Lead Exposure and Racial Disparities in Test Scores

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February 7, 2015

We consider a new source of racial disparities in test scores: African American students' disproportionate exposure to environmental toxins, and, in particular, lead. Using a unique individual-level dataset of children's preschool lead levels linked with future educational outcomes for children in RI, we document significant declines in racial disparities in child lead levels since 1997, due largely to state policies aimed at reducing lead hazards in homes. Exploiting the change in child lead levels as a result of the policy, we generate causal estimates of the impact of preschool lead levels on reading and math test scores through grade 8 in an IV framework. We find that a 5 ug/dl increase in child lead levels reduces test scores by 30-60 percent of a standard deviation, depending on the specification. The effects are strongest in the lower tail of the test score distribution and do not fade over time. We calculate that the decline in racial disparities in lead explains between 37 and 76% of the decline in racial disparities in test scores witnessed over the past decade in RI.

I. Introduction

Racial disparities in educational outcomes have declined over time but remain large, with black children scoring 70% of a standard deviation below their white counterparts as recently as 2012 (US DOE, 2013). In their review of the existing research on the factors responsible for the black-white test score gap, Jencks and Phillips (2011) conclude that family income, school segregation, family structure, parenting practices, and the quality of educational inputs explain only part of the gap, leaving a large share unexplained.

We consider an alternative explanation for the black-white test score gap: Black children's greater exposure to environmental toxins. Because African American students are disproportionately burdened by exposure to harmful environmental toxins, we argue that recent efforts to reduce exposure are likely to have the greatest impact on them. If exposure to environmental pollutants negatively affects cognitive development, it follows that environmental regulations that lead to disproportionate reductions in exposure among African American children are also likely to reduce racial disparities in educational outcomes.

Not only are African American children more likely to be exposed, but they may also suffer more harm from a given level of exposure. For example, good nutrition and cognitive stimulation may be protective against the negative effects of lead poisoning, and African American children may be less likely to enjoy these protective factors (Environmental Protection Agency, 2015).

To examine these questions, we focus on the environmental toxin lead, for which there is strong epidemiological evidence of a negative relationship between early exposure and future cognitive and non-cognitive outcomes. Using a unique dataset for the state of RI that contains individual child blood lead levels measured from birth through age six linked with educational

outcomes for grades three through eight, we first document the greater lead burden born by African American children in our sample. Among RI children born in 1997, (the first year of our data), African-American children had lead levels that were 58% higher than white children, and children on free lunch had lead levels 55% higher than those not on free lunch. These disparities are consistent with a large literature on environmental inequalities documenting that sources of potential environmental risk are concentrated among racial and ethnic minorities and the poor (see Ringquist, 2005; Mohai and Saha, 2006; Currie, 2011). However, debate over the cause of the inequality remains. While some argue that the pattern results from the deliberate placing of hazardous sites in minority communities (due to lack of organized opposition and/or low land prices), others argue that a more likely explanation is that housing prices reflect the quality of the environment (Chay and Greenstone, 2005; Banzhaf and Walsh, 2008; add Currie et al., 2015).[1] Since African American families are more likely to be poor, it is not surprising then that they tend to live in homes of lower environmental quality.

While there is an extensive literature documenting the negative relationship between elevated childhood lead levels and poor cognitive outcomes, identifying the causal impact of elevated lead levels on child outcomes is challenging for two reasons: First, the presence of multiple confounders leads to omitted variable bias, and second, measurement error in child lead levels leads to attenuation bias. Confounding arises from the fact that children with higher lead levels are more disadvantaged: they are more likely to be black, poor, live in single parent homes, and have mothers with lower human capital – all of which are independently associated with lower test scores. Measurement error arises from the imprecision in each blood test, and

[1] For example, Woverton (2009) finds that race and income predict current locations of manufacturing plants but to not predict the original citing of plants.

from the fact that blood tests capture only recent exposure, whereas lead that persists in the body in organs such as the brain still causes cognitive impairment.

To identify the effect of elevated lead levels in early childhood on future cognitive achievement, we employ an instrumental variable strategy to address both omitted variable bias and attenuation bias. Our instrument is based on a policy that took effect in 1997 requiring landlords to obtain "lead-safe" certificates in order to rent their properties. The state contracted with local community based organization to conduct outreach targeting the four "core cities" of RI which are characterized by greater poverty and a higher burden of lead. As a result, neighborhoods with a greater share of pre-war housing (where there is a much greater concentration of lead in the paint), and with higher average child lead levels (as measured in 1997) witnessed the biggest gains in the number of lead-safe certificates issued, and the biggest reductions in child lead levels. By including neighborhood (tract) fixed effects in our analysis, we focus on changes in the number of lead safe certificates over time within a tract to identify our effects. We assume that other underlying characteristics of the neighborhood did not change coincident with the growth in certificates, and provide empirical evidence to support our identifying assumption.

Using this instrumental variable strategy, we find that an average lead level of 5 (the level at which the CDC currently recommends intervention) reduces average reading test scores by 8 points, or 65 percent of a standard deviation. This effect does not appear to fade over time, remaining strong by eighth grade, the last year for which we have data. Moreover, the effects are greatest at the lower tail of the test score distribution and for math test scores, the disproportionate effects in the lower tail are even greater. The IV estimated effects are larger than the OLS estimates, consistent with considerable measurement error in lead scores.

Finally, we show that the policy change did disproportionately reduce lead levels in black children relative to white, with the black-white ratio in measured lead levels falling from 1.58 for the 1998 birth cohort to 1.38 for the 2004 birth cohort. Based on our causal estimates, we calculate that the falling racial gap in child lead levels over this period corresponds to a 1.1 to 2.2 point reduction in the black-white test score gap (depending on specification), thereby explaining between 37 and 76 percent of the total decline in the black-white test score gap witnessed over this period in RI. These results do not simply reflect changes in lead exposure and score among the disadvantaged more generally. When we compare outcomes for children on free lunch or not, we find much smaller (though still significant) declines in disparities in both lead levels and test scores. The greater effects for African Americans likely resulted from the targeting of outreach efforts by the state in the urban areas with a high concentration of inferior housing, areas that are disproportionately African American.

Our results suggest that African American children's disproportionate exposure to environmental pollutants that affect child development can potentially explain a sizeable share of the black-white gap in educational outcomes. Policies aimed at reducing the environmental burden borne by low income and minority households have the potential to be an effective tool in reducing disparities in educational outcomes and, by extension, future income.

II. Background

A. Disparities in Educational Outcomes and Environmental Quality

Racial disparities in test scores have declined significantly between 1971 and 2012. As measured by National Association of Educational Progress (NAEP) scores, the black-white gap represented roughly1.25 of a standard deviation in 1971, falling to 0.71 by 2012, with the

greatest gains made during the 1970s and 1980s (US DOE, 2013). Jencks and Phillips (2011) provide a comprehensive review of the existing research on various factors that have been considered to explain the gap and its decline over time. These include family income, family structure, school segregation, cultural influences, test bias, stereotype threat, parenting strategies, the quality of school inputs. They conclude that together these factors explain some, but not all of the racial gap in test scores and that alternative explanations should be considered. More recently, Chay, Guryan and Mazumder (2009) have done just that. They examined the impact of relative improvements in infant health among African American children due to hospital desegregation in the American south on black cognitive achievement. They find that among cohorts born between 1963 and 1971, increasing access to health care among African Americans during infancy and improvements in health explain a significant share of the closing of the black-white gap in the American South during the 1980s.

This recent work underscores the importance of looking at factors beyond the educational system and even family characteristics to explain the racial gap in test scores – a strategy we pursue here. We focus on the role of racial disparities in environmental quality in explaining racial gaps in test scores. A long literature documents racial disparities in environmental quality, exploring multiple sources of the disparities. These include differences in proximity to known hazards, exposure to air pollution, siting of landfills/other hazards, designation as Superfund sites, and other regulatory actions (Brown, 1995; Brulle and Pellow, 2006; Ringquist, 2005; Mohai and Saha, 2006).

Inequity in the regulatory response to pollution by race and/or class spawned new federal regulation in 1994 aimed at preventing discrimination in the "development, implementation, and enforcement of environmental laws, regulations and policies" (EPA), also referred to as

environmental justice. Since then, work examining the probability of a hazardous site receiving priority for clean-up efforts (ie, Superfund status) has found that sites in black and low income areas are still less likely to receive a Superfund designation (O'Neal, 2007). But, conditional on Superfund designation, there no longer appears to be any disparity in the duration of clean-up (Burda and Harding, 2014). As part of our analysis, we examine both the burden of lead by race and income as well as the targeting of efforts to reduce lead exposure across groups. Currie (2011) demonstrated that even within zip codes, black mothers were more likely to reside near Superfund sites or plants with toxic emissions.

