic Assessments of 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 covary 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 bicentennial censuses. Figure C1 (figs. B1, C1–C5, D1–D8, and E1–E6 are available online) 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 hold 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 changes nearly as great and widespread as lead during the observation period. Cd has previously been found to be associated with adverse health outcomes

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

(kidney damage, bone disease).³⁶ The data also include information on levels of copper (Cu) and zinc (Zn). 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 show in the next section, the estimates are similar when these key pollutants are controlled for, 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 change 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

³⁶ Early Cd exposure has been shown to produce neurotoxic effects in laboratory experiments (Andersson et al. 1997; Petersson-Grawé et al. 2004), and in utero exposure to Cd may affect infant health in humans (Currie and Schneider 2009). Air Cd and air lead 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 the estimates for lead 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 Cd 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, Bergbäck, and Häggerud 2007; Johansson, Norman, and Burman 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, and they provide better access to high-quality child care, schools, health care, and other amenities that are positively associated with child outcomes. Such local amenities could, in turn, e.g., via gentrification, understate the role of childhood lead exposure.

³⁹ These parental characteristics can be considered as predetermined in the present context, since more than 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, Rühling and Tyler (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 toward 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 as a result of 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 leaded gasoline. Exactly the same pattern is found in a study by Renberg et al. (2000). They use extraordinary data on lead levels in lake sediments to examine regional trends in lead depositions in Sweden over a period of 4,000 years. In particular, the lead concentrations in the lake sediments increased by 50% between 1960 and the peak year of 1970. Hence, the parents of the children were exposed to relatively low levels during their own childhood, and therefore the lead levels during the parents' childhood are not expected to influence the children's outcomes as adults.

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 prebirth 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 fixed differences across neighborhoods are taken 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 those in panel B are statistically distinguishable from zero and that parents' baseline socioeconomic 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 predetermined factors influencing children's long-term outcomes (parents' earnings, age, and level of education) are, at least in the setting 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 prereform lead exposure evolved across birth cohorts. Several heterogeneity and robustness checks follow.

TABLE 3
LINK BETWEEN PREDETERMINED PARENTAL CHARACTERISTICS AND THEIR CHILDREN'S LEAD EXPOSURE

	Parents Completed College	Mother Completed College	Father Completed College	Parents' Prebirth Earnings (log)	Father's Prebirth Earnings (log)	Mother's Prebirth Earnings (log)	Mother's Age at Birth
A. Cross-Sectional Correlation							
Lead exposure (mg/kg)	-.005 (.004)	-.004 (.004)	-.003 (.004)	.004 (.003)	.004 (.003)	-.001 (.006)	-.004 (.029)
Neighborhood fixed effects	No	No	No	No	No	No	No
B. With Neighborhood Fixed Effects							
Lead exposure (mg/kg)	.001 (.001)	.001 (.001)	-.0001 (.001)	.003 (.003)	.003 (.002)	.003 (.001)	-.014 (.028)
Neighborhood fixed effects	Yes	Yes	Yes	Yes	Yes	Yes	Yes

NOTE.—The table presents the coefficients from OLS regressions of baseline parental characteristics measured 2 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 parentheses) are clustered at the CZ level.

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 control for only 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

TABLE 4
PANEL DATA ESTIMATES OF THE EFFECT OF CHILDHOOD LEAD
EXPOSURE FOR THE FULL SAMPLE

	Grade 9 GPA (Percentile Rank) (1)	<i>P</i> (high school completion) (2)	<i>P</i> (ever convicted) (3)	<i>P</i> (property crime) (4)	<i>P</i> (violent crime) (5)
Baseline	-.1890** (.0961)	-.0034** (.0014)	.0014** (.0007)	.0007 (.0008)	.0004** (.0002)
+ Individual and 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 fixed effects	Yes	Yes	Yes	Yes	Yes
Year-of-birth fixed effects	Yes	Yes	Yes	Yes	Yes

NOTE.—The table presents the coefficient on early lead exposure from separate OLS regressions. The coefficients and standard errors are scaled by a factor of 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 1973–74, 1977–79, and 1982–84. Crime (in cols. 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 controls for only 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 2 years before birth (in 1985 for crime outcomes), and birth-order dummies. Municipality controls include population size and employment rate from the 1975, 1980, and 1985 censuses. Other traffic-related pollutants include cadmium, zinc, and copper. The sample means are shown in brackets. The individual data are aggregated into 2,559 neighborhoods of birth that are divided into 74 CZs 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 parentheses. The preferred (full) specification is in boldface.

** Significant at the 5% level.

*** Significant at the 1% level.

neighborhood fixed effects 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 (1) estimated regressions controlling for area-specific trends, (2) excluded the most populated areas, and (3) allowed for initial population density-specific trends. In line with the results from section III.B, 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.

