Lead Policy and Academic Performance: Insights from Massachusetts

JESSICA WOLPAW REYES
Amherst College

In this article, Jessica Wolpaw Reyes investigates the link between lead exposure and student achievement in Massachusetts. Childhood exposure to even low levels of lead can adversely affect neurodevelopment, behavior, and cognitive performance. Using a panel dataset of cohorts of children born in the 1990s who were third and fourth graders in the 2000s, Reyes finds that elevated blood lead levels in early childhood adversely impact performance on later standardized tests. Accordingly, the Massachusetts state policy to reduce lead levels effectively lowered the share of children scoring unsatisfactory on standardized tests by 1–2 percentage points. Reyes shows that public health policy targeting lead has clear potential to improve academic performance and urges policy makers to give it serious consideration in this larger context.

Education research aims to understand what happens in schools—what teachers and students do together—in order to improve and enrich educational endeavors. Education research is often therefore quite reasonably focused on what is happening *in* classrooms specifically and *in* schools more generally. But there is a growing realization that we need to shift more of our focus to what students and teachers bring to the classroom. Thomas (2012) writes that "education research becomes valuable only when it takes account of the reality of the educational endeavor" (p. 26). This paper turns its attention to one particular reality of the educational endeavor: the fact that myriad environmental influences—some good, some bad—shape the people children are when they arrive in their classrooms. In what follows I investigate the impact of lead, one critical environmental influence.

Children in modern society are exposed to numerous environmental toxins such as lead, methylmercury, polychlorinated biphenyls, and arsenic; the effects of these toxins on psychological and cognitive development are plau-

Harvard Educational Review Vol. 85 No. 1 Spring 2015

sibly substantial (Grandjean & Landrigan, 2014). This article examines one specific pathway by which an environmental exposure may affect childhood development and academic performance. Using data from Massachusetts in the 1990s and 2000s, I investigate the link between lead exposure in early childhood and student test scores in the elementary school years. Massachusetts has been a leader in the prevention of childhood lead poisoning and the reduction of childhood lead exposure. This paper quantifies the societal impact of this policy-induced reduction in childhood lead exposure.

There is strong evidence that lead exposure adversely affects children's development and, consequently, that reducing children's lead exposure has substantial potential benefits (Bellinger, 2008). However, it has proven more difficult to characterize the significance of these impacts on society: exactly what effects does lead have *at the societal level*? This paper aims to contribute to the literature by assessing whether and to what extent reductions in lead exposure have been responsible for improved academic performance of Massachusetts children. While I look to the past for answers, this question is not merely of historical interest. Children in many areas of the world, both in the United States and abroad, are still exposed to moderate and high levels of lead (Mielke, Gonzales, & Mielke, 2011; Mielke, Laidlaw, & Gonzales, 2010). Despite the fact that lead toxicity continues to threaten children, funding for public health programs combating lead has been gutted in recent years (Dell'Antonia, 2012). This article adds to the chorus of policy makers and scholars who argue that these program cuts are extremely shortsighted. In states where lead programs have been reduced, or where they were never robust, an alliance between the public health and education communities could be a catalyst for progressive policies. Thus, lead provides a compelling opportunity to leverage public health policy in furtherance of education policy goals.

Ambitious lead policy may have potentially broad implications. If lead policy improves health and childhood outcomes in substantial ways, it becomes more than public health policy and broadens to serve as effective social policy. Anyon (2005) argues that policy makers should think broadly and creatively about the larger set of public policies that can influence educational outcomes. A growing body of work in the social sciences investigates the strong and lasting effects of early life influences (Almond & Currie, 2011b; Case & Paxson, 2010; Heckman, 2008). This literature outlines countless cases in which, as Benjamin Franklin famously quipped, "an ounce of prevention is worth a pound of cure." Moreover, this is not just about levels or just about achievement on tests. By understanding the role environmental factors play in socioeconomic achievement gaps, we gain insight into the ways in which public policy addressing environmental injustice may also reduce academic disparities. In addition, given the well-established link between lead and social and behavioral outcomes, lead reduction policies may yield additional benefits for student behavior. Thus, by looking in detail at the societal-level effects of

Massachusetts' lead reduction policies, this paper aims to provide insight into the potentially broad significance of strong lead policy.

There are several reasons why Massachusetts is a good choice for this analysis. First, individual-level lead data are available for nearly all children born in Massachusetts since the early 1990s. The Massachusetts Childhood Lead Poisoning Prevention Program (CLPPP) of the Massachusetts Department of Public Health (DPH) manages an extensive monitoring program, screening 80–90 percent of children under the age of six. Second, individual-level test score data are available for all children in public schools in Massachusetts since the year 2001. The Massachusetts Department of Elementary and Secondary Education (DESE) collects and disseminates data on performance on state standardized tests of children in public schools, comprising 95 percent of the state's school-age children. Third, strong and aggressively implemented public health policy in Massachusetts has produced substantial reductions in lead exposure, providing variation that can be used to identify a relationship between lead exposure and academic performance. Fourth, with data on the approximately 700,000 children born between 1991 and 2000 and attending the approximately 1,200 public elementary schools in Massachusetts, this analysis has the potential to identify effects that might be elusive when using a smaller sample size or different research design.[1]

Previewing the results, this article provides support for the hypothesis that childhood lead exposure adversely affects academic performance. A preliminary investigation reveals a strong cross-sectional relationship between early childhood lead levels and elementary school test scores. This is then confirmed with a differences-in-differences analysis: schools with bigger lead declines displayed larger performance improvements. The primary regression analysis is a panel data analysis on *groups* of children identified by school and birth year. When looking only at group means (a rough measure of group lead and performance), this analysis yields substantial but not robust results. When investigating other moments of the within-group distributions, in particular the tails of the distributions, I find a significant and robust relationship between the share of children with high lead and the share of children with low test scores. Further analysis indicates that this result has potentially significant policy implications, so that lead policy is indeed serving as education policy.

Background

Lead

Research suggests that lead is a particularly dangerous toxin for young children, who not only absorb lead more efficiently than adults but are also at a sensitive stage of their neurobehavioral development (Bellinger, 2008; Hammond, 1988). Historically, the primary environmental sources of lead exposure for the average child have been lead-based paint, leaded gasoline, and lead water pipes. This article studies effects of exposure to lead from any of these sources, although it is likely that most exposure in Massachusetts in the 1990s arose from deteriorating lead-based paint in older housing and earlier deposition of lead in soil from leaded gasoline. With no leaded gasoline or leaded paint in general use since the mid 1980s, these were the primary dangers in the 1990s and 2000s (Mielke et al., 2011; Mielke et al., 2010).

Exposure to lead results in the absorption of lead into the human body. A blood lead level (BLL), the measure used in this paper, is a good measure of recent and current exposure. It is the concentration of lead in the body's blood supply at a given point in time and is measured in micrograms of lead per deciliter of blood (µg/dL).[2] Until recently the Centers for Disease Control and Prevention (CDC, 2010) identified 10 µg/dL as the "level of concern" for children. Data from the National Health and Nutrition Examination Survey 1999–2000 indicate that, circa 2000, only 1.7 percent of children in the United States exhibited blood lead levels above the 10 µg/dL "level of concern" (CDC, 2012b). However, with mounting evidence that lead is harmful even at lower levels (CDC, 2010; Bellinger, 2008; Gilbert & Weiss, 2006), in June 2012 the CDC decided to "use a childhood BLL reference value based on the 97.5th percentile of the population BLL in children ages 1–5 (currently 5 µg/dL)" (CDC, 2012a, p. 6).

At the same time, it is important to clarify the distinction between lead *exposure* and lead *poisoning*. Lead exposure is simply exposure to some level of lead, as indicated by a nonzero blood lead level. In contrast, lead poisoning encompasses a certain set of symptoms (such as anemia, seizures, brain damage, coma) and occurs at particularly high levels of exposure, generally those yielding BLLs in excess of 25 µg/dL. This article is primarily concerned with investigating the broad effects of common but more moderate lead exposure (exposure associated with lead levels of approximately 5–20 µg/dL).

Academic Performance

Both education researchers and economists use the term *human capital* to describe the bundle of knowledge, personal characteristics, competencies, and abilities through which an individual contributes to society in a meaningful and valuable way (Heckman, 2000). As individuals progress through life, they accumulate human capital at home, at school, and at work. Characteristics such as the ability to learn well, to listen, to cooperate, and to be respectful of others are established early in life and are foundational elements of human capital. On the other hand, characteristics such as easy distractibility, short temper, and reduced cognitive capacity may interfere with the development of human capital. Thus, investigating factors that influence development and cognitive performance early in life can help us understand how those factors, and policy intended to impact them, might affect the accumulation of human capital and with it later-life social behavior and labor market performance.

Academic performance in childhood is therefore a useful place to start when understanding human capital accumulation. A growing body of literature makes the argument that labor economics and the economics of education should cast a wider lens by looking back at formative early life experiences and environments as determinants of later life outcomes (Almond & Currie, 2011a; Almond & Currie, 2011b; Case & Paxson, 2010; Heckman, 2008; Zivin & Neidell, 2013). Almond & Currie (2011b), for example, cite abundant results showing that events in early childhood can have long-lasting impacts on adult outcomes, and argue that understanding such influences is important to understanding adult behavior and outcomes.

Lead Exposure and Academic Performance

Children are exposed to numerous environmental toxicants that may adversely affect their neurodevelopment, cognitive capacity, and behavior. Lead is one of these toxicants: it is a hazardous neurotoxicant with a wide range of adverse effects on human health and behavior. Extensive evidence indicates that childhood exposure to lead reduces intelligence quotient (IQ), impairs behavior, impairs academic performance, and increases learning disabilities (Canfield et al., 2003; Lanphear et al., 2005; Miranda et al., 2009; Reyes, 2007, 2014; Schnaas et al., 2006).

Moreover, these effects on children's cognitive performance are long-lasting and occur at very low levels of lead exposure (Canfield et al., 2003). Reviewing the literature, Bellinger (2008) reports that lead levels below 10 μg/dL are associated with reduced IQ, academic deficits, and neuropsychiatric disorders and that marginal effects may even be greater at the lowest lead levels. Bellinger concludes that "no level of lead exposure appears to be 'safe' and even the current 'low' levels of exposure in children are associated with neurodevelopmental deficits" (p. 172). The growing recent literature on lead's effects on academic performance includes, for example, Ferrie, Rolf, and Troesken (2012), McLaine et al. (2013), Zhang et al. (2013), Chandramouli et al. (2009), and Miranda et al. (2011). Thus, while lead and academic performance are each of independent interest, the link between the two is plausibly strong, practically relevant, and certainly of interest.