B. Lead Exposure in American Children

In 1970, the US Surgeon General issued his first formal statement on lead poisoning, naming it a national health problem (US Dept of Health, Education and Welfare, 1970). Since then, American children's exposure to lead has declined dramatically, due largely to two major regulatory changes involving the elimination of lead in gasoline and in household paint.[2] These regulatory changes have been credited with reducing the lead concentrations in the air from 2.5 ug/dl in 1980 to less than 0.5 by 2012.[3] Coincident with this decline, the share of pre-school age American children with lead levels in excess of 10 ug/dl has likewise declined from 8.6% to 1.4% over the period 1988 -2004, with declines witnessed across all groups, including historically high-risk groups of children (Jones et al, 2009).

Yet lead remains a significant hazard in many areas due to residual lead found in the air, dust and soil, as well as lead-based paint in older homes. The CDC reports that children in 4.5

[2] The key regulations and legislation that reduced exposure to lead included the 1970 Lead Paint poisoning Prevention Act, the Clean Air Act and EPA rules regarding leaded gasoline.

[3] (http://www.epa.gov/air/airtrends/lead.html).

million households in the US are still exposed to high levels of lead and a half a million preschool aged children have elevated blood lead levels (BLLs)(cite). The U.S. Public Health Service recently included the elimination of elevated blood lead levels as a goal in Healthy People 2020, the ten-year national objectives for improving the health of all Americans. Moreover, recent research has found that even small amounts of lead in a child's system can have deleterious effects, prompting the CDC to eliminate the use of the term "level of concern" when referring to blood levels and to lower the threshold for which children should receive case-management services from 10 to 5 ug/dl in 2012 (CDC, 2012).

C. The Biology of Lead Poisoning

Lead has no biological value and is toxic to the human body, regardless of the pathway of exposure (ingestion or inhalation). Lead is toxic because of its ability to inhibit or mimic the actions of calcium, thereby affecting all calcium-dependent or related biological processes. Many systems including the renal, endocrine and cardiovascular systems have been found to be affected by lead exposure but the nervous system appears to be the most sensitive target. Within the brain, lead levels lead to damage in the prefrontal cerebral cortex, hippocampus and cerebellum (Finkelstein, Markowitz, and Rosen, 1998). Many neurodevelopmental studies have concluded that infants, children, and the developing fetus are at the greatest risk of toxicity from low-level exposure (National Research Council, 1993). This sensitivity is due to the fact that ingested lead is more likely to be absorbed from the gastrointestinal tract of children, and, conditional on absorption, is more likely to affect the developing nervous system than the mature brain (Lidsky and Schneider, 2003).

D. Existing Evidence Regarding Elevated Lead and Child Outcomes

There is a long epidemiological literature documenting the significant negative relationship between exposure to lead and child outcomes. The negative health effects of lead ingestion have been known for thousands of years. But until the mid-twentieth century, health officials considered only severe cases of lead poisoning to be harmful. This changed in the 1960s, when medical professionals recognized that less acute lead exposure also had harmful effects and began characterizing lead poisoning as epidemic (Berney, 1993).

Recent research has focused on the impact of relatively low levels of lead during childhood on cognitive and behavioral outcomes. Chandramouli et al. (2009), Canfield et al. (2003), Lamphear et al. (2005), Nigg et al. (2010) and Wasserman (1997) have all documented a significant relationship between relatively low levels of lead (<10 ug/ml) during childhood and cognitive and behavioral outcomes including ADHD and hyperactivity. McLaine et al. (2013) uses data on preschool BLLs to estimate the relationship between elevated BLLs and reading readiness at kindergarten, a marker for later school performance, among Providence, RI school children. They find an inverse relationship between school readiness test scores and BLLs: among children with BLLs below 5, 68% scored above the benchmark levels, among children with levels between 5 and 9, 62% scored above the benchmark, and among children with a BLL of at least 10, only 49% exceeded the benchmark.

In addition to the above studies based on individual level data linking BLLs in early childhood with later outcomes, other research has relied on cohort level analyses. Ferrie, Rolf and Troesken (2012) exploit variation in the use of lead water pipes across time and place in the early part of the 20th century to estimate a negative relationship between exposure to leaded water and later intelligence test scores among WWII army enlistees. Interestingly, the effects are

greatest for children from low SES homes, consistent with work showing that good nutrition can reduce lead absorption. The focus of much of the cohort-level analyses has been to link declining lead levels in US children in the 1970s and 1980s to the dramatic declines in crime witnessed since the mid-1990s, exploiting cross-area and cross-cohort variation in lead exposure, typically from the staggered timing of de-leading gasoline during this period. These studies are based on aggregate level data that link average lead levels in a given state or city during a cohort's first years of life to crime levels 15-20 years later. Examples include Masters et al. (1998), Reyes (2007), Nevin (2000 and 2007), Mielke and Zahran (2012), Grönqvist, Nilsson and Robling (2014) and Reyes (2014). This work supports a strong relationship between declining lead exposure early in life with declines in crime later in life, though because it's based on cohort level analysis has been subject to the criticism of ecological fallacy.

E. Challenges to Estimation of the Impact of Elevated Lead on Child Outcomes

Despite the large body of evidence showing a strong inverse relationship between elevated BLLs in childhood and cognitive and behavior outcomes, there is still uncertainty regarding the magnitude of the effect as well as its duration. This uncertainty arises from two sources: the strong confounding of the relationship between lead exposure and child outcomes, and measurement error in child BLLs.

Regarding the former, disadvantaged children are more likely to be exposed to lead through older, substandard housing, and as a result, are more likely to have elevated BLLs. Based on analysis of the 1999-2002 NHANES, the CDC reported that the average blood level for children aged 1-5 was 1.9 ug/dL, but for African American children this figure was 50% higher (2.8 ug/dL) and 30% higher for low income households (2.5 ug/dL) (CDC MMWR, 2005).

Bellinger (2008) highlights how the use of inadequate controls for SES in existing analyses undermines the ability to draw credible inferences from existing lead studies. In particular, he notes that "It has long been recognized that due consideration must be given to the possibility that any observed association between lead exposure and neurodevelopment is, in part, an artifact of residual confounding…Indeed, adjusting for SES and related covariates can result in reductions of 50% or more in the magnitude of lead's regression coefficient (e.g, Bellinger et al, 1992)."

The second estimation challenge is measurement error in child lead levels. Child lead measures are typically measured through blood serum. Measurement error can arise from contamination of the blood sample especially when the sample is a capillary sample (also known as a finger prick), which is a common measure due to its lower expense and minimal discomfort to the child. An alternative measure (venous) is more costly but less prone to sample contamination. Even without contamination, there is significant error in measurement. According to the CDC, the "ratio of imprecision to measurement value, particularly at BLLs <10ug/dL, is relatively high" (MMWR, 2002).[4] This can lead to considerable attenuation bias. Moreover, the half-life of lead in blood is relatively short (36 days). As such, BLLs only capture very recent exposure and will not necessarily capture the amount of lead that has settled in body organs and bone.[5] Because over the first 72 months of a child's life, a child is typically only sampled a small number of times, this problem introduces an additional source of measurement

[4] According to the CDC guidelines "Federal regulations allow laboratories that perform blood lead testing to operate with a total allowable error of ± 4 ug/dL or ±10% whichever is greater." (MMWR, 2007)

[5] "Deleterious health effects of lead resulting from long-term lead exposure will only be correlated with current blood-lead levels if lead exposure has been relatively constant over a long period of time, up to the time of sampling." Moreover, "Physiologically, the measurement of lead in blood is not a direct assessment of target organ dose, since the red cell is not a critical target for lead toxicity. Kinetically, blood is not a good analog for critical targets, such as soft tissue, because of the relatively short half-life of lead in blood as compared to target organs or bone." Mount Sinai School of Medicine. Accessed on 12/16/2014: http://research.mssm.edu/xrf/why.html

error , though with ambiguity with respect to any bias that may result.[6] Below we discuss how we address these potential sources of bias in estimates of the impact of BLLs on later child cognitive outcomes.

III. Data and Empirical Strategy

A. Overview of Estimation

To explore the extent to which racial disparities in exposure to lead can explain the racial gap in test scores, we proceed in three stages. First we document disparities in BLLs by race and income, examining both levels at the beginning of our period (1997) and trends over time. We then examine the relationship between BLLs in early childhood and later cognitive achievement as measured by scores on standardized reading and math tests. As part of this effort we address issues related to confounding and measurement error in BLLs, relying on instrumental variable techniques to identify the causal effect of lead levels on future cognitive achievement. For identification, we exploit variation in lead safe housing over time induced by a policy change that required landlords to certify that their homes were lead safe. Neighborhoods with old housing stock and high lead burden in 1997 were targeted, and lead levels declined significantly faster in these area than in others. Importantly, neighborhoods that gain more lead-safe housing over this period (as measured by the number of certificates) do not appear to have changed over time in other dimensions that might influence child test scores.