1. 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 II, 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

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, Lubotsky, and Paxson 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

environmental policies affects adult nonhealth 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.

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

2. Effects on Cognitive and Noncognitive 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 BPb 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 PFC. PFC activity, in turn, is strongly correlated with noncognitive traits such as conscientiousness and grit.

However, previous research has not quantified whether cognitive or noncognitive skills are most important when accounting for the effects on later outcomes or whether the different skills respond more strongly

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 (see Neidell 2004). An 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 for the impact of lead on subsequent outcomes. Finally, since children from poorer backgrounds more often suffer from other health problems, an SES gradient on the effect of early-childhood lead exposure could result in interactive effects of lead exposure and other health problems (see Currie et al. 2010; Jans, Johansson, and Nilsson 2018). It would be interesting, although it is not possible to do so here, to fully differentiate the relative importance of the competing underlying mechanisms 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 2 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).

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. Panel F of table 5 provides the linear panel data estimates for cognitive and noncognitive skills in the full sample and separately by parental income. There is a significant negative relationship between early lead exposure and noncognitive skills but not one 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 a “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 nonparametric estimates of the slopes for noncognitive (*top*) and cognitive (*bottom*) skills. For noncognitive 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 that is similar to the shape for noncognitive 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 toward a nonlinear pattern at the bottom of the distribution (~20 mg/kg), but we cannot reject linearity. Figure C2 provides the same estimates for females. For females, there is a tendency toward 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 pattern similar to that for high school completion, but the relationship

⁴² We also examine the effects on the four subscores used to construct the noncognitive index. Semiparametric estimates for each of the subscores are presented in fig. 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.

TABLE 5
 HETEROGENEITY AND EFFECTS ON COGNITIVE AND NONCOGNITIVE SKILLS

	Grade 9 GPA (Percentile Rank) (1)	<i>P</i> (high school completion) (2)	<i>P</i> (ever convicted) (3)	<i>P</i> (property crime) (4)	<i>P</i> (violent crime) (5)	(6)
				A. Full Sample		
Lead exposure (mg/kg)	-.2097** (.0925)	-.0031** (.0014)	.0018** (.0008)	.0009 (.0009)	.0002 (.0003)	
Sample mean	50.72	.79	.164	.070	.025	
				B. Girls		
Lead exposure	-.1452 (.1110)	-.0011 (.0015)	.0004 (.0007)	.0001 (.0006)	.0000 (.0001)	
Sample mean	56.69	.808	.079	.045	.006	
				C. Boys		
Lead exposure	-.2773*** (.0989)	-.0051*** (.0019)	.0029** (.0013)	.0017 (.0014)	.0006 (.0005)	
Sample mean	44.85	.769	.244	.095	.042	

	D. Low-Income Parents		E. High-Income Parents		F. Effects on Cognitive and Noncognitive Skills (Boys)			
Lead exposure	-.2640** (.1170)	-.0037* (.0019)	.0017 (.0011)	.0014 (.0011)	Noncognitive High income	Noncognitive Low income	Cognitive High income	Cognitive Low income
Sample mean	45.39	.739	.191	.087	-.016** (.0007)	-.012 (.0007)	.004 (.0035)	-.005 (.0005)
Lead exposure	-.2120** (.1020)	-.0027** (.0013)	.0015* (.0007)	.0004 (.0007)	Noncognitive High income	Noncognitive Low income	Cognitive High income	Cognitive Low income
Sample mean	55.38	.834	.136	.054	-.016** (.0007)	-.012 (.0007)	.004 (.0035)	-.005 (.0005)

NOTE.—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 2 years before 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 table 4 note. Cluster robust standard errors (at the CZ level [74 cells]) are shown in parentheses.

* Significant at the 10% level.