Lead Policy in Massachusetts

Massachusetts has been at the forefront of the prevention of childhood lead poisoning and the reduction of childhood lead exposure. Since 1971 Massachusetts law has required the removal or covering of lead paint hazards in homes where any children under six reside and has mandated a program for the prevention, screening, diagnosis, and treatment of lead poisoning.[3] Massachusetts' lead laws are among the strongest and most comprehensive of the states, and the Childhood Lead Poisoning Prevention Program is viewed as a pioneering program that has been effective in reducing lead poisoning and exposure. CLPPP oversees the mandatory lead screening of all children under the age of six, the provision of appropriate medical and environmental services to affected individuals and their families, and the development and implementation of policies aimed at eliminating sources of lead exposure. To those ends, CLPPP also engages in a wide slate of research, educational, and clinical activities. These efforts have been successful in substantially reducing lead levels and lead poisoning rates: as of 2010 only 0.078 percent of screened Massachusetts children had lead levels above 20 μg/dL. This share was down more than tenfold from the 1995 rate of 0.94 percent.

Education Policy in Massachusetts

The Massachusetts Department of Elementary and Secondary Education oversees the public education system for Massachusetts comprised of approximately 1,200 elementary schools serving roughly 400,000 students in grades K–6. These schools are distributed across the 351 municipalities (towns or cities) of Massachusetts and organized into approximately 300 local or regional school districts. (For convenience, I will generally use the term “town” to encompass both cities and towns.)

In 1993 the Massachusetts Education Reform Act (MERA) instituted fundamental changes to public education in Massachusetts. One element of MERA was a new framework allocating state aid as the difference between a foundation budget (a basic level required to fund an adequate education) and a community’s ability to contribute its own funds. Downes, Zabel, and Ansel (2009) find that between 1993 and 2000 MERA significantly reduced funding gaps, thereby stemming increases in achievement gaps that would have occurred otherwise.[4] However, they caution that MERA was not entirely successful reducing within-state inequality: large within-state disparities remain, associated with the increasing concentration of low-income and limited-English-proficiency students in certain localities and with an unchanged gap in spending between the top spending quartile and the rest.[5]

A second major component of MERA was the institution of the Massachusetts Comprehensive Assessment System (MCAS) tests. MCAS exams are administered each April for specific subjects in particular grades and are required of all students educated in the public schools in Massachusetts. The goals of the MCAS exams are to monitor student, school, and district performance and thereby to inform curricular and instructional policy.[6] Because MCAS testing was already in place, the Federal No Child Left Behind Act of 2001 (NCLB) did not initiate a new testing framework but rather expanded the existing framework of statewide testing in Massachusetts. NCLB requires states to administer assessments in basic skills and to use the results of those assessments to inform education reform; it also links federal funding to performance improvements. This framework is similar to what was already in place from MERA, though arguably more extensive and more burdensome in its link between scores and funding.

Lead and Academic Performance in Massachusetts

The established relationship between childhood lead exposure and adverse neurobehavioral effects renders it likely that the moderate to low levels of lead (below 15 µg/dL) that were common in Massachusetts children born in the 1990s could plausibly have impaired cognitive performance of those children. In addition, public policy in Massachusetts greatly reduced lead levels of children during that time period. Consequently, it is reasonable to expect lead and lead policy to have had measurable effects on academic performance in Massachusetts as those children grew up. This paper employs this window of opportunity to understand lead's impact: the analysis will study the effects of early childhood lead on academic achievement of cohorts of children born in Massachusetts during the 1990s.

Data

Data Structure

The ideal data for this analysis would be comprised of individual-level observations that include lead level in early childhood, test scores in elementary school, and other individual, family, community, and school characteristics. However, data confidentiality restrictions have made it infeasible to link data on lead and test scores for each individual child. Instead, the data for this analysis is a panel comprised of *groups* of Massachusetts schoolchildren. This creates a number of challenges that will be discussed below in the section titled *Challenges of Group-Level Analysis*.

The dataset for this study draws on a variety of sources, including administrative data from the Massachusetts Departments of Education and Public Health, as well as publicly available data from other government sources. The dataset was constructed from data for all children born between 1991 and 2000 in Massachusetts who attended third and fourth grades between 2000 and 2009 at public elementary schools in Massachusetts and who took MCAS tests between April 2001 and April 2009. Each group of children is identified by the year in which the children were born and the elementary school they attended. For example, one group might be children who were born in 1994 and attended Leverett Elementary; this group would have attended third grade at Leverett Elementary in the school year 2002–2003 and taken the third-grade MCAS test in April 2003. For each group of children, elementary school academic achievement measures are constructed from MCAS data and early childhood lead levels are constructed from CLPPP data.[7]

Lead Data

Data on childhood lead levels come from the screening and monitoring program operated by the Massachusetts Childhood Lead Poisoning Prevention Program (CLPPP) of the Massachusetts Department of Public Health (DPH).

As discussed above, Massachusetts law mandates lead screening of all children under the age of 6. The blood lead levels measured by these mandatory screenings are entered into a database used by CLPPP to monitor lead levels and lead poisoning, and it is this database that is employed.

A child may be screened for lead multiple times in his or her young life—either in separate years or possibly multiple times in the same year. All of these measures are entered into the database along with the geographic location of the child's residence and sparsely reported demographics. The dataset allows the linking over time of lead measurements for an individual child. Overall, of children attending public elementary school in Massachusetts, approximately 80–90 percent have at least one early childhood lead measure in the CLPPP database.[8] To represent a child's early childhood lead level, I use the second-highest lead measured for that child between the ages of 0 and 6. Results are generally robust to alternate methods of measuring each child's lead level.[9]

Table 1 summarizes the data for the entire sample and for subsamples broken out by cohort (early vs. late)[10] and income (bottom quartile, or "low," vs. top quartile, or "high").[11] These distinctions will prove helpful, as we can think of later and lower-income cohorts as those most likely to be significantly positively affected by lead policy. We see that the average lead level in the sample was 4.2 μg/dL and that lead levels dropped substantially over the 1990s, falling from 5.2 to 3.1 μg/dL. The table also shows that in the early cohort, average lead levels were significantly higher in low-income towns relative to high-income towns (5.4 vs. 4.1 μg/dL). By comparing the difference between high- and low-income towns across cohorts, it is also apparent that this income-related gap closed somewhat over time. Turning our attention to the share of children with elevated blood lead, we see a similar pattern but a more significant income gap and closing thereof. In the early cohorts, the average share with blood lead above 10 μg/dL was 11 percent; for the late cohorts that drops to 3 percent. This reduction is particularly intense in low-income towns where the share drops from 12 percent to 3 percent, compared with high-income towns that saw a decline from 5 percent to 1 percent.

To get a better sense of these temporal movements, figure 1a displays the rate of elevated BLL (above 10 μg/dL) for cohorts born in the years 1990 to 2008. The figure shows that the share of children with lead levels above 10 μg/dL declined significantly in the 1990s and more modestly in the subsequent decade. For children born in 1991, 7 percent had lead levels exceeding 10 μg/dL; that share drops to 1 percent for the 2000 birth cohort and 0.3 percent for the 2008 birth cohort. Figure 1b shows similarly striking declines in the share of children with lead levels above 5 μg/dL, from more than one-third of the 1991 birth cohort to approximately one-twentieth of the 2008 birth cohort. What is also evident is that lower-income communities started the 1990s with much higher rates of elevated blood lead but that this gap closed substantially over time. In other words, lead went down across the board but even more so for low-income children.

FIGURE 1a *Rate of blood lead ≥ 10 μg/dL*

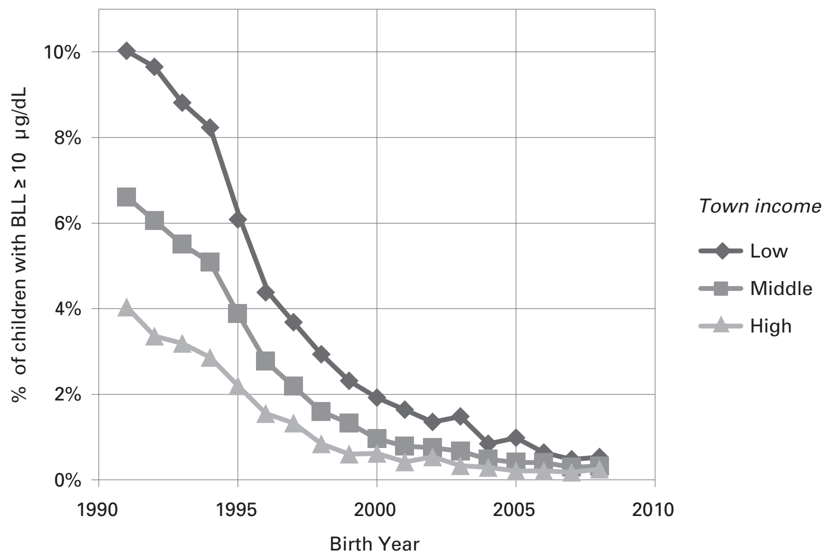
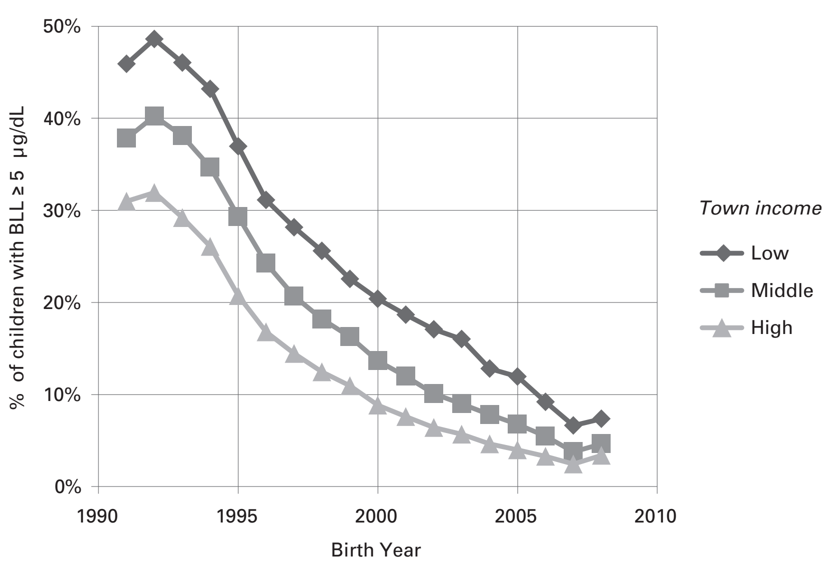


FIGURE 1b *Rate of blood lead ≥ 5 μg/dL*



Note: Sample includes Massachusetts children born between 1991 and 2008. Income categories are based on town per capita income in the year 2000. "Low income" includes towns with income in the bottom quartile (<$20,000); "middle income" includes towns in the middle two quartiles ($20,000–$30,000); and "high income" includes towns in the top quartile (>$30,000).