[6] If this results in BLLs consistently underestimating the amount of lead in the child's system, this would result in an overestimate of the impact of lead on child outcomes. However, it is also possible that this could result in overestimates of the amount of lead in the child's system if the measurement coincides with brief exposure that does not result in sustained BLLs.

We conclude by examining the role of lead in explaining the racial gap in test scores, conducting two exercises. We calculate how much of the decline in the gap in black-white test scores can be explained by declines in the gap in black-white lead levels over this period using our causal estimates.[7] We also directly examine the relationship between the falling gap in child lead levels and the falling black-white test scores in each county in Rhode Island over this period. Below we describe the data we use for this analysis and our strategy for the IV estimates of the impact of lead on cognitive achievement in greater detail.

B. Data

Our sample consists of all children in the state of RI born between 1997 and 2004 with at least one BLL measure before the age of six, and who are enrolled in any RI public school. The data on BLLs comes from the RI Department of Health (RIDOH) and includes the age at each test, the test method (capillary or venous), the census tract in which the child lived at the time of the test, and the BLL. These data are linked with data from the RI Department of Education (RIDE) that includes NECAP[8] scaled test scores in grades three through eight, whether and when the child received an Individual Education Plan (IEP), and school lunch (free or reduced) status. The data are also linked with birth certificate data for the child. The latter include: birth order, child gender, maternal race, maternal age and marital status at birth, maternal education, the child's birth weight and gestational age at birth as well as when the mother initiated prenatal

[7] We find no evidence that the causal impact of lead exposure on test scores differs by race. Therefore we use a single causal estimate of the effect for this calculation.

[8] NECAP stands for the New England Common Assessment Program. It is a series of exams developed collaboratively by the New Hampshire, Rhode Island and Vermont departments of education, with assistance from the National Center for the Improvement of Educational Assessments. The NECAP tests measure students' academic knowledge and skills relative to Grade Expectations which were created by teachers from the three states.

care. The birth certificate data include a mother identifier so siblings can be identified in the dataset.

The sample includes roughly 57,000 RI children born between 1997 and 2004 with linked BLL and test score data. As of 2002, 78 percent of all children in the state of RI were screened for lead compared with national rates of closer to 25% (RI DOH, 2002; Vivier et al 2001). Of 84,343 children born between 1997 and 2004 with BLL levels, 59,298 were linked with RIDE data, roughly consistent with rates of private school attendance in the state (Ewert, 2013). Of the 59,298 children with RIDE data, 57,213 have a valid third grade test score.

Children have on average 4.7 BLL measures over the first 72 months of life (median of 4), after which they are typically not tested.[9] To construct a single measure we calculate the arithmetic mean of all samples, but also consider alternative measures: The maximum level and the geometric mean.

While all children in our sample have a third grade reading test score, the number of children with 4^{th} – 8^{th} grade scores declines steadily so that only 18,000 children (those born in 1997 and 1998) have 8^{th} grade test scores. The decline in the availability of later test scores is due almost entirely to the age of the cohorts in 2010, the last year for which we have data. There does not appear to be any other significant source of attrition (eg – migration out of the state or absence of scores for other reasons such as IEP). For example, of those children born in 1997, we have 3^{rd} grade test scores for 7084 students and of those, we have 8^{th} grade test scores for 6756 of them, losing less than 5% of the sample.

To estimate the effect of early childhood lead levels on future child outcomes we exploit variation in BLLs induced by changes in policies and resources devoted to reducing exposure to lead among RI children over the period 1990-2010 in an instrumental variable (IV) framework.

[9] When there is a capillary and venous sample available for the same month, we drop the capillary measure.

Below we discuss the nature of RI lead mitigation policies that underlie the exogenous variation we exploit for identification and the construction of our instrument.

C. Background on Lead Mitigation Policies in RI

Rhode Island, and particularly its urban areas, is characterized by old housing, with much of it built prior to WWII.[10] As a result, many of the state's children are exposed to lead through deteriorating lead-based paint, with African American and low income children disproportionately exposed due to the fact they are more likely to live in old housing that may be poorly maintained. Previous work has established that within RI, children living in high (top quintile) poverty neighborhoods are nearly four times more likely to have elevated BLLs than those in low (bottom quintile) poverty neighborhoods (Vivier et al, 2011).

In an effort to reduce childhood exposure to lead through old deteriorating paint, RI state policy makers established programs to encourage landlords to mitigate lead hazards in the homes they rent. In particular, they provided landlords with training in the importance of lead hazard mitigation, information and training on how to reduce lead hazards in the homes in the least costly manner, and they required all landlords to get a lead-safe certificate for homes they plan to rent. There were no resources dedicated to enforcement and though non-compliance was illegal, landlords were not penalized if they did not comply. However, landlords could (and were) sued in civil court when children living in their homes were found to have elevated lead levels if the home did not have a lead-safe certificate.(add cite for lawsuits)

[10] In our sample, the average student lives in a tract in which 43% of the housing units are pre-war units, based on census data. This number increases to 49% for Providence County.

Over the period 1997-2010, the total number of lead-safe certificates issued to landlords increased from 333 to over 41,000. The state contracted with community based non-profit organizations to provide outreach to families and landlords in the four "core cities" of RI where the housing stock is the oldest, the families most disadvantaged and the lead burden the greatest. As a result, census tracts that witnessed the greatest increase in certificates issued per capita were characterized by older housing stock, a greater share of children with high lead levels in 1997, and a greater share of poor families (Figure 1).[11] This growth in the number of lead safe certificates provides the exogenously occurring variation in exposure to lead that is the basis of our empirical strategy.

A potential concern over our ability to use certificates as an exogenous source of variation in area lead levels is that areas that saw greater growth in certificates may have been characterized by other changes in demographic composition, for example, that could also influence test scores. This does not appear to be the case. Tracts with the greatest increase in certificates do not appear to have experienced other demographic changes over the period 1997-2010. They are no different in terms of changes in the share black, the share poor, or median family income over this period than tracts that witnessed no or small changes in certificates per capita (Figure 2).

D. Instruments and First Stage

We collected data on the date and address of all certificates issued in the state of RI from 1997-2010. For each tract and year we generated the total number of certificates that had been issued in that tract in the past five years as of the child's date of birth. We then constructed two

[11] Certificates are scaled by 100

measures to account for the fact that tracts differed in their lead burden and thus need for certificates. The first multiplied the number of certificates by the average lead level of all tests of children in the tract in 1997 (Certificates*Average Lead Levels in 1997). The second measure scaled the number of certificates by the number of housing units in the tract built before WWII (Certificates/Housing Units Built Pre-war).[12] Because these measures are linked with RI children based on the tract and year of birth, they are not a function of any (potentially endogenous) migration of the family after the birth of the child. The first stage is:

$$(1) \qquad Lead_i = \alpha_0 + \alpha_1 Certificates_{nt-5} + \alpha_2 \mathbf{X}_i^c + \alpha_3 \mathbf{X}_i^m + \alpha_4 \mathbf{X}^n_{tn} + \gamma_n + \gamma_t + \mu_i$$

The vector $\mathbf{X}^c$ includes the child's birth weight, gestation at birth, birth order, gender, measure of the child's free/reduced lunch status, and the month in which the child's mother initiated prenatal care (a measure included to capture prenatal investments). The vector $\mathbf{X}^m$ includes maternal characteristics including maternal age at birth, marital status at birth, race, and educational attainment (years of schooling). The vector $\mathbf{X}^n$ includes time-varying neighborhood (tract) characteristics from census data including share poor, median family income, the share black, share Hispanic, total housing units, share housing built pre-war, share housing built post 1979 and the natural log of the tract population. [13] Year of birth and tract fixed effects are also included.

The estimates reveal a strong negative relationship between the number of lead-safe certificates in the five years prior to birth interacted with the original (1997) lead levels in the tract, and child lead levels (Table 1, column 1). We present results with additional first stage

[12] The average lead level is actually the leave-out average (ie, removing the focal child from the calculation of the average).
[13] Values for intercensal years from linear interpolations between census years.

instruments – a quadratic in the number of certificates (column 2), and adding the number of certificates issued in the tract between birth and age 5 (columns 3 and 4). Finally, we recalculate the instrument as the number of certificates/housing units built pre WWII (Table 1, columns5-8). All subsequent instruments also appear to be predictive of child lead levels and the results are qualitatively similar. Ultimately we focus on results using the first instrument because it yields the most precise first stage with the highest F statistic on the excluded instrument. While we focus on this one instrument for most of the analysis, we present second stage estimates based on the other instruments for comparison in the robustness section.