** Significant at the 5% level.

*** Significant at the 1% level.

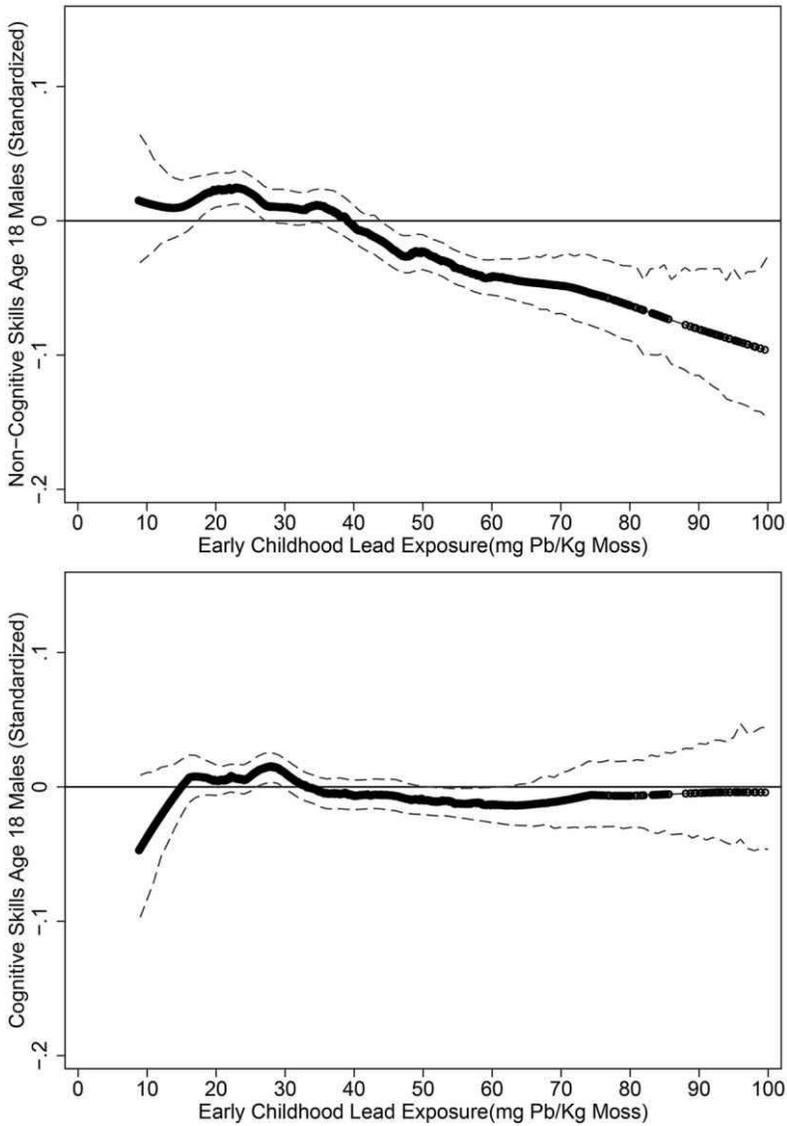


FIG. 7.—Semiparametric fixed effects estimates (see Baltagi and Li 2002) for noncognitive and cognitive skills, with 95% bootstrapped confidence intervals (1,000 repetitions). The dependent variable in the top graph is noncognitive skills, and that in the bottom graph is cognitive skills, as measured at age 18 at military enlistment. See table 4 for the full set of controls.