TABLE 1 *Summary of data*

	Full sample	Early cohorts[a] (born 1991–1992)			Late cohorts[a] (born 1999–2000)		
		All	*Low income*[b]	*High income*[b]	*All*	*Low income*[b]	*High income*[b]
Lead Measures							
Mean childhood lead (mg/dl)	4.23 (1.00)	5.20 (0.62)	5.42 (0.59)	4.10 (0.63)	3.14 (0.54)	3.24 (0.55)	2.25 (0.53)
Share with childhood lead 5-10 mg/dL	0.28 (0.09)	0.36 (0.05)	0.37 (0.04)	0.28 (0.06)	0.17 (0.04)	0.19 (0.04)	0.10 (0.04)
Share with childhood lead > 10 mg/dL	0.07 (0.04)	0.11 (0.03)	0.12 (0.04)	0.05 (0.03)	0.03 (0.01)	0.03 (0.01)	0.01 (0.01)
Share with childhood lead > 20 mg/dL	0.006 (0.00)	0.010 (0.00)	0.011 (0.00)	0.004 (0.00)	0.002 (0.00)	0.002 (0.00)	0.001 (0.00)
MCAS scores: 3rd grade							
ELA — average percentage correct	0.76 (0.09)	0.76 (0.09)	0.70 (0.08)	0.83 (0.04)	0.72 (0.08)	0.66 (0.08)	0.79 (0.04)
ELA — share of students scoring satisfactory	0.61 (0.19)	0.64 (0.20)	0.50 (0.18)	0.81 (0.10)	0.57 (0.19)	0.43 (0.17)	0.74 (0.10)
Math — average percentage correct	0.75 (0.09)				0.75 (0.09)	0.69 (0.09)	0.82 (0.05)
Math — share of students scoring satisfactory	0.58 (0.19)				0.61 (0.19)	0.48 (0.18)	0.76 (0.11)
MCAS scores: 4th grade							
ELA — percentage correct	0.70 (0.07)	0.69 (0.08)	0.63 (0.06)	0.75 (0.05)	0.70 (0.07)	0.64 (0.07)	0.76 (0.04)
ELA — share of students scoring satisfactory	0.53 (0.21)	0.52 (0.21)	0.36 (0.16)	0.71 (0.13)	0.51 (0.21)	0.35 (0.16)	0.70 (0.13)
Math — percentage correct	0.66 (0.10)	0.59 (0.10)	0.52 (0.08)	0.68 (0.07)	0.69 (0.09)	0.63 (0.08)	0.77 (0.05)
Math — share of students scoring satisfactory	0.43 (0.20)	0.37 (0.20)	0.23 (0.14)	0.56 (0.15)	0.49 (0.19)	0.37 (0.16)	0.66 (0.13)

Town characteristics							
Population density (thousands per sq. mile)	3.78 (4.11)	3.83 (4.13)	4.05 (3.36)	3.01 (3.80)	3.87 (4.28)	4.11 (3.49)	2.93 (3.72)
Poverty rate	0.09 (0.07)	0.08 (0.06)	0.13 (0.05)	0.03 (0.02)	0.10 (0.08)	0.17 (0.07)	0.04 (0.03)
Income per capita ($k year 2000)	27.00 (17.71)	28.12 (19.47)	14.96 (2.98)	51.96 (26.81)	27.96 (18.27)	14.92 (3.33)	50.32 (23.57)
Share with < high school education	0.16 (0.10)	0.16 (0.10)	0.26 (0.09)	0.06 (0.02)	0.15 (0.10)	0.26 (0.09)	0.06 (0.02)
Share with BA or beyond	0.33 (0.17)	0.33 (0.17)	0.17 (0.07)	0.56 (0.11)	0.33 (0.17)	0.17 (0.07)	0.57 (0.11)
School characteristics							
Share of students that are low-income	0.32 (0.30)	0.31 (0.29)	0.53 (0.25)	0.07 (0.12)	0.34 (0.30)	0.60 (0.25)	0.08 (0.11)
Expenditures per pupil ($k year 2000)	8.87 (2.10)	7.50 (1.33)	7.30 (0.78)	7.85 (1.75)	10.26 (2.10)	10.02 (0.98)	10.44 (2.74)
Share of students that are Black	0.09 (0.15)	0.10 (0.16)	0.11 (0.12)	0.05 (0.10)	0.09 (0.14)	0.10 (0.12)	0.05 (0.09)
Share of students that are Hispanic	0.14 (0.20)	0.12 (0.18)	0.22 (0.23)	0.02 (0.04)	0.16 (0.21)	0.30 (0.26)	0.04 (0.05)
Sample sizes							
Number of groups	18,342	4,263	1,447	997	3,927	1,271	951
Average group size	68.38 (24.20)	68.63 (24.03)	61.87 (25.46)	73.22 (20.01)	66.80 (24.03)	65.85 (25.90)	67.67 (20.32)

Notes: Means are shown, with standard deviations in parentheses. Each observation is a school/town-cohort-birth-year group. Data sources as described in text: lead data from the Massachusetts Department of Public Health, test score data from the Massachusetts Department of Elementary and Secondary Education.

[a] Early cohorts are the 1991 and 1992 birth-year cohorts; late cohorts are the 1999 and 2000 birth-year cohorts. For 3rd grade test scores, the earliest cohort in the data is the 1992 cohort and the latest is the 2000 cohort. For 4th grade test scores, the earliest cohort in the data is the 1991 cohort and the latest is the 1999 cohort.

[b] Income categories are based on town per capita income in the year 2000. "Low income" includes towns with income in the bottom quartile (<$20,000); "middle income" (not shown) includes towns in the middle two quartiles ($20,000–$30,000); and "High income" includes towns in the top quartile (>$30,000).

Student and School-Level Data

Data on academic achievement come from the Massachusetts Comprehensive Assessment System (MCAS) tests administered by the Massachusetts Department of Elementary and Secondary Education (DESE). Both Massachusetts and federal laws mandate regular standardized testing of all children in public schools in the commonwealth. MCAS scores from these tests are entered into a database used by the DESE to monitor student, school, and district performance. While limited demographics such as income and race/ethnicity are provided with publicly available statewide MCAS score summaries, they are *not* provided with the individual-level MCAS score data that are broken out by school and used here.

For this analysis, I used third- and fourth-grade MCAS scores for English language arts (ELA) and mathematics. These scores are available for the test years 2001–2009, except for the third-grade math scores, which are not available for 2001–2005. Numeric scores are reported as the number of items answered correctly, and these are rescaled to the percentage correct. Numeric scores are also translated into qualitative categories of "Advanced/Above Proficient," "Proficient," "Needs Improvement," and "Warning/Failing" (Massachusetts DESE, 2011). A score of "Proficient" or higher is deemed "satisfactory" by the DESE; a score of "Needs Improvement" or below is deemed "unsatisfactory." Both the quantitative (percent correct) and qualitative (categorical) measures will be used in this article.

In table 1 I summarize third- and fourth-grade MCAS scores for the sample period. The average percentage correct is in the range of two-thirds to three-quarters: 76 percent for third-grade ELA, 75 percent for third-grade math, 70 percent for fourth-grade ELA, and 66 percent for fourth-grade math. The average share of students scoring satisfactory ranges from 37 to 64 percent depending on the grade and subject. While some of these shares, such as 37 percent, seem relatively low, others, such as 64 percent, are high relative to other states. The table also shows large and persistent income gaps in performance: the satisfactory rate is approximately 30 percentage points lower in low-income towns, and this gap persists to the end of the sample period. For example, while 71 percent of fourth graders in the early cohorts in high-income towns display satisfactory performance in ELA, only 36 percent of fourth graders in low-income towns do so; the gap is almost identical for the late cohorts. Considering trends over the sample period, we see that third-grade ELA scores declined, fourth-grade ELA scores held steady, and fourth-grade math scores improved. These changes had little effect on income gaps, however, with only the fourth-grade math gap closing slightly, by 3 percentage points in the percent correct and 4 percentage points in the share scoring satisfactory. However, because the MCAS tests and their scoring may be modified from time to time, we must be cautious about concluding too much from time trends.

Community and School Data

In order to understand the characteristics of the communities in which students live and the schools they attend, I drew on data from multiple sources. Massachusetts contains 351 municipalities (towns or cities), and the assumption is that they represent the "community" in which a child lives and attends school. Characteristics of municipalities include population density, poverty rate, per capita income and its square, share of adults with less than a high school education, and share of adults with a bachelor's degree or beyond. Data on population, population density, and educational attainment are from the Decennial U.S. Census for the year 2000 and subsequent estimates created by the U.S. Bureau of the Census for the years 2001–2009. Data on income are from the Massachusetts Department of Revenue. All dollar values are adjusted for inflation into year-2000 dollars.

In any given year, there are approximately a thousand separate elementary schools in Massachusetts.[12] Data on school characteristics are from the Massachusetts DESE and include data obtained via school and district reporting as well as data calculated by DESE analysis of census data. Using these data, I create measures of the following school characteristics: spending per pupil, the share of students who are from low-income families, and the shares of students who are Black or Hispanic. I summarize these variables in table 1; we see that these characteristics show much less movement over time than either lead or test scores. The most notable exceptions are the relative increases in the shares of low-income and Hispanic students in schools located in low-income municipalities, as discussed in Downes et al. (2009).

Empirical Approach

Empirical Strategy

I use quasi-experimental methods to estimate the causal impact of early childhood blood lead on later academic performance. The central empirical strategy I employ is regression analysis that links blood-lead levels in early childhood (ages 0–6) to educational outcomes in the elementary school years. As described above, this regression analysis is performed on cohorts of Massachusetts schoolchildren, where each observation is a group of children identified by the elementary school the children attend and by the year in which the children were born. Panel data analysis is conducted for cohorts of children born between 1991 and 2000 and attending third and fourth grades between 2000 and 2009 at the more than one thousand public elementary schools in Massachusetts.[13] The idea is to assess the impact of Massachusetts' lead policy and the effect of lead on academic performance by investigating the relationship between lead and achievement for the cohorts most affected by the policy.