IV. Results

A. Lead Levels and Child, Family and Neighborhood Characteristics

In RI, as in the rest of the nation, disadvantaged children are characterized by higher lead levels, as they are nationally. Among children born between 1997 and 2004 in RI, African American children have average lead levels of 4.8 compared with 4.2 for Hispanic children and 3.1 for white children. Likewise, children of less educated mothers have lead levels of 4.6 on average, compared to children with more educated mothers whose lead levels are 3.2. The same patterns are true if we classify children by whether they are eligible for free lunch or not (4.1 vs. 2.7), their mothers are single or married (4.2 vs. 3.0) and whether they live in a poor neighborhood (Table 2, column 1). We see the same pattern if we limit our sample to those living in the county of Providence, where most of the disadvantaged children live, and where the concentration of old housing is greatest (Table 2, column 2). For comparison, we present

differences in third grade reading test scores for these same groups. Groups with high lead levels are characterized by lower reading test scores across all types (Table 2, columns 3 and 4).[14]

B. Trends in Lead Levels Over Time

Overall, average lead levels have fallen dramatically over the past 12 years (Figure 3A). The average blood lead level for those born in 1997 was 4.25, falling to 2.5 for those born in 2004.[15] The declines in lead levels over this period were greatest for the most disadvantaged children: black children as well as those eligible for free lunch witnessed greater declines over this period than other groups, reducing, but not eliminating, the gap in lead levels (Figure 3B). However, inspection of the figures show that no only were the initial BLLs of free lunch students considerably lower than they were for African American children, but that the decline over time was also less steep. This is likely due to the fact that both generally and among the disadvantaged population in RI, African American children are disproportionately located in the four core cities which is where the highest concentration of old housing is located and the state targeted its efforts.

We investigate the role of the intervention in explaining the decline in these disparities. Figure 1 shows that neighborhoods with a greater share of African American and poor families received more certificates, with a stronger relationship for the former than the latter. We also estimate whether the certificates appear to have had a greater impact on the lead levels of certain groups within a neighborhood or tract. To do so we repeat the first stage interacting certificates with the following child characteristics: Race, free-lunch status, maternal education and child

[14] The higher lead burden born by disadvantaged children is not due entirely to neighborhood segregation. Even within a neighborhood, disadvantaged children are more likely to have elevated lead levels (Appendix Table 1).

[15] Changes in the distribution of average lead levels for the 1998 vs. 2004 birth cohort are in Appendix Figure 1.

gender. The results suggest that in addition to certificates being disproportionately issued in neighborhoods with a high share of black families (Figure 1), that conditional on the neighborhood, the certificates did disproportionately reduce the lead levels of African American children, consistent with their living in housing with greater risk of lead exposure (Table 3, column 1). Interactions with free lunch, gender and maternal education are smaller and imprecisely estimated (columns 2 and 3).

C. Lead Levels and Future Child Cognitive Achievement – Preliminary Evidence

Visual inspection reveals a strong linear relationship between child BLLs and reading test scores as averaged over grades 3-8 (Figure 4). Children with an average BLL of 0 score nearly 5 points higher (42% of a standard deviation) than those with BLLs of 5. Similar patterns are observed for math test scores, as well as for the probability of having an IEP which increases from 20 percent for those with 0 BLLs, to 27 percent for those with a BLL of 5 and over 30 percent for those with a BLL of 10.

For comparison we also graph average maternal years of schooling by child lead levels in the final panel of Figure 4. One also observes a clear linear and negative relationship between maternal schooling and elevated BLLs, underscoring the potential for confounding.

D. Lead Levels and Future Child Cognitive Achievement – IV Results

We present estimates of the impact of preschool BLLs on future academic achievement as measured by NECAP scaled scores. The equation estimated is as follows:

(2) $$Y_i = \beta_0 + \beta_1 \text{Lead} + \beta_2 \mathbf{X}_i^c + \beta_3 \mathbf{X}_i^m + \beta_4 \mathbf{X}^n_{tn} + \boldsymbol{\tau}_n + \boldsymbol{\tau}_t + \varepsilon_i$$

Where Yi is a measure of test scores (in reading or math, in grades 3-8), $\mathbf{X}^c$, $\mathbf{X}^m$ and $\mathbf{X}^n$ are defined as above and year of birth and tract fixed effects are also included. Child lead levels are instrumented based on the first stage described above. For the first set of results, the outcome is the average reading test score over all grades for which a score is available (grades three through eight). The OLS results with no controls except cohort fixed effects suggest that going from a lead level of 0 to 5 (the threshold at which the CDC recommends action), would decrease test scores by 5 points or 38% of a standard deviation (Table 4, column 1). In columns 2 and 3, we add more controls including tract FE. The estimated effects decline in magnitude but are still precisely estimated and represent non-negligible effects.

IV estimates of the impact of average lead levels on child reading test scores are larger than the tract FE estimates and suggest that going from a lead level of 0 to 5 would decrease average test scores by nearly 9 points, or 67% of a standard deviation (Table 5, column 4). This larger IV estimate is consistent with considerable measurement error in lead levels. We explore the potential attenuation bias inherent in a mismeasured lead level by constructing an additional measure of lead based on a single, randomly drawn lead BLL for each child. This measure should reflect greater classical measurement error than the one based on multiple measures and this should be reflected in attenuation of the OLS estimate. When we regress test scores on this measure (Table 4, column 5), the relationship between lead levels and test scores is 69% lower (-0.087) than the estimate based on a lead level constructed from an average of multiple test scores (-0.279). These results are consistent with considerable attenuation bias from mismeasurement of lead levels, which is why we prefer IV the estimates.

We also present estimates that include mother FE, limiting our comparison to differences across siblings. Including a mother FE significantly reduces the coefficient estimate to -0.115 (Table 4, column 6). This would be consistent with both omitted variable bias in the estimates that include only tract FE, but is also consistent with previous work showing that the inclusion of a maternal FE can exacerbate the attenuation bias from measurement error (Griliches and Hausman, 1986). Interestingly, when we include the maternal FE and instrument for lead levels, the estimate is considerably larger than the maternal FE estimator (-4.8) though very imprecisely estimated (Table 4, column 7). Given the loss of precision with mother FE, we focus on IV with tract FE in what follows.

E. Extensions: Achieving Proficiency, Math Scores and Changes Over Time

The NECAP tests are used primarily to assess the extent to which students have met certain benchmarks. A score of at least 40 indicates "proficiency" in the subject (71% of students for reading), while a score below 30 indicates "substantial deficiency" (10%) and a score of greater than 56 indicates "passing with distinction" (21%). We examine the extent to which lead levels affect a student's reaching each of these three benchmarks in both reading and math test scores as well as changes in the effects of lead over time.

We find the largest effects of elevated lead in the lower tail of the distribution of test scores. Elevated lead levels are associated with a greater likelihood of substantial deficiency in both Reading (Table 5, panel A, columns 1 and 2) and Math (Table 5, Panel B, columns 1 and 2). Specifically, students with a lead level of 5 are 34 percentage points more likely to be substantially deficient in reading and 27 percentage points more likely to be substantially

deficient in math. They are 66 percentage points more likely to not meet the standard of proficiency in reading (Table 5, Panel A, columns 3 and 4). There are no effects found at the high end of the distribution of test scores for either reading or math.

We also explore the extent to which these estimated effects increase or decrease over time by examining the impact of lead on reaching proficiency in 3rd, 6th and 8th grade. The estimated effects do not decline over time and remain large (Table 6).

Finally, we explore whether the effects are heterogeneous across children. Specifically we estimate whether the estimated effect of lead on reading scores differs by child race or school lunch status. The results, not presented here, are very imprecisely estimated (particularly for the IV estimates) but suggest no significant heterogeneity by race or school lunch status.

F. Robustness

We consider additional specifications in order to assess the robustness of our results. First we consider alternative measures of lead. To rule out that the possibility that the results are be driven by outliers, we trim observations with lead levels in excess of 25 and the results are unchanged (Table 7 column1 and 2). We also consider as an alternative measure of lead the single highest measure recorded for each child (Max lead level). This measure is negatively and significantly associated with children's test scores though the estimates are lower in magnitude in both the FE and IV FE specifications (columns 3 and 4). We try to account for the timing of the lead measures in a third alternative measure. In particular, we assume that between two lead measures, lead decays (or increases) linearly, interpolate lead measures for non-tested months based on this assumption, and calculate a mean. These results (columns 5 and 6) are smaller but

still large. Finally, we calculate the geometric mean of lead as is sometimes done in this literature, which yields somewhat larger point estimates (columns 7 and 8).