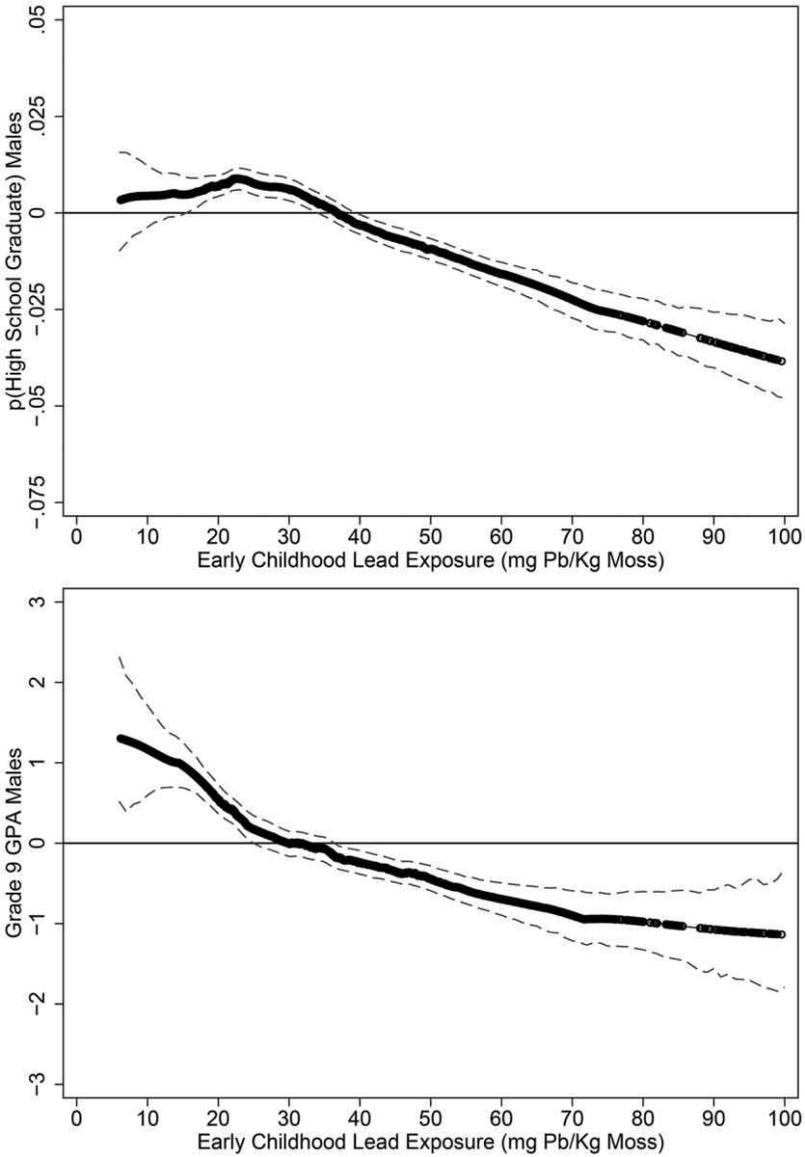


FIG. 8.—Semiparametric fixed effects estimates (see Baltagi and Li 2002) for human capital (males), with 95% bootstrapped confidence intervals (1,000 repetitions). The dependent variable in the top graph is the share that completed high school, and that in the bottom graph is GPA. See table 4 for the full set of controls.

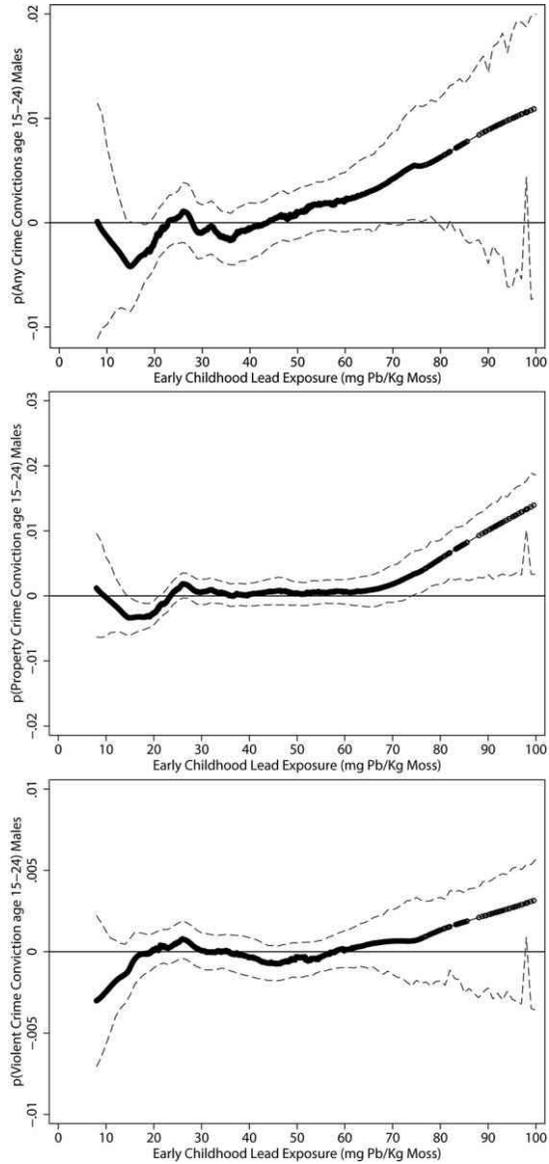


FIG. 9.—Semiparametric fixed effects estimates (see Baltagi and Li 2002) for crime (males), with 95% bootstrapped confidence intervals (1,000 repetitions). The dependent variable in the top graph is $P(\text{ever convicted})$, that in the middle graph is $P(\text{property crime})$, and that in the bottom graph is $P(\text{violent crime})$. See table 4 for the full set of controls.

is less precisely estimated. For 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 pattern similar to that for property crime, but it is not statistically significant.