The primary regression equation (Equation 1) is

$$MCAS\ Score_{g(s,b)y} = \alpha_0 + \alpha_1 Lead\ Level_{g(s,b)} + C_{c(s)y}\beta_1 + S_{sy}\beta_2 + \gamma_y + \varepsilon_{g(c,b)y}$$

where the subscripts represent group *g*, school *s*, community *c*, birth year *b*, and year of MCAS test *y*. Note that the group *g* is defined by the school *s* and the birth year *b* and hence is denoted *g(s,b)*. Similarly, the community *c* is the community in which the school is located and is denoted *c(s)*. The variable *MCAS Score* represents a measure of the group's MCAS performance, either the group's average percentage correct or the share of the group achieving a certain qualitative performance standard (such as "satisfactory"). The variable *Lead Level* represents a measure of the group's childhood lead levels, either the average of the individual lead levels or the share of the group with lead levels in a certain range (such as above 10 µg/dL). The coefficient α_1 indicates the effect of lead on achievement and, hence, is the main coefficient of interest. The vector *C* is a set of community characteristics: population density, poverty rate, per capita income and its square, share of adults in the town with less than a high school education, and share of adults in the town with a bachelor's degree or beyond. The vector *S* is a set of school- or district-level characteristics: school spending per pupil in the district, share of students at the school who are low-income, and the shares of students who are Black or Hispanic. Fixed effects for year are included as γ_y to account for statewide time trends. When both grade-levels are pooled into a single regression, a dummy variable for fourth grade is also included in order to allow for different scoring and/or performance in the two grades. The regression is run as ordinary least squares, with weights representing group size (to reflect the number of individual children included in a group's observation) and Huber-White robust standard errors clustered on school.

Because of the inclusion of controls and fixed effects, identification of the effect of lead on test scores derives primarily from the variation in lead and test scores within each school over time. That is, cross-school differences in test scores that are caused by cross-school differences in characteristics such as poverty, income, or race have been accounted for by including the community characteristics as controls. Similarly, over-time differences in test scores that are caused by any number of larger year-to-year changes in Massachusetts in the 2000s have been accounted for by including fixed effects for year. What is left to be used to estimate the relationship between lead and test scores is the covariation in lead and test scores *within* each school *over* time.

Challenges of Group-Level Analysis

Although, by necessity, this article uses groups of children as the unit of analysis, it is instructive to consider the differences between an individual and group-level analysis, as well as the challenges of a group-level analysis in particular. A regression run with individual children as the unit of analysis would estimate the linear effect of an individual's childhood lead level on that individual's academic outcomes. Moreover, given the evidence indicating nonlinear effects of lead, it would be advisable and feasible to test other functional forms such as log, spline, or quadratic.

In the present case, we do not have individual-linked lead and performance. A number of papers address the question of inferring individual-level relationships when only group-level data are available. Firebaugh (1978) shows that the group-level coefficient will be unbiased relative to the individual-level coefficient only when the group average independent variable does not directly affect the group average dependent variable. However, with the possibility of peer effects by which group lead may affect individual test scores, the latter is likely not true in the present case. More recently, a number of authors have presented a variety of creative approaches to these issues, highlighting the complexity of the econometrics, the necessity for care in estimation, and the possibilities for bias when moving between group and individual analyses (e.g., Berhane, Gauderman, Stram, & Thomas, 2004).

For this particular situation, comparing the estimation procedure and coefficients for the individual-level analysis to those of the group-level analysis can shed some light on the challenges of this group-level analysis. First, there is less variation with which to identify the coefficient of interest. Group means by definition are less variable than individual values, and high or low values will be "smoothed out" by the averaging. With less useful variation there will be a lower signal-to-noise ratio and possibly attenuation bias.[14] Second, to the extent that the effects of lead on achievement vary across levels of lead exposure, averaging will make it difficult to identify those changes in marginal effects. Group means will rarely show a mean lead of 15 µg/dL or a mean MCAS score of 30 percent correct, but this may be where the relationship is strongest. With "observations" pulled toward the middle of the distributions, the tails are unreachable by analysis. To the extent that the more important effects are among those with high lead and low scores, averaging will bias estimates downward.

To address these concerns, I conducted analyses using other moments of the lead and test score distributions. Moments are quantitative measures of the shape of a distribution of data points: medians, percentiles, or shares of the group above or below particular thresholds for lead and test scores. We can describe a distribution of test scores, for example, merely by stating the mean of that distribution. However, such a description is incomplete; while the mean gives us a sense of the level of test scores, it doesn't give us any information about the spread of test scores. Other moments can tell us more, such as how spread out the distribution is or how many students do extremely well or extremely poorly. Using other moments of the distribution makes better use of the within-group variation and distributions of both lead and test scores, positioning the analysis more effectively between the infeasible but detailed individual level analysis and the feasible but overly simplified group mean analysis.

Thus, if a higher share of children in a group has lead above the "level of concern" of 10 μg/dL, perhaps a higher share of children in that group will have test scores that are below the "satisfactory" level defined by the DESE. This still leaves a lower signal-to-noise ratio and possible attenuation bias if most of the action is happening in only a small portion of the sample population near the tails, but it gets closer to matching the empirical reality.

These strategies may be at least partially effective in addressing the shortcomings of the group-level analysis, but we should not assume that they solve all of the problems. It remains the case that this analysis will depend on movements in distributions over time; after controlling for community and school characteristics, there will be much less movement in distributions of lead and scores than there would be in individual values. Thus, I endeavor to make the best possible use of these movements, but I am cognizant of the fact that estimated coefficients may be substantially attenuated. While this is the most important empirical issue at hand, several others, such as the assignment of individuals to groups, are discussed in detail in Reyes (2011).

Descriptive Results

Cross-Sectional Variation

The goal of this paper is to determine how childhood lead exposure affects test scores. In this section, I first use a simple descriptive analysis to get a sense of how lead and test scores covary in Massachusetts. To do this, I consider covariation between a school's elementary school MCAS test scores and the early childhood lead levels of children in that community. Of course, any covariation could be driven by many other factors, such as income, race/ethnicity, or socioeconomic status, so this truly is just a first glimpse into the data. Figures 2a and 2b display the group average fourth-grade MCAS scores (percent correct) for ELA and math against the group average childhood lead level (μg/dL) for all of the groups in the sample at the beginning (born in 1992, in fourth grade in 2001–2002), and end (born in 1999, in fourth grade in 2008–2009) of the sample period.

From this first look, it is clear that higher early childhood lead levels are associated with lower test scores in fourth grade for both subjects. Each cloud of points clearly slopes downward, so that a group with higher average lead has lower average test scores. We also see steeper slopes in some of the graphs, suggesting that this effect is stronger for math than for ELA and stronger in the early cohort than in the later cohort. Lastly, we see that the later distributions (for the 1999 birth-year cohort) are shifted to the left and up relative to the earlier distributions (for the 1992 birth-year cohort). This indicates that as lead levels declined over time, test scores also rose. The entire state moved toward markedly lower early childhood lead (in the 1990s) and to somewhat higher MCAS scores (in the 2000s). The bottom line of this analysis? Lower

FIGURE 2a *MCAS ELA score vs. childhood lead*

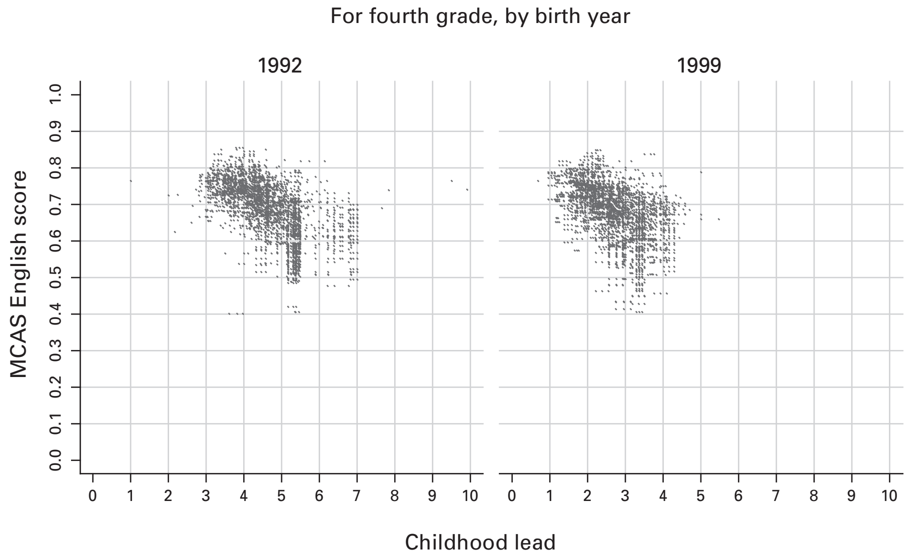
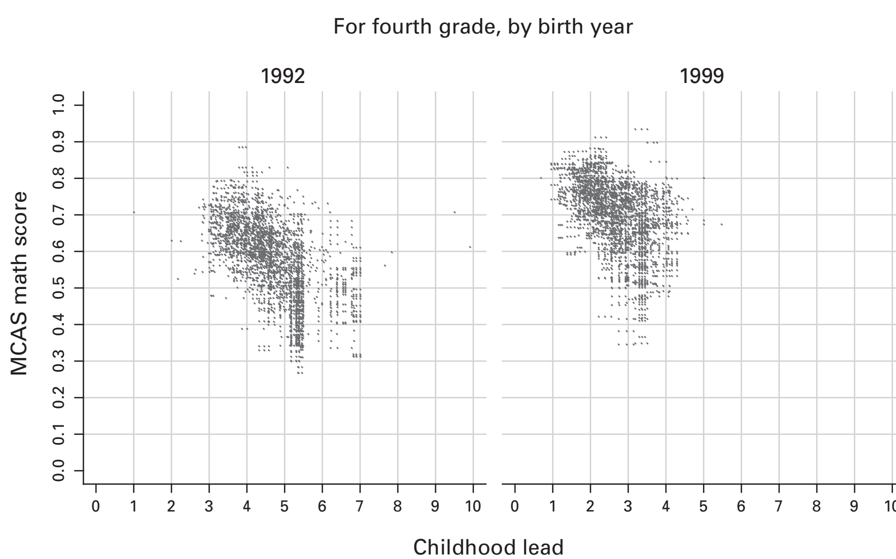


FIGURE 2b *MCAS math score vs. childhood lead*



Note: Author's calculations as described in text. MCAS score is the group average percent correct. Childhood lead is the group average blood lead in mcg/dl.

lead levels are associated with higher test scores. The exact nature of this relationship, and whether this is in fact causal, remains to be determined. The next section will get us a little closer to establishing causality.