Finally, we present IV estimates based on alternative sets of instruments: certificates at birth*initial(1997)lead level and is square (column 2), adding the number of certificates gained between birth and age 5 (columns 3 and 4) and recalculating the IV as the number of certificates/pre-war housing units (columns 5-7). The IV estimates across all specifications range from -0.823 to -1.7 and the precision also varies. The strongest and most precise estimates are based on the instrument with the strongest first stage (certificates*1997 lead levels), but even the smallest point estimate suggests a strong relationship.

V. Implications for Disparities in Child Educational Outcomes

Finally we explore whether and to what extent African American children's greater lead burden can explain racial disparities in test scores. We previously documented how RI state efforts to reduce lead levels among RI children disproportionately targeted urban neighborhoods with a greater initial lead burden and thus a greater share of African American and low income families (Figure 1). Even within neighborhoods, African American children's lead levels appear to have been disproportionately affected by state efforts to ensure that homes were lead safe (Table 3) and as a result of these efforts, the lead levels of African American, and to a lesser extent, low income children have fallen disproportionately faster (Figure 3B).

The decline in racial disparities in lead levels over this period was accompanied by a decline in racial disparities in test scores as well. We present the distribution of third grade reading scores by race and year of birth (1998 vs. 2004) in order to examine how the gap has

changed over time (Figure 5). It is clear that the test scores of white children have improved slightly over this period, but black test scores have improved considerably more, thereby reducing the racial gap in test scores. Calculating disparities based on average test scores by race, we find that the racial gap in test scores fell from 9.2 points for those born in 1997 to 6.3 for those born in 2004, while the gap in lead scores fell from 2.2 to 0.9 over this period. Based on the full range of our causal estimates of the impact of lead on test scores, we calculate that the falling disparity in lead levels would correspond to between a 1.1 and 2.2 point decline in the racial gap in test scores, explaining 37-76 % decline in the racial test score gap witnessed over this period. This does not simply represent trends in lead levels and test scores by family income. When we examine trends in disparities in lead and test scores by free lunch status, the declines in both are considerably smaller: over this same period, the income gap (linear difference) in lead levels fell from 1.83 to 0.99, while the test score gap fell from 9.3 to 8.4 (67% of a standard deviation to 60%). That the decline in the disparity in lead levels by race was so much larger than the decline by income is likely due to two factors: that African American children bore a much greater lead burden in 1997 than poor children more generally, and that African American children were more likely to reside in areas of the state that were targeted for lead hazard mitigation – the four core urban centers.[16]

We examine the relationship directly, relating racial gaps in lead levels to racial gaps in test scores at the tract (n=217) and county (n=5) level. Specifically, we calculate for each tract (county) and birth cohort, racial disparities in lead levels and the racial disparities in test scores,

[16] These differences in trends by income and race are comparable to national trends. According to the CDC, the share of BLLs above 5 ug/ul fell more quickly for African Americans than for poor children over the period 1999-2010. In particular the share of African American children with BLLs above 5 fell from 18.5 to 5.6 of the national population, while for children below 130% of the FPL, the share fell from 12.9 to 4.4. (MMWR, 2013)

as measured by differences in mean levels.[17] We then regress disparities in test scores (white-black, a positive number) on disparities in lead levels (white-black, a negative number) including tract (county) and year of birth fixed effects, as well as some controls for time varying characteristics of the tract (county) from census data. The tract (county) fixed effect estimates show that the lower the racial gap in lead levels, the lower the gap in test scores, with a 1 ug/dl decline in the gap in lead levels corresponding to between a .4 and 1.4 point drop in the black-white test score gap (Table 9, columns 1 and 3).[18] When we instrument for that black-white gap in lead levels with the number of certificates (Table 9, columns 2 and 4), the resulting estimates are slightly larger, but imprecise and not statistically significantly different from the OLS estimates, To show this relationship graphically, we plot the relationship between racial disparities in test scores and disparities lead levels for each county*year (left panel of Figure 6) as well as the predicted disparities in lead levels (based on the first stage) in the right panel of Figure 6. A clear negative relationship emerges.

VI. Conclusions

The racial gap in family income has, depending on measure considered, either held steady or increased over time.[19] As a result, one would not necessarily have expected the racial gap in child test scores to decline, but it did. We consider a new explanation for both racial disparities in test scores and their recent decline – African American children's disproportionate exposure to

[17] This generates a dataset of 40 observations – 8 birth cohorts for each of the five counties in RI.
[18] We do not present the first stage estimates, but the instrument does significantly predict reductions in lead levels within tract and county over time.
[19] The share of African Americans in poverty increased from 23 to 28% between 2000 and 2011, after years of steady declines and for whites it increased from 7 to 10% (Pew Research Center). The ratio of White to Black income has held steady since 1983 to 2010, increasing from 1.8 to 1.9 over this period; however, the wealth ratio has increased from 5.3 to 5.7 (Urban Institute, Interactive Race Graphic).

environmental toxins that can affect cognitive achievement. We find that since 1997, when the state of RI instituted measures to reduce lead hazards in the homes of RI families, lead levels fell across the state, but significantly more so for African American children. This is likely because their lead levels were considerably higher than other children in the state in 1997, including other low income children, and African American families were disproportionately located in high-concentration poverty areas where outreach efforts were focused. We find that this translated into reductions in the black-white test score gap in RI witnessed over this period.

Jencks and Phillips (2011) have argued that eliminating black-white disparities in test scores would dramatically reduce not only educational inequality but economic inequality as well. Moreover, they argue that traditional sources of the gap (eg, inequalities in school inputs or family income), likely explain only a small share of the racial gap in test scores, and alternative sources of the gap should be considered. Our results suggest that environmental regulation, if targeted to those children at greatest risk, is effective in reducing environmental exposure to toxins, then declines in racial disparities in exposure, test scores and, perhaps, economic outcomes, are likely to follow.

References - incomplete

US Dept of Education NAEP 2012 Trends in Academic Progress NCES 2013-456.

Brown, Phil "Race, Class, and Environmental Health: A Review and Systematization of the Literature." Environmental Research Volume 69, Issue 1, April 1995, Pages 15–30

Brulle, Robert J. and David N. Pellow, ENVIRONMENTAL JUSTICE: Human Health and Environmental Inequalities Annu. Rev. Public Health 2006. 27:103–24

O'Neil SG "Superfund: evaluating the impact of executive order 12898**."** *Environ Health Perspec* 2007**,** 115:1087-1093.

Burda, Martin and Matthew Harding "Environmental Justice: Evidence from Superfund Cleanup durations" Journal of economic behavior and organization 107: 380-401. 2014.

Currie, Janet. "Inequality at Birth: Some Causes and Consequences," American Economic Review, May 2011, 101 #3, 1-22.

Currie, Janet, Davis, Lucas, Greenstone, Michael and Reed Walker "Environmental Health Risks and Housing Values: Evidence from 1600 Toxic Plant Openings and Closings," American Economic Review, February 2015

Ewart, Stephanie. "The Decline in Private School Enrollment" SEHSD Working Paper Number FY12- 117 January, 2013, U.S. Census Bureau
Social, Economic, and Housing Statistics Division

Griliches, Zvi and Jerry Hausmn. 1986. "Errors in Variables in Panel Data."
Journal of Econometrics. 31:93-118.

Grönqvist, Hans, Nilsson, Peter and Per-Olof Robling, Early-childhood Lead Exposure and Criminal Behavior: Lessons from the Swedish Phase-out of Leaded Gasoline SOFI working paper 2014/9, October, 2014, Stockholm University.

Lead Poisoning in Rhode Island: The Numbers (2002). Providence, RI: Rhode Island Department of Health

U.S. Environmental Protection Agency, "Lead and a Healthy Diet: What You Can Do to Protect Your Child," 2015, www2.epa.gov/sites/production/files/documents/**nutrition**.pdf.