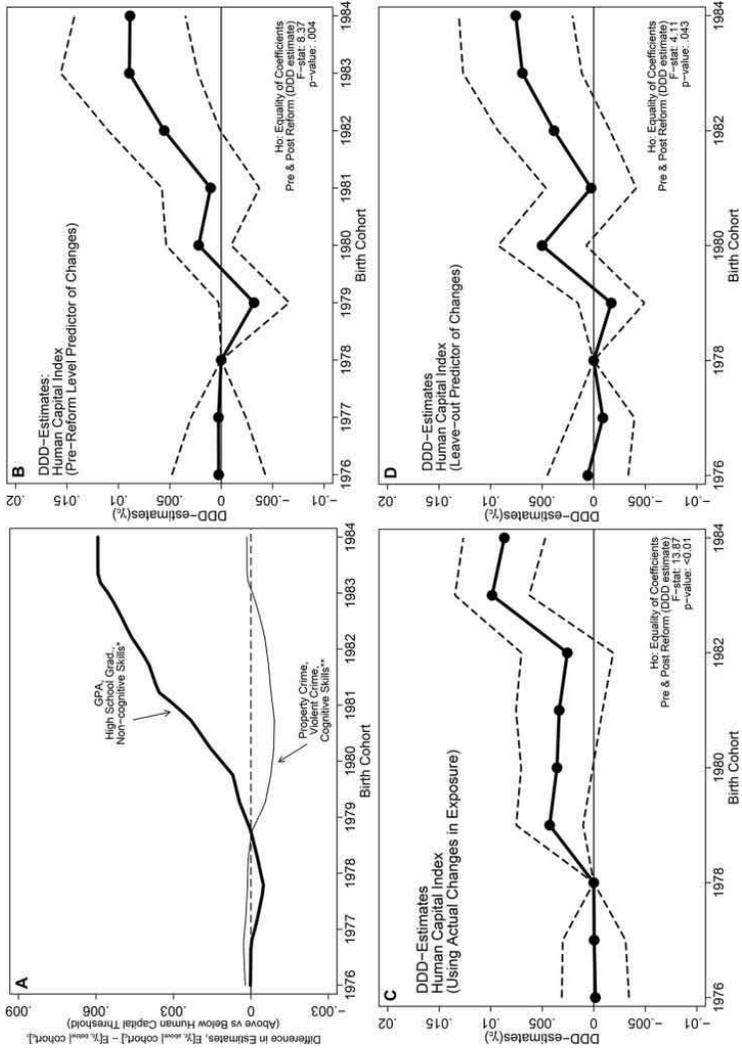
The semiparametric regressions suggest that 65 mg/kg could be a candidate for a threshold for (property) crime. For human capital outcomes, the relevant threshold seems to be located at a lower level (~30 mg/kg). To formally test for the location of these thresholds, we follow standard practice and estimate a series of piecewise linear regressions that allow for a change of slope at different assumed thresholds (see 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 semiparametric regressions. An alternative interpretation for this pattern could be a nonlinear relationship between MPb and BPb. However, piecewise linear spline regressions reveal no evidence of meaningful nonlinearities in the moss-blood relationship at the suggested thresholds (see table B2; tables B1–B4, C1, D1, E1, E2, and F1 are available online).

In summary, the semiparametric 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 $\mu\text{g}/\text{dL}$ (app. B3), the threshold for the adverse effects of lead exposure on adult outcomes is, if anything, located significantly lower than 10 $\mu\text{g}/\text{dL}$. We verify this in section V, where we use the estimates of the moss-blood link to find out which BPb levels the estimated neighborhood MPb 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

⁴³ The criterion for locating the true threshold is the assumed threshold at which the sum of squared residuals (SSR) in the piecewise linear regression is minimized. Hansen's (1999) LR statistic is $nT[(\text{SSR}_s - \text{SSR}_{\min})/\text{SSR}_{\min}]$ 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.



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 focuses 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 allow 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 reforms were fully implemented. Hence, the 1976–78 cohorts represent cohorts “unexposed” to the reforms. 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 reforms.

FIG. 10.—Summary of estimates of the 1980–81 reforms on the long-term outcomes of males. This figure shows results from four ways of summarizing the effects of the 1980–81 reforms. *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 eq. [2]) against the cohort of birth for the two domains of outcomes. The thick line shows the differences across neighborhoods above and below the human capital threshold for outcomes predicted to be affected by the reforms (*GPA, high school completion, and noncognitive skills). The thin line shows the differences across neighborhoods above and below the human capital threshold for outcomes predicted to be unaffected by the reforms (**[-]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. *B* displays point estimates (γ 's) from the DDD version of equation (2) (i.e., the differential impact of an additional unit of prereform 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 completion, and noncognitive skills; see the text for details). The index is constructed by first standardizing the outcomes (mean of 0, SD of 1) and then taking the average of the three standardized outcomes, restricting the sample to individuals for whom all three outcomes are nonmissing. The dashed lines represent 90% pointwise confidence intervals. Standard errors are clustered at the CZ and cohort-neighborhood level. *C* shows the same estimates as in *B* after replacement of the prereform MPb 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). *D* shows the same estimates as in *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.

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 reforms on adult outcomes of being exposed to an additional unit higher prereform MPb level relative to the 1978 cohort. We plot the estimates to illustrate how the effects of exposure to the reforms 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 reforms on BPb levels (fig. 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 prereform MPb level was 29 mg/kg. Hence, at least for the human capital outcomes, the estimated threshold documented in the semiparametric analysis in section IV.B is well within the boundaries of the prereform exposure levels. This provides us with four predictions of how $\hat{\gamma}_c$ should evolve across cohorts, depending on the outcome, and the prereform 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 noncognitive skills (domain 1)—for

1. children in neighborhoods with prereform levels above 30 mg/kg to improve after the reforms and
2. children in neighborhoods with prereform 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

3. cognitive skills and property and violent crime (domain 2) for children in neighborhoods above 30 mg/kg should all remain unchanged after the reforms and
4. those for 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 IV.B, 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 reforms.