Difference-in-Differences

One way to understand the variation in lead and test scores across communities and over time is to use a difference-in-differences analysis. Such an analysis compares how MCAS outcomes changed differentially over time in schools that experienced larger changes in lead levels as compared with schools that experienced smaller changes in lead levels. If a high level of lead in the blood does reduce academic performance, then we would expect the schools that experienced larger lead changes in early childhood in the 1990s to show relatively larger increases (or smaller declines) in academic performance in elementary schools in the subsequent decade.

In this difference-in-differences analysis, the "differences" are differences *over time*: how did academic performance change in the 2000s? These differences in academic performance are calculated separately for two sets of communities: those that experienced big declines in lead in the 1990s (the "treatment" group) and those that only experienced small declines in lead in the 1990s (the "control" group). The "difference" is the difference between these two differences: how does the change over time in one group compare to the change over time in the other group? The goal of this method is to get the difference-in-differences: did the treatment group experience greater improvements in test scores than the control group? If so—and if it is unlikely that other factors that might affect test scores were changing differentially in these two groups over time—then we get one step closer to being able to say that the "treatment" of substantially reduced lead *caused* the improvement in test scores.

To do this comparison, I investigate the effects on average (mean) MCAS score and on the share of children scoring unsatisfactory on the MCAS. I include only schools for which MCAS data are available at both the start (2002) and end (2009) of the sample, which leaves 898 schools for this analysis. I calculate the "lead change" for each school as the change from the early cohort (born in 1992) to the late cohort (born in 1999) in the share of children with lead above 10 µg/dL. The mean "lead change" in this time period is a decline of 0.044, or 4.4 percentage points fewer children with lead above 10 µg/dL. Schools are classified as "high lead change" (treatment) or "low lead change" (control) based on the size of their "lead decline" (i.e., the decline in the share of students with lead above 10 µg/dL). This classification is done in two ways. The first classification, shown in *DD1*, classifies schools as "high lead change" if their decline was larger than the mean decline and "low lead change" if their decline was smaller than the mean decline. The second classification, shown in *DD2*, classifies schools as "high lead change" if their decline was in the top

TABLE 2 *Differences-in-Differences*

Outcome measure			*DD1* [a,b]		*DD2* [a,c]	
Mean score on MCAS						
	3rd grade	ELA	0.016**	(0.007)	0.029**	(0.010)
	4th grade	ELA	0.009	(0.006)	0.010	(0.009)
	4th grade	Math	0.031**	(0.008)	0.045**	(0.124)
Share unsatisfactory on MCAS						
	3rd grade	ELA	–0.022	(0.016)	–0.052**	(0.022)
	4th grade	ELA	–0.025	(0.017)	–0.038	(0.027)
	4th grade	Math	–0.047**	(0.016)	–0.065**	(0.027)

Notes: Difference-in-difference estimates and standard errors calculated from means and standard errors of each group (before/after, control/treatment). Significance is indicated by * for a p-value < 0.10 and ** for a p-value < 0.05.

[a] The sample is broken into "Treatment" and "Control" based on 1992–1999 changes in the share of the group with lead above 10 mg/dl.

[b] For DD1: "Treatment" groups are those with above-average changes; "control" groups are those with below-average changes.

[c] For DD2: "Treatment" groups are those with top-quartile changes; "control" groups are those with bottom-quartile changes.

quartile of declines (down more than 0.086) and "low lead change" if their decline was in the bottom quartile of declines (down less than 0.027).[15]

In table 2 I show the difference-in-differences for various combinations of grade, subject, and outcome measure. It is clear that schools experiencing relatively *larger* declines in the percentage of children with lead above 10 μg/dL exhibited relatively *larger* increases in mean percent correct score on the MCAS. For example, the first number in the table, 0.016, indicates that over this time period, the mean third-grade ELA MCAS score went up by 1.6 percentage points *more* in schools with *larger* declines in lead. Looking at the second panel of the table, we see that these schools that experienced *larger* declines in lead also exhibited relatively *larger* decreases in the share scoring unsatisfactory on the MCAS. For example, the –0.052 in the *DD2* column indicates that the share of children scoring unsatisfactory declined by 5.2 percentage points *more* in schools with *larger* declines in lead.

The estimated differences-in-differences are of the expected sign in all cases and are significant in most cases. Furthermore, the differences-in-differences are larger and more significant when comparing the top quartile to the bottom quartile (*DD2*) than when comparing the top half to the bottom half (*DD1*), as one would expect. For example, the share of children scoring unsatisfactory on the fourth-grade math MCAS declined by 4.7 percentage points more in schools with above-average lead declines (relative to schools with below-average lead declines) and 6.5 percentage points more in schools with top-quartile lead declines (relative to schools with bottom-quartile lead declines.)

Overall, this difference-in-differences analysis supports the hypothesis that early childhood blood lead levels adversely affect later academic performance. Schools whose student population experienced larger decreases in lead exposure in the 1990s appear to have experienced larger increases in MCAS scores in the 2000s. Moreover, if one considers the great difficulty encountered in using policy to achieve performance improvements, effects such as a 4 to 6 percentage point reduction in unsatisfactory performance are notable. At first blush, lead policy appears to be remarkably effective.

Relationship Between Lead and Academic Performance

Regression of Average Test Scores on Average Lead

I now turn to panel regression analysis on various moments of the within-group distributions. The earlier discussion of group-level analysis makes the point that while there may not be substantial relationships between group means (average lead and average test scores), we understand that much of the action may take place at the tails of the distribution (high lead and low test scores). However, it still seems reasonable to begin the regression analysis by investigating the relationship between a group's average lead and that group's average test scores. I estimate these models and display the results in tables 3 and 4. Both tables show highly significant coefficients on lead in all specifications except the fully controlled specification. In table 3, we see that, in the baseline regression model, the mean blood lead level for the group has a statistically significant effect of –0.0258 on the mean ELA test score for the group. This means that an increase of 1.0 µg/dL in average blood lead for a group is associated with a decline of 2.6 percentage points in the average percentage answered correctly by the group on the ELA MCAS. This effect is unchanged by the inclusion of weights for group size and is doubled when year fixed effects are included. The coefficient is diminished by approximately a factor of 5 with the inclusion of either town or school characteristics, and drops drastically to an insignificant –0.0012 with the inclusion of both sets of characteristics, indicating that mean lead has no effect on mean test scores when these other characteristics are taken into account.

The coefficients for the control variables are generally of the expected sign, with population density, poverty, and share low-income students decreasing test scores, and town income per capita, educational attainment, and school expenditures increasing test scores. Some of these signs are reversed in the full specification, but further investigation indicates that this is due to multicollinearity between some of these variables.

TABLE 3 *Regression of group average MCAS ELA scores on group average childhood lead*

	(1) *Base*	*(2)* *Weighted*	*(3)* *Include year fixed effects*	*(4)* *Include town characteristics*	*(5)* *Include school characteristics*	*(6)* *Full*
Childhood lead measures						
Mean childhood lead (mg/dL)	–0.0258*** (0.0011)	–0.0275*** (0.0011)	–0.0551*** (0.0021)	–0.0070*** (0.0018)	–0.0059*** (0.0015)	–0.0012 (0.0015)
Town characteristics						
Town population density				–0.0038*** (0.0005)		0.0007 (0.0005)
Town poverty rate				–0.4646*** (0.0474)		0.1472*** (0.0443)
Town income per capita ($k)				0.0004* (0.0002)		0.0007*** (0.0002)
Town income per capita squared				–0.0000* (0.0000)		–0.0000*** (0.0000)
Share of town with < high school education				0.0005 (0.0320)		–0.0054 (0.0234)
Share of town with BA or beyond				0.0980*** (0.0181)		0.0599*** (0.0132)
School characteristics						
School expenditures per pupil ($k)					0.0026*** (0.0007)	–0.0029*** (0.0007)
Share of students who are low-income					–0.1621*** (0.0084)	–0.1370*** (0.0105)
Share of students who are Black					–0.0528*** (0.0114)	–0.0842*** (0.0121)
Share of students who are Hispanic					–0.0614*** (0.0117)	–0.0908*** (0.0127)
Constant	0.8432*** (0.0038)	0.8480*** (0.0038)	0.9870*** (0.0088)	0.7842*** (0.0101)	0.8132*** (0.0076)	0.7800*** (0.0084)
Year fixed effects	No	No	Yes	Yes	Yes	Yes

Notes: Regression on 18,154 groups of 3rd and 4th grade students in Massachusetts, where groups are defined by school and birth cohort. Test scores from MCAS tests taken from 2001 to 2009. Lead distributions based on second-highest lead measured in childhood by the Massachusetts Department of Public Health. Dummies for MCAS test year are included as indicated. Observations are weighted by weights derived from number of children in the data group (truncated below at 10 and above at 100). Standard errors are Huber-White robust and clustered by school. Significance is indicated by *** for p<0.01, ** for p<0.05, and * for p<0.10.

Table 4 shows the corresponding results for a group's average lead and that group's average math test scores. The math results are qualitatively similar to the ELA results, although the uncontrolled specification yields a coefficient on lead more than twice as large. In this case, an increase of 1.0 µg/dL in average blood lead is associated with a decline of 6.1 percentage points in average percentage correct on the math MCAS. After including year fixed effects and all of the control variables, the coefficient is reduced to a statistically insignificant –0.0013, almost identical to that for the ELA scores.

Relationships Between Other Moments of the Distributions

The literature would suggest that lead's effects are nonlinear and also likely to be modified by other factors. That is, the effect on test scores of an additional 1 µg/dL of blood lead might be higher if that single microgram is added to a lead level that started out very low or higher for a child who experiences other risk factors or adverse environmental conditions. As discussed above, because data confidentiality restrictions require that the present analysis be conducted at the group level, this analysis cannot precisely account for those factors at the individual level. However, beyond just using group means, the within-group detail makes it possible to ascertain whether there are significant relationships between other moments of the two distributions.