CDC MMR Weekly "Blood Lead Levels in Children Aged 1–5 Years — United States, 1999–2010 **April 5, 2013 / 62(13);245-248**

Table 1: First Stage - Predicting a Child's Average Lead Levels

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Certificates at birth/1000*Original (1997) Lead Level	-0.653	-1.132	-0.507	-0.811					-0.365	-0.933
	[0.0586]	[0.111]	[0.0610]	[0.119]					[0.104]	[0.156]
Certificates at birth/1000* Original (1997) Lead Level Squared		0.230		0.140						0.223
		[0.0452]		[0.0468]						[0.0456]
Gain in Certificates Birth Through Age 5 * Original (1997) Lead Leve			-0.230	-0.209						
			[0.0266]	[0.0275]						
Certificates at birth/pre-war units					-0.0210	-0.0460	-0.0208	-0.0448		
					[0.00331]	[0.00608]	[0.00332]	[0.00614]		
Certificates at birth/pre-war units squared						0.000732		0.000703		
						[0.000150]		[0.000151]		
Certificates Birth-Age 5/pre-war units							-0.00192	-0.00128		
							[0.000945]	[0.000955]		
Certificates*Number of Tests									-0.871	-0.551
									[0.278]	[0.285]
Predicted # of tests									3.302	3.295
									[0.0480]	[0.0480]
Married at Birth	-0.237	-0.238	-0.237	-0.238	-0.239	-0.238	-0.239	-0.238	0.0190	0.0186
	[0.0214]	[0.0214]	[0.0214]	[0.0214]	[0.0214]	[0.0214]	[0.0214]	[0.0214]	[0.0217]	[0.0217]
Maternal age at birth	-0.0284	-0.0284	-0.0285	-0.0285	-0.0283	-0.0283	-0.0283	-0.0283	-0.0426	-0.0426
	[0.00174]	[0.00174]	[0.00174]	[0.00174]	[0.00174]	[0.00174]	[0.00174]	[0.00174]	[0.00174]	[0.00174]
Mother African-American	0.564	0.568	0.576	0.577	0.546	0.551	0.548	0.552	0.702	0.705
	[0.0354]	[0.0354]	[0.0354]	[0.0354]	[0.0353]	[0.0353]	[0.0353]	[0.0353]	[0.0352]	[0.0352]
Mother Hispanic	-0.332	-0.327	-0.317	-0.316	-0.350	-0.347	-0.349	-0.346	-3.348	-3.352
	[0.0367]	[0.0367]	[0.0367]	[0.0367]	[0.0366]	[0.0366]	[0.0366]	[0.0366]	[0.0574]	[0.0574]
Mother White	0.245	0.251	0.260	0.262	0.224	0.231	0.226	0.232	1.443	1.448
	[0.0271]	[0.0271]	[0.0271]	[0.0271]	[0.0270]	[0.0270]	[0.0270]	[0.0271]	[0.0320]	[0.0320]
Mother Asian	0.156	0.159	0.163	0.164	0.149	0.153	0.151	0.153	-0.0579	-0.0554
	[0.0462]	[0.0462]	[0.0462]	[0.0462]	[0.0462]	[0.0462]	[0.0462]	[0.0462]	[0.0463]	[0.0463]
Maternal education in years	-0.0518	-0.0518	-0.0519	-0.0519	-0.0520	-0.0520	-0.0520	-0.0520	0.164	0.164
	[0.00371]	[0.00370]	[0.00370]	[0.00370]	[0.00371]	[0.00371]	[0.00371]	[0.00371]	[0.00484]	[0.00484]
Mother has at least one risk factor	0.170	0.170	0.170	0.170	0.172	0.172	0.172	0.172	0.122	0.122
	[0.0223]	[0.0223]	[0.0223]	[0.0223]	[0.0223]	[0.0223]	[0.0223]	[0.0223]	[0.0223]	[0.0223]
Always free/reduced lunch	0.374	0.376	0.379	0.380	0.371	0.372	0.372	0.373	-1.074	-1.071
	[0.0267]	[0.0267]	[0.0267]	[0.0267]	[0.0267]	[0.0267]	[0.0267]	[0.0267]	[0.0339]	[0.0339]
Sometimes free/reduced lunch	0.246	0.246	0.245	0.245	0.247	0.247	0.247	0.247	-0.396	-0.394
	[0.0238]	[0.0238]	[0.0238]	[0.0238]	[0.0238]	[0.0238]	[0.0238]	[0.0238]	[0.0257]	[0.0257]
male	0.168	0.168	0.169	0.168	0.168	0.168	0.168	0.168	-0.125	-0.125
	[0.0159]	[0.0159]	[0.0159]	[0.0159]	[0.0159]	[0.0159]	[0.0159]	[0.0159]	[0.0164]	[0.0164]
Birth order	0.208	0.208	0.208	0.208	0.207	0.207	0.207	0.207	0.239	0.239
	[0.00868]	[0.00868]	[0.00868]	[0.00868]	[0.00869]	[0.00869]	[0.00869]	[0.00869]	[0.00871]	[0.00871]
Birth weight/100	-0.0147	-0.0147	-0.0147	-0.0147	-0.0146	-0.0146	-0.0146	-0.0146	0.00470	0.00469
	[0.00180]	[0.00180]	[0.00180]	[0.00180]	[0.00180]	[0.00180]	[0.00180]	[0.00180]	[0.00182]	[0.00182]
Gestation in Weeks	0.0387	0.0388	0.0386	0.0387	0.0385	0.0386	0.0385	0.0386	0.00991	0.0100
	[0.00509]	[0.00509]	[0.00509]	[0.00509]	[0.00510]	[0.00509]	[0.00510]	[0.00509]	[0.00511]	[0.00511]
Month prenatal care initiated	0.0847	0.0844	0.0843	0.0842	0.0846	0.0842	0.0845	0.0842	-0.00539	-0.00569
	[0.00728]	[0.00728]	[0.00728]	[0.00728]	[0.00729]	[0.00729]	[0.00729]	[0.00729]	[0.00744]	[0.00744]
Share of tract black	-0.476	-0.0169	0.977	1.124	-0.514	-0.507	-0.496	-0.496	-15.19	-14.67
	[0.954]	[0.958]	[0.969]	[0.970]	[0.959]	[0.959]	[0.959]	[0.959]	[0.975]	[0.980]
Share of tract below poverty	1.023	1.109	0.716	0.796	1.879	1.837	1.888	1.845	1.164	1.176
	[0.737]	[0.737]	[0.738]	[0.738]	[0.733]	[0.733]	[0.733]	[0.733]	[0.735]	[0.734]
Median family income in tract ($1000)	0.0181	0.0171	0.0151	0.0148	0.0203	0.0196	0.0198	0.0193	0.000925	2.43e-05
	[0.00426]	[0.00427]	[0.00428]	[0.00428]	[0.00426]	[0.00426]	[0.00427]	[0.00427]	[0.00427]	[0.00427]
Housing units in tract in 1000s	0.0598	0.0530	0.0440	0.0413	0.0648	0.0596	0.0610	0.0573	-0.0333	-0.0390
	[0.0113]	[0.0114]	[0.0114]	[0.0115]	[0.0113]	[0.0114]	[0.0115]	[0.0115]	[0.0114]	[0.0115]
Share housing in tract built post 1979	1.113	1.235	1.266	1.326	1.267	1.281	1.437	1.393	-1.104	-0.996
	[0.599]	[0.599]	[0.599]	[0.599]	[0.601]	[0.601]	[0.607]	[0.607]	[0.598]	[0.598]
Share pre war housing in tract	-3.400	-3.162	-2.616	-2.542	-4.672	-4.489	-4.624	-4.465	2.789	3.035
	[0.490]	[0.493]	[0.498]	[0.499]	[0.471]	[0.472]	[0.471]	[0.472]	[0.496]	[0.499]
Ln(tract population)	-1.439	-1.293	-1.240	-1.170	-1.515	-1.352	-1.521	-1.363	2.759	2.874
	[0.418]	[0.419]	[0.418]	[0.419]	[0.419]	[0.420]	[0.419]	[0.420]	[0.421]	[0.422]
Observations	53,994	53,994	53,994	53,994	53,994	53,994	53,994	53,994	53,994	53,994
R-squared	0.210	0.211	0.211	0.211	0.209	0.210	0.210	0.210	0.262	0.263
F-test	124.2	75.05	99.56	69.35	40.31	32.14	22.23	22.03	64.98	51.30

Table 2: Average Lead Levels and Reading Test Scores by Child, Family and Neighborhood Characteristics

	Lead Levels		3rd Grade Reading Scores	
	RI	Providence	RI	Providence
Black	4.77	4.83	40	40
Hispanic	4.2	4.21	38	38
White	3.14	3.39	48	46
Difference (black-white)	1.63	1.44	-8	-6
Difference (Hispanic-white)	1.06	0.82	-10	-8
Maternal education <HS	4.62	4.76	39	38
Maternal education >=HS	3.19	3.46	47	46
Difference	1.43	1.3	-8	-8
Free lunch	4.1	4.3	41	41
No free lunch	2.68	2.78	50	50
Difference	1.42	1.52	-9	-9
Unmarried	4.2	4.42	41	41
Married	2.97	3.19	48	47
Difference	1.23	1.23	-7	-6
Poor neighborhood	4.36	4.43	41	40
Non - poor neighborhood	2.95	3.1	48	48
Difference	1.41	1.33	-7	-8
High Share Black Neighborhood (white children)	4.15	4.18	43	43
Low Share Black Neighborhood (white children)	2.94	3.08	48	47
Difference	1.21	1.1	-5	-4