For brevity, comparability, and a clearer overview of the results, we summarize the estimates from the 12 separate estimations of model (2) on (standardized measures of) the domain 1 and domain 2 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-versus below-threshold neighborhoods. The thick line shows the difference for the domain 1 outcomes, and the thin line shows the difference for the domain 2 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 reforms at older ages do not differ before the reforms between children in above- and below-threshold neighborhoods, for either the domain 1 or the domain 2 outcomes, and are not statistically distinguishable from zero. For the domain 1 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-versus postreform 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 the domain 2 outcomes, as predicted, the differences before versus after the reforms in the above- and below-threshold neighborhoods are small and not statistically distinguishable from zero, with few exceptions.⁴⁵

⁴⁴ F -test in above-threshold neighborhoods: 11.04 for GPA, 32.08 for high school completion, 7.65 for noncognitive skills; F -test in below-threshold neighborhoods: 1.44 for GPA, 1.22 for high school completion, 0.02 for noncognitive 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 fig. D5, despite the uncertainty of the location (if any) of the threshold and the failure to detect nonlinearities. From fig. D5, we can see that after the reforms, there is some indication that the probability of ever being convicted decreases after the reforms in high-exposure areas but not in low-exposure areas. This might suggest that for the noisier any-crime category, there exists a threshold at locations similar to those for human capital outcomes. However, we interpret this finding with caution, given the poor precision of the estimates in the top panel of fig. 8 and the top-left graph in fig. C4.

An alternative approach to concisely summarizing the effects of the 1980–81 reforms on long-term outcomes can be seen in figure 10*B*, 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 1) predicted to be affected by the 1980–81 reforms. We follow 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 noncognitive 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 prereform 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- versus postreform coefficients in figure 10*B*.

The estimates in figure 10*B* follow a pattern similar to those in figure 10*A* and the impact of the reforms on BPb levels (fig. 6): (1) no evidence of differential trends across the neighborhoods in the prereform cohorts, (2) cumulative increasing differences after the implementation, and (3) a leveling off some time after the reforms were fully in place. These three figures support the common-trends assumption and the differential effects of the reforms, depending on baseline lead exposure and the outcome of interest. Consistent with lasting environmental contamination, they also indicate a lag of approximately 2 years before the benefits of the 1980–81 reforms fully materialized. This lag length is identical to the one used by Strömberg, Schütz, and Skerfving (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 fig. 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 sorting. However, endogenous residential sorting before birth could potentially bias our reform analysis estimates. We showed in section III.B 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 prereform lead level in the neighborhood of birth around the time of the reforms and whether maternal mobility between the year before birth

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

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 prereform MPb level to predict changes in exposure is to simply use the actual changes in exposure within the neighborhood of birth before versus after the reforms. 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 prereform MPb 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 prereform MPb level predictor of changes in exposure provides a similar picture of the impact of the reforms.

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 versus 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 prereform MPb 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 postreform cohorts, using only the prereform MPb level in neighborhood n and the “leave-out” first-stage estimates.⁴⁹ We repeat the procedure for all CZs. The resulting neighborhood-specific treatment

⁴⁷ These results are available upon request. We also tested for systematic mobility around the reforms (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 higher-prereform exposure neighborhood of birth to a lower-prereform exposure neighborhood during childhood decreased somewhat after the reforms. Reduced mobility out of higher-prereform exposure areas is consistent with parents valuing the reform-induced reduction in lead exposure. The slight reductions in out-migration from higher-prereform 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.

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

⁴⁹ The predicted change in neighborhood n is calculated for the 1982–84 cohorts using estimates from all other CZs is given by $\text{ReformChange}_n = \sum_i^N (\hat{\pi}_1 \text{Lead}_n^{\text{Prereform}} + \hat{\pi}_2 (\text{Lead}_n^{\text{Prereform}})^2) / N$, where i denotes children in the neighborhood of birth and $\hat{\pi}_1$ and $\hat{\pi}_2$ are, respectively, the linear and quadratic estimates from the augmented first-stage equation. We use the same controls in both specifications.

intensity variable is the predicted change in neighborhood MPb levels based on the experiences of neighborhoods in other CZs with the same prereform MPb 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 reforms 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 semiparametric panel data analyses reflects changes in childhood lead exposure caused by the reforms. Importantly, if the results in our panel analysis were due to unobserved differential trends across high- and low-exposure neighborhoods, we would not expect to find such a systematic pattern around the timing of the 1980–81 reforms. Since there are no clear or significant effects of the reforms on children in the below-threshold neighborhoods, these results provide support for the much weaker relationship below certain thresholds, as documented in the semiparametric regressions. The reform analysis also supports the differential impact of early lead exposure across genders, since the effect of the reforms 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). Because of 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- and above-threshold neighborhoods, suggesting less risk for a weak-instrument bias. For comparison, using the 1976–84 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 pattern similar to that for the 2SLS estimates but are smaller in magnitude, consistent with measurement error attenuating the OLS estimates.