To do this, I investigate the relationship between the share of a group with elevated blood lead levels and the share of that group scoring unsatisfactory on the MCAS. In table 5 I show results from three different sets of regressions estimated for ELA and math, using different measures of lead. Set A uses share of children within each group with lead above 10 µg/dL, set B uses share of children with lead above 20 µg/dL, and set C uses both the share with lead between 10 and 20 µg/dL and share with lead above 20 µg/dL simultaneously. Only the coefficients on the lead variables are shown in the table.

In nearly all specifications, the share of a group with elevated blood lead has a statistically significant positive effect on the share of that group scoring "unsatisfactory" on the MCAS in both subjects. The specifications that do not control for town or school characteristics yield large effects. A one percentage point increase in the share of children within a group with lead above 10 µg/dL is associated with a 2–4 percentage point increase in the share scoring unsatisfactory on the MCAS, while a 1 percentage point increase in the share with lead above 20 µg/dL is associated with 10–18 percentage point increase in the share scoring unsatisfactory. These effects decline substantially with the inclusion of control variables but remain significant and do not disappear. In the fully controlled specification, a 1 percentage point increase in the share with lead above 10 µg/dL is associated with an increase of 0.2 percentage points in the share of that group scoring unsatisfactory. For higher lead levels, the effects are larger. A 1 percentage point increase in the share with lead above 20 µg/dL is associated with a 1 percentage point increase in

TABLE 4 *Regression of group average MCAS math scores on group average childhood lead*

	(1) Base	(2) Weighted	(3) Include year fixed effects	(4) Include town charac-teristics	(5) Include school charac-teristics	(6) Full
Childhood lead measures						
Mean childhood lead (mg/dL)	–0.0605*** (0.0016)	–0.0627*** (0.0015)	–0.0650*** (0.0025)	–0.0098*** (0.0026)	–0.0087*** (0.0023)	–0.0013 (0.0022)
Town characteristics						
Town population density				–0.0039*** (0.0007)		0.0017** (0.0007)
Town poverty rate				–0.4380*** (0.0557)		0.2120*** (0.0547)
Town income per capita ($k)				0.0010*** (0.0003)		0.0012*** (0.0002)
Town income per capita squared				–0.0000*** (0.0000)		–0.0000*** (0.0000)
Share of town with < high school education				–0.0143 (0.0393)		–0.0348 (0.0319)
Share of town with BA or beyond				0.1119*** (0.0235)		0.0766*** (0.0193)
School characteristics						
School expenditures per pupil ($k)					0.0038*** (0.0009)	–0.0044*** (0.0010)
Share of students who are low-income					–0.1864*** (0.0125)	–0.1418*** (0.0150)
Share of students who are Black					–0.0619*** (0.0157)	–0.1162*** (0.0170)
Share of students who are Hispanic					–0.0532*** (0.0170)	–0.1021*** (0.0172)
Constant	0.9168*** (0.0044)	0.9209*** (0.0043)	0.9221*** (0.0106)	0.6824*** (0.0129)	0.7271*** (0.0107)	0.6797*** (0.0115)
Year fixed effects	No	No	Yes	Yes	Yes	Yes

Notes: Regression on 12,962 groups of 3rd and 4th grade students in Massachusetts, where groups are defined by school and birth cohort. Test scores from MCAS tests taken from 2001 to 2009. Lead distributions based on second-highest lead measured in childhood by the Massachusetts Department of Public Health. Dummies for MCAS test year are included as indicated. Observations are weighted by weights derived from number of children in the data group (truncated below at 10 and above at 100). Standard errors are Huber-White robust and clustered by school. Significance is indicated by *** for p<0.01, ** for p<0.05, and * for p<0.10.

TABLE 5 *Regression of share "unsatisfactory" MCAS on share lead in certain ranges*

Childhood lead measure(s)	*Subject*	*(1) Base*	*(2) Weighted*	*(3) Include year fixed effects*	*(4) Include town characteristics*	*(5) Include school characteristics*	*(6) Full*
A. Share lead > 10 μg/dL	ELA	2.174*** (0.087)	2.345*** (0.085)	3.536*** (0.132)	0.788*** (0.096)	0.413*** (0.078)	0.210*** (0.077)
Share lead > 10 μg/dL	Math	2.890*** (0.097)	3.067*** (0.092)	3.317*** (0.132)	0.773*** (0.108)	0.441*** (0.096)	0.203** (0.092)
B. Share lead > 20 μg/dL	ELA	10.569*** (1.329)	15.369*** (0.876)	13.542*** (1.847)	3.619*** (0.413)	1.700*** (0.343)	1.024*** (0.301)
Share lead > 20 μg/dL	Math	12.940*** (1.610)	18.682*** (0.975)	12.170*** (1.721)	3.383*** (0.480)	1.616*** (0.441)	0.946** (0.391)
C. Share lead 10–20 μg/dL	ELA	2.207*** (0.112)	2.237*** (0.108)	3.765*** (0.156)	0.734*** (0.117)	0.429*** (0.094)	0.198** (0.094)
Share lead > 20 μg/dL	ELA	3.010*** (0.721)	4.746*** (0.645)	4.035*** (0.883)	2.063*** (0.365)	0.782** (0.320)	0.645** (0.297)
Share lead 10–20 μg/dL	Math	3.048*** (0.125)	3.116*** (0.118)	3.553*** (0.160)	0.732*** (0.126)	0.478*** (0.111)	0.193** (0.107)
Share lead > 20 μg/dL	Math	3.123*** (0.735)	4.521*** (0.589)	3.497*** (0.811)	1.753*** (0.399)	0.561 (0.395)	0.558 (0.360)
Year fixed effects		No	No	Yes	Yes	Yes	Yes
Town characteristics		No	No	No	Yes	No	Yes
School characteristics		No	No	No	No	Yes	Yes

Notes: Regression on groups of 3rd and 4th grade students in Massachusetts, where groups are defined by school and birth cohort. Only coefficients on lead measures are shown. Test scores from MCAS tests taken from 2001 to 2009. Lead distributions based on second-highest lead measured in childhood by the Massachusetts Department of Public Health. Dummies for MCAS test year are included as indicated. Observations are weighted as described in the text. Standard errors are Huber-White robust and clustered by school. Significance is indicated by *** for p<0.01, ** for p<0.05, and * for p<0.10.

the share of that group scoring unsatisfactory. Put another way: in a group of one hundred children, the movement of one child's lead level past the 20 µg/dL mark causes one child's performance level to fall below the satisfactory mark. Of course, in the group analysis we cannot say whether this is the same child. However, assuming no spillover effects (whereby one child's reduced performance might drag his classmates down as well), this one-to-one result seems reasonable: one child experiences a higher lead level, and one child does poorly on the MCAS test. This also suggests that the town and school characteristics may effectively address the upward bias caused by cross-sectional variation in lead and scores, yielding a believable coefficient in the final fully controlled specification.

The final rows of table 5 show results from a variant of a spline. While a traditional spline at the individual level would allow the effect of lead to vary depending on the level of lead, to adapt this to a group analysis I instead include both the share of the group with lead between 10 and 20 µg/dL *and* the share of the group with lead above 20 µg/dL in the same regression as measures of the group's lead. This analysis confirms that any lead above 10 µg/dL reduces test scores and that lead above 20 µg/dL is more detrimental to scores. In the fully controlled specification, the results are very similar for both ELA and math: a 1 percentage point increase in the share with lead between 10 µg/dL and 20 µg/dL is associated with an increase of 0.2 percentage points in the share of that group scoring unsatisfactory, while a 1 percentage point increase in the share with lead above 20 µg/dL is associated with an increase of 0.6 percentage points in the share of that group scoring unsatisfactory. Note that these results do not imply that the marginal effect of lead is increasing with lead (in fact, it appears to be declining) but, rather, that the *total* effect of lead is increasing with lead. If the lead level for 10 children in a group of 100 goes above 10 µg/dL, approximately 2 children's scores will slip below satisfactory; if the lead level for those 10 children goes above 20 µg/dL, 4 more children's scores will fall below that mark.

Implications

The results of this study indicate that what might seem like relatively small social policy changes can substantially affect academic outcomes. In the 1990s, public health policy achieved large reductions in the childhood lead levels of Massachusetts children. Having established a relationship between lead and academic performance, it is now possible to assess the full impact this lead reduction may have had on academic performance. It is important to acknowledge that the foregoing analyses are not able to fully establish causality, and consequently these assessments necessarily push this study's results beyond the empirical domain in which they were established.

If lead indeed adversely affects test scores—and we know that lead declined in Massachusetts in the 1990s—can we estimate how much test scores in Massachusetts in the 2000s might consequently have increased as the children progressively became "less leaded"? Applying the main results for how the share of children with high lead influences the share of children with poor academic performance (table 5), the 8.2 percentage point decline in the share of Massachusetts children with blood lead above 10 μg/dL would produce a decline of 1.7 percentage points in the share of children scoring unsatisfactory on the MCAS.[16] This improvement, when compared to the base rate of 30–35 percent of children scoring unsatisfactory, amounts to an approximate 5 percent reduction in unsatisfactory performance statewide. Put another way, *if lead had stayed at 1990 levels, unsatisfactory performance statewide would have been 5 percent higher.* Because the CLPPP reduced children's lead when they were very young, those children performed substantially better when they were in elementary school.

A second way to assess the practical significance of lead policy is to investigate the extent to which reductions in lead gaps (between high- and low-income communities) coincided with reductions in achievement gaps. I calculate that declines in the 1990s in the lead gap (measured as share of children with lead above 10 μg/dL) reduced the achievement gap (measured as share of children scoring unsatisfactory) by 1.0 percentage point in the 2000s. This suggests that the benefits of lead reduction disproportionately benefited low-income children, slightly reducing inequality in academic outcomes.

A third way to understand these results is to compare the effects of lead policy with the effects of tax or spending policy that increases family income. Consider low-income communities—what income increase would they need to be given to provide them as much performance improvement as they received from the lead decrease? Over the sample period, the share of children in low-income communities with lead above 10 μg/dL dropped by 9.2 percentage points, from 11.9 percent to 2.7 percent. Applying the regression results, this decline in lead would cause a drop of 1.9 percentage points in the share scoring unsatisfactory. To achieve this same improvement via a shift of the income profile of low-income communities toward the income profile of middle-income communities, that shift would have to close 22 percent of the gap between the low- and middle-income communities. Specifically, low-income communities would need to experience an increase of $2,200 of per capita income, a decrease of 1.3 percentage points in poverty rate, and a decrease of 5.4 percentage points in the share of students who are low income. Accordingly, to get, *without the decline in lead*, the same test score performance that was seen at the end of the period, per capita income would have had to go up by 15 percent for low-income communities.