Table 3: Certificates and Lead Levels: Heterogeneity by Child Characteristic

	(1)	(2)	(3)	(4)
Certificates at birth/1000*Original (1997) Lead Level	-0.690	-0.570	-0.701	-0.827
	[0.115]	[0.239]	[0.126]	[0.257]
Certificates*Black	-0.326			
	[0.151]			
Certificates*Free Lunch		-0.206		
		[0.231]		
Certificates*male			-0.108	
			[0.114]	
Certificates*Maternal education				0.00587
				[0.0203]
Married at Birth	-0.230	-0.231	-0.231	-0.231
	[0.0271]	[0.0271]	[0.0271]	[0.0271]
Maternal age at birth	-0.0288	-0.0289	-0.0289	-0.0289
	[0.00219]	[0.00219]	[0.00219]	[0.00219]
Mother African-American	0.806	0.762	0.761	0.761
	[0.0516]	[0.0473]	[0.0473]	[0.0473]
Mother Hispanic	-0.205	-0.200	-0.202	-0.200
	[0.0505]	[0.0505]	[0.0505]	[0.0506]
Mother White	0.429	0.422	0.423	0.423
	[0.0393]	[0.0393]	[0.0392]	[0.0392]
Mother Asian	0.277	0.276	0.277	0.278
	[0.0602]	[0.0602]	[0.0602]	[0.0602]
Maternal education in years	-0.0562	-0.0562	-0.0562	-0.0567
	[0.00460]	[0.00460]	[0.00460]	[0.00488]
Mother has at least one risk factor	0.166	0.166	0.166	0.166
	[0.0300]	[0.0300]	[0.0300]	[0.0300]
Always free/reduced lunch	0.482	0.489	0.483	0.482
	[0.0355]	[0.0364]	[0.0355]	[0.0356]
Sometimes free/reduced lunch	0.259	0.264	0.259	0.258
	[0.0289]	[0.0295]	[0.0289]	[0.0290]
male	0.166	0.166	0.172	0.166
	[0.0199]	[0.0199]	[0.0209]	[0.0199]
Birth order	0.213	0.213	0.213	0.213
	[0.0110]	[0.0110]	[0.0110]	[0.0110]
Birth weight/100	-0.0150	-0.0150	-0.0150	-0.0150
	[0.00224]	[0.00224]	[0.00224]	[0.00224]
Gestation in Weeks	0.0402	0.0401	0.0401	0.0401
	[0.00637]	[0.00637]	[0.00637]	[0.00637]
Month prenatal care initiated	0.0956	0.0954	0.0955	0.0955
	[0.00915]	[0.00915]	[0.00915]	[0.00915]
Observations	53,994	53,994	53,994	53,994
R-squared	0.100	0.100	0.100	0.100

Table 4: Lead and Reading Test Scores (Avg)

	(1) OLS	(2) OLS	(3) Tract FE	(4) FE IV	(5) Tract FE	(6) Mom FE	(7) Mom FE IV
Avg lead level	-1.029	-0.277	-0.279	-1.763		-0.115	-4.847
	[0.0188]	[0.0188]	[0.0189]	[0.778]		[0.0402]	[3.298]
Randomly Drawn Lead Level					-0.0872		
					[0.0141]		
Married at Birth		0.907	0.880	0.521	0.926	-0.596	-0.00751
		[0.120]	[0.120]	[0.227]	[0.120]	[0.471]	[0.798]
Maternal age at birth		0.0998	0.0951	0.0523	0.100	0.256	-0.271
		[0.00966]	[0.00968]	[0.0246]	[0.00969]	[0.265]	[0.533]
Mother African-American		-0.690	-0.733	0.381	-0.883		
		[0.206]	[0.208]	[0.624]	[0.209]		
Mother Hispanic		-0.119	-0.278	-0.567	-0.237		
		[0.222]	[0.224]	[0.281]	[0.225]		
Mother White		1.016	1.052	1.694	0.975		
		[0.169]	[0.173]	[0.382]	[0.173]		
Mother Asian		3.073	2.994	3.415	2.931		
		[0.265]	[0.268]	[0.358]	[0.268]		
Maternal education in years		0.734	0.718	0.632	0.730		
		[0.0203]	[0.0204]	[0.0501]	[0.0204]		
Mother has at least one risk factor		-0.677	-0.682	-0.417	-0.713		
		[0.133]	[0.133]	[0.197]	[0.133]		
Always free/reduced lunch		-4.435	-4.349	-3.662	-4.442	1.818	1.799
		[0.155]	[0.156]	[0.396]	[0.156]	[0.801]	[1.167]
Sometimes free/reduced lunch		-3.197	-3.114	-2.715	-3.169	0.603	1.232
		[0.127]	[0.127]	[0.248]	[0.128]	[0.748]	[1.174]
male		-4.106	-4.099	-3.838	-4.139	-3.962	-3.497
		[0.0880]	[0.0879]	[0.165]	[0.0880]	[0.154]	[0.394]
Birth order		-0.813	-0.808	-0.493	-0.849	-0.669	-0.698
		[0.0487]	[0.0488]	[0.173]	[0.0488]	[0.202]	[0.294]
Birth weight/100		0.121	0.121	0.0983	0.124	0.0919	0.0420
		[0.00991]	[0.00991]	[0.0157]	[0.00992]	[0.0231]	[0.0484]
Gestation in Weeks		-0.0432	-0.0439	0.0153	-0.0494	-0.0474	0.0423
		[0.0282]	[0.0282]	[0.0429]	[0.0282]	[0.0613]	[0.109]
Month prenatal care initiated		-0.0481	-0.0660	0.0801	-0.0854	-0.0175	0.188
		[0.0403]	[0.0403]	[0.0875]	[0.0404]	[0.0823]	[0.187]
Share of tract black		-2.106	9.348	11.38	9.012	-0.284	6.662
		[1.010]	[6.345]	[6.762]	[6.355]	[2.792]	[6.321]
Share of tract below poverty		-0.407	13.56	15.59	13.24	1.444	1.507
		[0.905]	[5.771]	[6.166]	[5.780]	[2.560]	[3.727]
Median family income in tract ($1000)		0.0396	-0.0287	-0.000126	-0.0338	0.00292	0.0229
		[0.00378]	[0.0324]	[0.0372]	[0.0324]	[0.0127]	[0.0232]
Housing units in tract in 1000s		0.0433	0.0696	0.170	0.0556	-0.0650	0.0396
		[0.0152]	[0.0608]	[0.0829]	[0.0609]	[0.0443]	[0.0974]
Share housing in tract built post 1979		-0.261	7.373	10.18	6.930	-1.220	-0.631
		[0.517]	[4.483]	[4.942]	[4.490]	[1.653]	[2.441]
Share pre war housing in tract		2.038	10.67	3.189	11.69	1.340	5.524
		[0.375]	[3.668]	[5.503]	[3.673]	[1.094]	[3.323]
Ln(tract population)		-0.411	0.797	-2.661	1.282	1.036	-0.164
		[0.272]	[3.070]	[3.705]	[3.075]	[0.780]	[1.411]
Observations	60,582	57,310	57,310	57,310	57,310	59,227	59,227
R-squared	0.048	0.249	0.140		0.138	0.061	