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- and full-reform changes in exposure only.

TABLE 6
OLS AND 2SLS ESTIMATES OF THE EFFECT OF THE 1980–81 REFORMS ON MALES

	DEPENDENT VARIABLE							
	GPA		P(high school graduation)		Noncognitive Skills		Cognitive Skills	
	OLS	2SLS	OLS	2SLS	OLS	2SLS	OLS	2SLS
A. Above Threshold								
Lead exposure (mg/kg)	-.164*** (.030)	-.256*** (.0769)	-.0022*** (.0006)	-.0044*** (.0008)	-.0056** (.0020)	-.0124*** (.0030)	.0003 (.0009)	-.0015 (.0024)
Commuting zones (CZs)	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
No. of children	172,786	172,786	172,786	172,786	172,786	172,786	172,786	172,786
First-stage <i>F</i> -statistic	NA	49.1	NA	49.1	NA	49.1	NA	49.1
B. Below Threshold								
Lead exposure (mg/kg)	-.0109 (.0571)	-.0534 (.1633)	.0009 (.0008)	-.0014 (.0033)	.00251 (.0024)	.01095 (.0085)	.0007 (.0016)	-.0031 (.0037)
CZs	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
No. of children	123,780	123,780	123,780	123,780	123,780	123,780	123,780	123,780
First-stage <i>F</i> -statistic	NA	19.4	NA	19.4	NA	19.4	NA	19.4

NOTE.—The table presents OLS and 2SLS estimates from eq. (3) for the reform sample. For comparability across outcomes, the estimation sample consists of males for whom all outcomes are nonmissing. The instrument is the interaction term (postreform-cohorts dummy \times prereform 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 (1979–81) interacted with the prereform MPb level and interpolated pollution measures (Cd, Cu, Zn) for the cohorts 1980–82, 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 CZ levels. NA = not applicable.

** Significant at the 5% level.

*** Significant at the 1% level.

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.

1. Prenatal, Early-Childhood, or Later Exposure?

The results presented so far focus on the effects of lead exposure in early childhood (ages 1–3). As discussed above, this setup is natural, given that children's BPb levels peak at the age of 2 because of increased exposure and take-up and also because it is a period when children are more susceptible to damage because of 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 with later-childhood exposure.

To provide evidence of the role of prenatal exposure, we tested whether birth weight, low birth weight, and prematurity were affected by the 1980–81 reforms. 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), birth weight and gestational age are only crude proxies of neonatal health, and consequently our results should be viewed as indicative only for the relative impact of pre- and postnatal 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 3 or older at the time

⁵¹ In app. 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. Key determinants of this age exposure profile are 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. Xue et al. (2007) find that the child hand-to-mouth activity decreases with age but find no gender differences. Infants and toddlers are also likely to be more susceptible to damage from lead because of the sensitive and rapid period of development.

of the reforms 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 3–5 at the time of the reforms (the cohorts born between 1976 and 1978; see fig. 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.

2. 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 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.

3. Comparing Siblings

Previous studies argue that sibling fixed effects 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, table E2 provides sibling fixed effects estimates. Overall, the sibling fixed effects 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

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.

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 E2 may suggest that parental investments do not vary with respect to early lead exposure.

V. Interpretation

The existence and location of MPb 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 MPb thresholds to BPb 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.

A. *Assessing the Magnitude of the Estimates*

We start by using our estimates of the moss-blood elasticity (see eq. [B1], in app. 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.

Evaluating 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 $\mu\text{g}/\text{dL}$. The corresponding number for the high school completion and noncognitive skills thresholds is approximately 5 $\mu\text{g}/\text{dL}$.

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

⁵³ Twenty-eight 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, Oregon, 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).

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 to 5 $\mu\text{g}/\text{dL}$. 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 5 have BPb levels above 5 $\mu\text{g}/\text{dL}$, and 150,000 out of these are above 10 $\mu\text{g}/\text{dL}$ (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 $\mu\text{g}/\text{dL}$ 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% (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 $\mu\text{g}/\text{dL}$ leads to around 4.4% higher lifetime earnings (among males). Naturally, this estimate captures only 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 $\mu\text{g}/\text{dL}$ are economically disadvantaged (see Currie 2009), our results suggest that the expected effects on individual earnings could be greater.