These initial calculations suggest that public health policy has improved academic outcomes and reduced inequality. How significant are these effects in a practical sense? All of these assessments of benefits can be considered relative to government spending on lead policy, which in Massachusetts is currently less than $5 million annually, or less than $100 per child.[17] Since this is orders-of-magnitude smaller than education spending in Massachusetts, it would appear that spending on lead policy provides a comparatively high return. Indeed, given that other benefits such as reduced special education dollars are not yet accounted for, it is likely that the benefit-cost analysis is quite favorable (Gould, 2009; Muennig, 2009).[18] While it is difficult to do direct dollar-for-dollar comparisons, these results should make a case for careful consideration of the use of public health interventions alongside more traditional education policies.

Thus, lead policy appears to be not only an effective policy intervention but also a relatively cost-effective one when considered in the context of other policies that aim to influence educational outcomes either directly or indirectly. The particular estimates contained in this article and the strength of these results may indeed be novel, but the general sentiment is not new. We have known for decades that early interventions that address children's health and developmental needs at key moments can be pivotal (e.g., Shonkoff & Phillips, 2000). Examples of such policy interventions include Head Start, Women Infants and Children (WIC), and Medicaid. More recently we have seen multifaceted endeavors that are built on the premise that early intervention strategies can greatly reduce risk outcomes among youth. Because lead is so harmful to young children, and because reducing children's exposure to it is relatively easy, lead policy may provide a unique opportunity to positively influence the health and developmental trajectories of children. Certainly, it is easier than going back later to correct the deficits lead causes.

Writing about the Harlem Children's Zone, Jennifer Steele (2009) quotes Barack Obama's 2008 inaugural statement that the bundle of public policies pursued should be chosen based on data, or via what Steele describes as "tough, data-driven decisions" (p. 527). While education policy makers aspire to make well-informed choices driven by reliable data, these decisions are often challenged by the unpredictability and uncertainty of the available information. Thus, the education policy community is routinely engaged in debate about what policies to endorse and how to most effectively implement those policies. I would argue that the public health community often has greater agreement about what to do but faces a different challenge: convincing the rest of society to do it. For the public health community, this challenge of translating what we *should* do to what we *will* do is particularly acute when the harms to be prevented are not obvious, as is the case with lead exposure. (They may have trouble convincing people to care about obesity, lead, or endocrine disruptors but not a lot of trouble getting support to address fast-spreading fatal diseases.) A key to achieving the optimal bundle of public policies and to improving societal welfare might be found in better communication across disciplines. If the education policy community hears what the public health community has to say about what to do, it may find one or two public health policies to add to its education policy arsenal. Lead policy is one example, but there are likely others that represent similarly low-hanging fruit. Enhanced dialogue and coordination between state departments of education and state departments of health could potentially reveal a variety of opportunities to improve social welfare in a cost-effective manner.

Conclusion

Using comprehensive data on lead levels in the 1990s and test scores in the 2000s, this study has investigated whether childhood lead levels are linked to academic performance. One motivation for this study was to assess any measurable societal-level effects of Massachusetts' strong public health policy. Another motivation was to seek additional evidence on the more general question of whether and how lead may affect children's development and life outcomes, and what that means for society.

This paper has shown a strong cross-sectional relationship between early childhood lead levels and elementary-school test scores. Groups of children with higher childhood lead levels perform substantially worse on standardized tests in the third and fourth grades. These results are greatly diminished with the introduction of controls for community and school characteristics. Analyses on other moments of the distributions—in particular, the share of children with elevated blood lead and the share of children scoring unsatisfactory on the MCAS test—yield more substantial, statistically significant, and robust results. Over the time period under consideration, reductions in lead have yielded a drop of 1–2 percentage points in the share of children scoring unsatisfactory on the MCAS test. Continuing research will employ individual-level data to better identify these effects and will extend the analysis with a more comprehensive benefit-cost analysis of lead policy in the context of broader social policy.

In conclusion, this article confirms our hypothesis not only that lead adversely affects academic performance but also that the aggregate societal impact of strong public health policy to reduce lead is a worthwhile effort in improving academic prosperity in children. Consequently, policy makers concerned with improving academic outcomes may want to broaden their view, looking beyond traditional education policies such as pay for performance to also consider other environmental and public health policies that can significantly influence children's cognitive and social development. These policies and their impacts are certainly of historical interest, but it would be a mistake to think they exist only in hindsight. Rather, they continue to provide compelling potential opportunities to leverage public health policy in ways that could provide substantial societal benefits in the future.

Notes

1. It is important to acknowledge one significant limitation of this analysis: confidentiality restrictions prevent linking of the lead data to the test score data at the individual level. Instead, it is possible to link small groups of children (defined by school and birth year) and perform analysis relating various moments of the within-group lead and test score distributions. This strategy of using moments of the distributions, rather than just means, takes some (but not all) of the sting out of this limitation.
2. Dentine and bone lead levels reflect the concentration of lead in teeth and bone, respectively, and are regarded as good indicators of cumulative past exposure.
3. See the MA CLPPP Act of 1970, MGL 111, ss 189A-199B, and http://www.lawlib.state.ma.us/subject/about/lead.html for more information on Massachusetts' lead law.
4. This result for Massachusetts aligns with the more general conclusion of Card and Payne (2002) that equalization of education spending in various states reduced gaps in test scores. See Berger and McLynch (2006), Hoxby (2001), and Murray, Evans, and Schwab (1998) for more discussion of school finance reform.
5. For this analysis, it is important to note that nearly all of the impact of MERA on relative spending occurred in the 1990s and so was complete by the 2000s.
6. The relevant performance level definitions in the MCAS are: "*Advanced* (Grades 4–10): Students at this level demonstrate a comprehensive and in-depth understanding of rigorous subject matter, and provide sophisticated solutions to complex problems. *Above Proficient* (Grade 3): Students at this level demonstrate mastery of challenging subject matter and construct solutions to challenging problems. *Proficient* (Grades 3–10): Students at this level demonstrate a solid understanding of challenging subject matter and solve a wide variety of problems. *Needs Improvement* (Grades 3–10): Students at this level demonstrate a partial understanding of subject matter and solve some simple problems. *Warning* (Grades 3–8)/*Failing* (Grade 10): Students at this level demonstrate a minimal understanding of subject matter and do not solve simple problems." More information about the details of MCAS scoring and administration can be found at http://www.doe.mass.edu/mcas/overview.html.
7. The set of cohorts used in the analysis is described in appendix table 1, available at http://isites.harvard.edu/k109357.
8. This does not mean that 90 percent of children who later attend public school are screened for lead in every year but rather that 90 percent of those children are screened at least once between birth and age 6. CLPPP data indicate that approximately 50 percent of children ages 0–6 are screened in any given year. Compared to the full population of children in Massachusetts, 80–90 percent are screened at some point between ages 0–6. For more detail, consult "Screening and Incidence Statistics by Community" produced by the CLPPP for fiscal years 1998 to 2010.
9. There are several possible ways to calculate a representative lead measure for a child with more than one lead measurement. The CLPPP data provided to the author included one measure for each child in each year, the most reliable (based on screening method) and highest measure. Note that the number of measures for a child is endogenous: once a high lead measurement is found for a particular child, CLPPP offers the family treatment and services to lower the lead level and endeavors to retest the child until the lead level drops to a safe level. Hence, averaging all lead measures for a particular child would bias the calculated lead level downward, particularly for children with high lead. As a way of avoiding both downward bias from averaging and upward bias from erroneously high measures, the analysis in this paper has been conducted using the second-highest reliable measure available (as long as that is not also the lowest measure available; for children with two or fewer measures, the highest measure is used).

10. Early cohorts are the 1991 and 1992 birth-year cohorts; late cohorts are the 1999 and 2000 birth-year cohorts. For third-grade test scores, the earliest cohort in the data is the 1992 cohort and the latest is the 2000 cohort. For fourth-grade test scores, the earliest cohort in the data is the 1991 cohort and the latest is the 1999 cohort.
11. Income categories are based on the town's (or city's) per capita income in the year 2000. "Low income" includes towns with income in the bottom quartile (<~$20,000); "middle Income" includes towns in the middle two quartiles (~$20,000–30,000); and "high income" includes towns in the top quartile (>~$30,000). These cutoffs are very close to the actual quartile cutoffs and hence are employed as convenient markers for cutting the sample into subsamples.
12. The number of elementary schools in the sample declined slightly from 1,114 in the year 2001 to 971 in the year 2009 as some schools were closed or consolidated.
13. Panel data are multidimensional data that include multiple measurements of particular cohorts over time. In this situation, each cohort of children is observed for ten years. We observe multiple cohorts of children in each town/school. Panel data analysis allows the researcher to learn as much as possible while taking account of the structure of the data—that we see the same cohorts multiple years in a row and that we know which cohorts live in the same town as each other.
14. One way to understand this is to consider a mean-preserving spread of the lead and MCAS distributions. Such a transformation would provide no new useful information in a regression of group means on group means. However, if indeed *Lead* and *MCAS* exhibit a significant covariance, such a transformation could provide substantial new useful information in a regression of individual values on individual values. This follows as a direct consequence of the fact that the regression coefficient is the ratio of the sample covariance of *Lead* and *MCAS* to the sample variance of *Lead*. The covariances and variances of group means are necessarily smaller, reducing the valuable signal for identifying a regression coefficient.
15. The average changes in share of children with blood lead above 10 μg/dL in the treatment and control groups were –0.089 for treatment group 1, –0.025 for control group 1; and –0.126 for treatment group 2, –0.013 for control group 2.
16. Coefficients are from the fully controlled regressions of share scoring unsatisfactory on share with blood lead above 10 μg/dL, as shown in table 5 (and in appendix tables 2 and 3, available at http://isites.harvard.edu/k109357). Because the coefficients on lead are nearly identical between the ELA and math regressions, values quoted in the text are based on the average of the two for convenience.
17. In fiscal year 2011, spending of all government agencies in Massachusetts on lead reduction efforts totaled approximately $4 million (P. Hunter, director of CLPPP, personal communication, May 2011).
18. See Muennig (2009) and Gould (2009) for comprehensive benefit-cost analyses of societal reductions in childhood lead levels to as low as a population average below 1 μg/dL.

References

Almond, D., & Currie, J. (2011a). Human capital development before age five. In O. Ashenfelter & D. Card (Eds.), *Handbook of labor economics* (pp. 1315–1486). North Holland: Elsevier.

Almond, D., & Currie, J. (2011b). Killing me softly: The fetal origins hypothesis. *Journal of Economic Perspectives, 25*(3), 153–172.

Anyon, J. (2005). What "counts" as educational policy? Notes toward a new paradigm. *Harvard Educational Review, 75*(1), 65–88.

Bellinger, D. C. (2008). Very low lead exposures and children's neurodevelopment. *Current Opinions in Pediatrics, 20*(2), 172–177.

Berger, N., & McLynch, J. (2006). *Public school funding in Massachusetts: Where we are, what has changed, and options ahead.* Boston: Massachusetts Budget and Policy Center.

Berhane, K. W., Gauderman, J., Stram, D. O., & Thomas, D. C. (2004). Statistical issues in studies of the long-term effects of air pollution: The southern California children's health study. *Statistical Science, 19*(3), 414–434.

Canfield, R. L., Henderson, C. R., Cory-Slechta, D., Cox, C., Jusko, T., & Lanphear, B. (2003). Intellectual impairment in children with blood lead concentrations below 10 μg per deciliter. *New England Journal of Medicine, 348*(16), 1517–1526.

Card, D., & Payne, A. A. (2002). School finance reform, the distribution of school spending, and the distribution of student test scores. *Journal of Public Economics, 83*(1), 49–82.

Case, A., & Paxson, C. (2010). Causes and consequences of early-life health. *Demography, 47*(1), S65–S85.

Centers for Disease Control and Prevention [CDC], National Center for Environmental Health. (2010). *Advisory Committee on Childhood Lead Poisoning Prevention recommends a work group to investigate lowering limits on elevated blood lead levels.* Retrieved from http://www.cdc.gov/nceh/lead/lower_blood_levels.htm

Centers for Disease Control and Prevention [CDC], National Center for Environmental Health. (2012a). *CDC response to Advisory Committee on Childhood Lead Poisoning Prevention recommendations in "low level lead exposure harms children: a renewed call of primary prevention."* Retrieved from http://www.cdc.gov/nceh/lead/ACCLPP/CDC_Response_Lead_Exposure_Recs.pdf

Centers for Disease Control and Prevention [CDC], National Center for Health Statistics [NCHS]. (2012b). National Health and Nutrition Examination Survey Data, 1999–2000. Hyattsville, MD: U.S. Department of Health and Human Services.

Chandramouli, K., Steer, C. D., Ellis, M., & Emond, A. M. (2009). Effects of early childhood lead exposure on academic performance and behaviour of school age children. *Archives of Disease in Childhood, 94*(11), 844–848.

Childhood Lead Poisoning Prevention Program [CLPPP], Massachusetts Department of Public Health. (1995–2010). Screening and incidence statistics by community.

Childhood Lead Poisoning Prevention Program [CLPPP], Massachusetts Department of Public Health. (2011). *Childhood lead poisoning prevention program overview.* Retrieved from http://www.mass.gov/dph/clppp

Childhood Lead Poisoning Prevention Program [CDLPP], Massachusetts Department of Public Health. (2004). *Massachusetts strategic plan to end lead poisoning by 2010.* Retrieved from http://www.cdc.gov/nceh/lead/StrategicElimPlans/MAStrategic%20Plan8-04.pdf

Dell'Antonia, K. J. (2012, March 7). Drastic cuts to lead poisoning and prevention funds. *New York Times.* Retrieved from http://parenting.blogs.nytimes.com/2012/03/07/drastic-cuts-to-lead-poisoning-and-prevention-funds/

Downes, T., Zabel, J., & Ansel, D. (2009). *Incomplete grade: Massachusetts education reform at 15.* Retrieved from http://www.massinc.org/~/media/Files/Mass%20Inc/Research/Incomplete%20Grade/incomplete_grade_full.ashx

Ferrie, J. P., Rolf, K., & Troesken, W. (2012). Cognitive disparities, lead plumbing, and water chemistry: Prior exposure to water-borne lead and intelligence test scores among World War Two U.S. Army enlistees. *Economics and Human Biology, 10*(1), 98–111.

Firebaugh, G. (1978). A rule for inferring individual-level relationships from aggregate data. *American Sociological Review, 43*(4), 557–572.

Gilbert, S. G., & Weiss, B. (2006). A rationale for lowering the blood lead action level from 10 to 2 μg/dL. *Neurotoxicology, 27*(5), 693–701.

Gould, E. (2009). Childhood lead poisoning: conservative estimates of the social and economic benefits of lead hazard control. *Environmental Health Perspectives, 117*(7), 1162–1167.

Grandjean, P., & Landrigan, B. (2014). Neurobehavioural effects of developmental toxicity. *Lancet Neurology, 13*(3), 330–338.
Hammond, P. B. (1988). Metabolism of lead. In J. J. Chisholm & D. M. O'Hara (Eds.), *Lead absorption in children* (pp. 11–20). Baltimore: Urban and Schwartzenberg.
Heckman, J. J. (2000). Policies to foster human capital. *Research in economics, 54*(1), 3–56.
Heckman, J. J. (2008). Schools, skills, and synapses. *Economic Inquiry, 46*(3), 289–324.
Hoxby, C. M. (2001). All school finance equalizations are not created equal. *Quarterly Journal of Economics, 116*(4), 1189–1231.
Lanphear, B. P., et al. (2005). Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environmental Health Perspectives, 113*(7), 894–899.
Massachusetts Department of Elementary and Secondary Education [DESE]. (2011). *Massachusetts Comprehensive Assessment System overview*. Retrieved from http://www.doe.mass.edu/mcas/overview.html
Massachusetts Law about Lead Poisoning and Control. (2013, June). Massachusetts Trial Court law libraries. Retrieved from http://www.lawlib.state.ma.us/subject/about/lead.html
McLaine, P., Navas-Acien, A., Lee, R., Simon, P., Diener-West, M., & Agnew, J. (2013). Elevated blood lead levels and reading readiness at the start of kindergarten. *Pediatrics, 31*(6), 1081–1089.
Mielke, H. W., Gonzales, C. R., & Mielke, P. W. (2011). The continuing impact of lead dust on children's blood lead: Comparison of public and private properties in New Orleans. *Environmental Research, 111*(8), 1164–1172.
Mielke, H. W., Laidlaw, M. A., & Gonzales, C. R. (2010). Lead (Pb) legacy from vehicle traffic in eight California urbanized areas. *Science of the Total Environment, 408*(19), 3965–3975.
Miranda, M. L., Kim, D., Osgood, C., & Hastings, D. (2011). *The impact of early childhood lead exposure on educational test performance among Connecticut schoolchildren, phase 1 report.* Children's Environmental Health Initiative Research Report. Retrieved from http://www.nicholas.duke.edu/cehi/about/news/leadct.htm.
Miranda, M. L., Kim, D., Reiter, J., Galeano, M. O., & Maxson, P. (2009). Environmental contributors to the achievement gap. *Neurotoxicology, 30*(6), 1019–1024.
Muennig, P. (2009). The social costs of childhood lead exposure in the post–lead regulation era. *Archives of Pediatric and Adolescent Medicine, 163*(9), 844–849.
Murray, S. E., Evans, W. N., & Schwab, R. M. (1998). Education finance reform and the distribution of education resources. *American Economic Review, 88*(4), 789–812.
Reyes, J. W. (2007). Environmental policy as social policy? The impact of childhood lead exposure on crime. *B.E. Journal of Economic Analysis and Policy, 7*(1): article 51.
Reyes, J. W. (2011). *Childhood lead and academic performance in Massachusetts* (Working Paper 11-3). New England Public Policy Center. Retrieved from https://bostonfed.org/economic/neppc/wp/2011/neppcwp113.pdf
Reyes, J. W. (2014). Lead exposure and behavior: effects on antisocial and risky behavior among children and adolescents. *Economic Inquiry.*
Schnaas, L., et al. (2006). Reduced intellectual development in children with prenatal lead exposure. *Environmental Health Perspectives, 114*(5), 791–797.
Shonkoff, J. P., & Phillips, D. A. (Eds.). (2000). *From neurons to neighborhoods: The science of early childhood development.* Washington, DC: National Academies Press.
Steele, J. (2009). Editor's review of *Whatever it takes: Goeffrey Canada's quest to change Harlem and America. Harvard Educational Review, 79*(3), 520–529.
Thomas, G. (2012). Changing our landscape of inquiry for a new science of education. *Harvard Educational Review, 82*(1), 26–51.

Zhang, N., Baker, H. W., Tufts, M., Raymond, R. E., Salihu, H., & Elliott, M. R. (2013). Early childhood lead exposure and academic achievement: Evidence from Detroit Public Schools, 2008–2010. *American Journal of Public Health, 103*(3), e72–e77.

Zivin, J. G., & Neidell, M. (2013). Environment, health, and human capital. *Journal of Economic Literature, 51*(3), 689–730.

Acknowledgments

The author would like to thank Sarah Carleton, Carrie Conaway, Chris Foote, Paul Hunter, Jun Ishii, Robert Knorr, Yolanda Kodrzycki, Alicia Sasser Modestino, Sarah Neslund, René Reyes, Lisa Sanbonmatsu, Jeffrey Zabel, Bo Zhao, and seminar participants at the New England Public Policy Center, the Federal Reserve Bank of Boston, the Bureau of Environmental Health of the Massachusetts Department of Public Health, and the Healthy Homes Lead Poisoning Prevention Branch of The Centers for the Disease Control and Prevention for valuable advice and comments. Research staff at the Massachusetts Department of Public Health and the Massachusetts Department of Elementary and Secondary Education provided invaluable support with data acquisition and processing. Christopher Morrison provided outstanding research assistance. Any remaining errors are my own. This research was generously supported by the New England Public Policy Center of the Federal Reserve Bank of Boston. The views expressed in this paper are those of the author and not necessarily those of the Federal Reserve System or the Federal Reserve Bank of Boston.

An earlier version of this work was distributed by the Federal Reserve Bank of Boston as "Childhood Lead and Academic Performance in Massachusetts," New England Public Policy Center Working Paper No. 11-3, by Jessica W. Reyes, http://www.bostonfed.org/economic/neppc/wp/2011/neppcwp113.htm The Federal Reserve Bank of Boston retains a license to use, reproduce, and make derivative works of the Working Paper.