B. 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, Pinto, and Savelyev (2013). Using the enlistment data, we decompose the effects of lead exposure on later outcomes into the effects via measured cognitive and noncognitive 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 noncognitive skills and other (unmeasured) factors to the total effect (normalized to 100%).

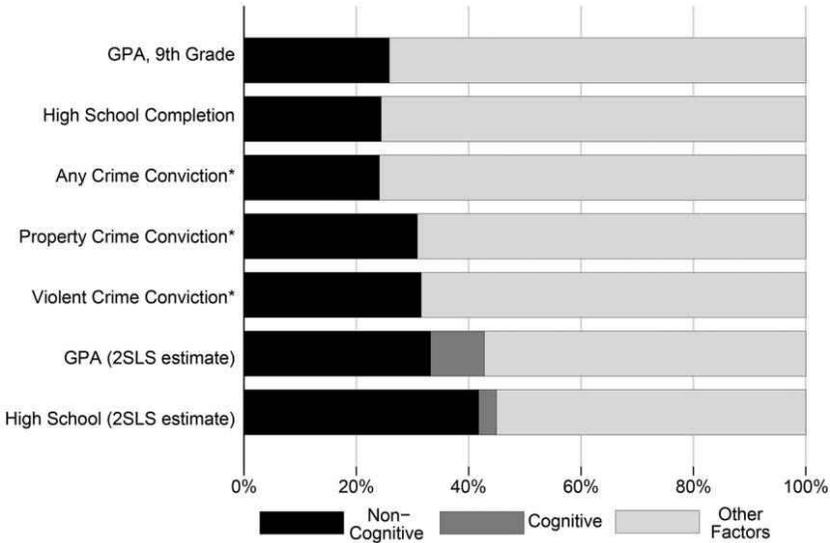


FIG. 11.—Decomposing the effects of early lead exposure on long-term outcomes by source. Each bar represents the total treatment effect normalized to 100%. The top five bars use the panel data fixed effects model for the full male sample, and the bottom two bars decompose the 2SLS estimates for males in the above-threshold neighborhoods. The method and data used to construct the figure are presented in appendix F. Following Heckman, Pinto, and Savelyev (2013), we set small and statistically insignificant contributions of the opposite sign to zero. See appendix F for information about the simplifications made to produce the figure. For the middle three bars (asterisks), note that the crime data are not linked to the enlistment data, and to construct this figure for the crime outcomes, we use aggregated neighborhood measures of cognitive and noncognitive skills to calculate the contribution of each skill. For human capital outcomes, we use linked individual data.

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 noncognitive skills. Changes in noncognitive skills explain between 20% and 30% of the effect of changes in lead exposure on human capital and 25%–30% 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 noncognitive skills from the 2SLS-model account 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. Noncognitive 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 links between lead and crime and between lead and PFC development (Cecil et al. 2008), we did not preclude finding an impact on noncognitive 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 those 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 that are typically described as “counterintuitive” (Canfield, Jusko, and Kordas 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 look 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 noncognitive personality traits (Borghans et al. 2011, 2016). (4) The effects on cognitive ability may be 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 the 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 noncognitive 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 noncognitive skills to vary with the instrument.

⁵⁴ A potential explanation is that the composition of children who 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 United States 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 phaseout, only a share of the children have been exposed, namely, children living in low-standard housing with deteriorating water pipes and/or 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.

While the returns to noncognitive skills are stable and not significantly affected by the changes in exposure, the returns to cognitive skills (for high school completion) become significantly lower. Since the decomposition uses the estimates of the factor loadings for the full-reform sample, the estimated reduction in the returns 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 assumes not only 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 phaseout 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, but consistent with the benchmark toxicological dose-response model, we find that the relationship between early lead exposure and noncognitive 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 ($5 \mu\text{g}/\text{dL}$); 150,000 of these children have levels of at least $10 \mu\text{g}/\text{dL}$. Globally, the WHO estimates that more than 40% of children have BPb levels above $5 \mu\text{g}/\text{dL}$. Our results suggest that early lead exposure may have deleterious effects on academic performance among children with BPb at levels from $5 \mu\text{g}/\text{dL}$ and above and on criminal convictions from around $7 \mu\text{g}/\text{dL}$. 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 $5 \mu\text{g}/\text{dL}$, solely via the effects on high school graduation, increases the lifetime earnings by around 4%. If the effects via crime and noncognitive skills are taken into account, the effects are presumably larger.

In terms of mechanisms, noncognitive 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 noncognitive skills are more malleable than cognitive skills. Hence, our study not only provides 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 noncognitive skills are likely to be more successful than those focusing on cognitive skills.

Stark disparities in lead exposure continue to exist in the United States, where the age of the housing stock and 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 toward noncognitive skills more effective in girls than in boys?

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