Note: Cohort fixed effects included in all regressions

Table 5: Lead Levels and Third Grade Reading and Math Proficiency

	(1)	(2)	(3)	(4)	(5)	(6)
	Substantially below Proficient <30		Proficient >=40		with Distinction >56	
Panel A: Reading Proficiency	Tract FE	IV	Tract FE	IV	Tract FE	IV
Avg lead level	0.00547	0.0696	-0.00870	-0.132	-0.00288	0.00677
	[0.000537]	[0.0235]	[0.000794]	[0.0372]	[0.000728]	[0.0284]
Married at Birth	-0.00753	0.00736	0.0174	-0.0112	0.0143	0.0165
	[0.00338]	[0.00665]	[0.00500]	[0.0105]	[0.00458]	[0.00803]
Maternal age at birth	-0.000739	0.00111	0.00228	-0.00126	0.00185	0.00213
	[0.000273]	[0.000743]	[0.000404]	[0.00118]	[0.000370]	[0.000897]
Mother African-American	0.00247	-0.0460	-0.0246	0.0685	-0.0169	-0.0242
	[0.00590]	[0.0190]	[0.00873]	[0.0300]	[0.00800]	[0.0229]
Mother Hispanic	0.0248	0.0380	-0.0345	-0.0600	-0.000736	0.00126
	[0.00629]	[0.00859]	[0.00930]	[0.0136]	[0.00853]	[0.0104]
Mother White	-0.0178	-0.0446	0.0462	0.0978	0.0224	0.0183
	[0.00489]	[0.0113]	[0.00724]	[0.0178]	[0.00663]	[0.0136]
Mother Asian	-0.0469	-0.0646	0.0763	0.110	0.0476	0.0449
	[0.00750]	[0.0107]	[0.0111]	[0.0169]	[0.0102]	[0.0129]
Maternal education in years	-0.00779	-0.00417	0.0186	0.0116	0.0170	0.0175
	[0.000574]	[0.00148]	[0.000849]	[0.00233]	[0.000778]	[0.00178]
Mother has at least one risk factor	0.0165	0.00562	-0.0155	0.00526	-0.0109	-0.0126
	[0.00374]	[0.00579]	[0.00553]	[0.00915]	[0.00507]	[0.00698]
Always free/reduced lunch	0.0495	0.0188	-0.123	-0.0640	-0.101	-0.106
	[0.00443]	[0.0123]	[0.00656]	[0.0195]	[0.00601]	[0.0149]
Sometimes free/reduced lunch	0.0205	0.00387	-0.0669	-0.0349	-0.0873	-0.0898
	[0.00361]	[0.00732]	[0.00533]	[0.0116]	[0.00489]	[0.00884]
male	0.0507	0.0401	-0.0894	-0.0691	-0.0721	-0.0737
	[0.00248]	[0.00478]	[0.00367]	[0.00756]	[0.00336]	[0.00577]
Birth order	0.0121	-0.00155	-0.0201	0.00613	-0.0161	-0.0181
	[0.00138]	[0.00523]	[0.00203]	[0.00828]	[0.00187]	[0.00632]
Birth weight/100	-0.00209	-0.00113	0.00375	0.00190	0.00286	0.00300
	[0.000279]	[0.000472]	[0.000413]	[0.000746]	[0.000379]	[0.000569]
Gestation in Weeks	0.00106	-0.00150	-0.00263	0.00230	-0.00168	-0.00206
	[0.000794]	[0.00130]	[0.00117]	[0.00205]	[0.00108]	[0.00157]
Month prenatal care initiated	0.00279	-0.00334	-0.00455	0.00724	-0.00186	-0.00279
	[0.00114]	[0.00259]	[0.00169]	[0.00410]	[0.00155]	[0.00313]
Share of tract black	0.464	0.374	-0.0862	0.0865	0.375	0.361
	[0.179]	[0.203]	[0.264]	[0.322]	[0.242]	[0.246]
Share of tract below poverty	-0.286	-0.361	0.342	0.485	0.229	0.218
	[0.162]	[0.184]	[0.239]	[0.291]	[0.219]	[0.222]
Median family income in tract ($1000)	0.00123	-6.90e-05	-0.00373	-0.00125	0.00134	0.00114
	[0.000905]	[0.00112]	[0.00134]	[0.00178]	[0.00123]	[0.00136]
Housing units in tract in 1000s	0.00440	-0.000378	-0.00255	0.00663	0.00887	0.00815
	[0.00171]	[0.00261]	[0.00253]	[0.00412]	[0.00232]	[0.00314]
Share housing in tract built post 1979	0.0975	-0.0163	0.00565	0.224	0.332	0.315
	[0.126]	[0.147]	[0.186]	[0.233]	[0.170]	[0.178]
Share pre war housing in tract	-0.0839	0.253	0.165	-0.481	0.0582	0.109
	[0.103]	[0.169]	[0.152]	[0.267]	[0.140]	[0.204]
Ln(tract population)	-0.189	-0.0346	0.166	-0.129	-0.0944	-0.0712
	[0.0860]	[0.112]	[0.127]	[0.177]	[0.117]	[0.135]
Observations	53,994	53,994	53,994	53,994	53,994	53,994
R-squared	0.034		0.068		0.059	
Mean of Dependent Variable	10%		71%		21%	

	(1)	(2)	(3)	(4)	(5)	(6)
Panel B: Math Proficiency	Tract FE	IV	Tract FE	IV	Tract FE	IV
Avg lead level	0.00487	0.0543	-0.00491	0.00434	-0.00183	0.0182
	[0.000622]	[0.0258]	[0.000843]	[0.0332]	[0.000589]	[0.0234]
Observations	54,198	54,198	54,198	54,198	54,198	54,198
R-squared	0.032		0.067		0.035	
Mean of Dependent Variable	14%		60%		12%	

Table 6: Lead Levels and Proficiency Over Time

	(1)	(2)	(3)	(4)	(5)	(6)
	Grade 3 Reading		Grade 6 Reading		Grade 8 Reading	
	FE	IV	FE	IV	FE	IV
Avg lead level	-0.00870	-0.132	-0.00798	-0.0951	-0.00785	-0.145
	[0.000794]	[0.0372]	[0.000959]	[0.0512]	[0.00111]	[0.0810]
Married at Birth	0.0174	-0.0112	0.0186	-0.00388	0.0299	-0.00878
	[0.00500]	[0.0105]	[0.00643]	[0.0151]	[0.00776]	[0.0252]
Maternal age at birth	0.00228	-0.00126	0.00335	0.000962	0.00245	-0.00128
	[0.000404]	[0.00118]	[0.000520]	[0.00152]	[0.000623]	[0.00237]
Mother African-American	-0.0246	0.0685	-0.0261	0.0539	-0.0399	0.0964
	[0.00873]	[0.0300]	[0.0118]	[0.0488]	[0.0148]	[0.0831]
Mother Hispanic	-0.0345	-0.0600	-0.00417	-0.0161	-0.00316	-0.0241
	[0.00930]	[0.0136]	[0.0127]	[0.0159]	[0.0160]	[0.0250]
Mother White	0.0462	0.0978	0.0350	0.0764	0.0432	0.100
	[0.00724]	[0.0178]	[0.00993]	[0.0268]	[0.0122]	[0.0377]
Mother Asian	0.0763	0.110	0.107	0.126	0.107	0.128
	[0.0111]	[0.0169]	[0.0152]	[0.0204]	[0.0186]	[0.0281]
Maternal education in years	0.0186	0.0116	0.0185	0.0127	0.0173	0.00780
	[0.000849]	[0.00233]	[0.00108]	[0.00363]	[0.00130]	[0.00592]
Mother has at least one risk factor	-0.0155	0.00526	-0.0283	-0.0110	-0.00278	0.0347
	[0.00553]	[0.00915]	[0.00774]	[0.0134]	[0.00960]	[0.0257]
Always free/reduced lunch	-0.123	-0.0640	-0.136	-0.0886	-0.0965	-0.0296
	[0.00656]	[0.0195]	[0.00862]	[0.0295]	[0.0103]	[0.0420]
Sometimes free/reduced lunch	-0.0669	-0.0349	-0.0702	-0.0429	-0.0607	-0.0293
	[0.00533]	[0.0116]	[0.00658]	[0.0177]	[0.00780]	[0.0214]
male	-0.0894	-0.0691	-0.105	-0.0870	-0.0840	-0.0552
	[0.00367]	[0.00756]	[0.00468]	[0.0116]	[0.00559]	[0.0187]
Birth order	-0.0201	0.00613	-0.0259	-0.00631	-0.0206	0.00923
	[0.00203]	[0.00828]	[0.00263]	[0.0119]	[0.00322]	[0.0182]
Birth weight/100	0.00375	0.00190	0.00335	0.00201	0.00194	0.000299
	[0.000413]	[0.000746]	[0.000530]	[0.000987]	[0.000634]	[0.00130]
Gestation in Weeks	-0.00263	0.00230	-0.00431	-0.000503	-0.00464	0.000302
	[0.00117]	[0.00205]	[0.00152]	[0.00282]	[0.00182]	[0.00383]
Month prenatal care initiated	-0.00455	0.00724	-0.00290	0.00782	-0.00304	0.0137
	[0.00169]	[0.00410]	[0.00216]	[0.00676]	[0.00263]	[0.0105]
Observations	53,994	53,994	31,230	31,230	17,860	17,860
R-squared	0.068		0.077		0.072	

Table 7: Different Measures of Lead and Reading Test Scores (Avg)

	(1) OLS	(2) IV	(3) OLS	(4) IV	(5) OLS	(6) IV	(7) OLS	(8) IV
Average lead, truncate at 25	-0.282	-1.757						
	[0.0191]	[0.775]						
Max lead level			-0.142	-1.126				
			[0.0107]	[0.506]				
Lead area under curve					-0.256	-0.986		
					[0.0147]	[0.423]		
Geometric Mean of Lead							-0.315	-3.111
							[0.0207]	[1.722]
Married at Birth	0.880	0.525	0.898	0.551	0.865	0.627	0.883	0.294
	[0.120]	[0.225]	[0.120]	[0.220]	[0.120]	[0.184]	[0.121]	[0.388]
Maternal age at birth	0.0950	0.0524	0.0979	0.0618	0.0954	0.0736	0.0964	0.0214
	[0.00968]	[0.0246]	[0.00968]	[0.0213]	[0.00967]	[0.0161]	[0.00977]	[0.0476]
Mother African-American	-0.730	0.374	-0.801	0.174	-0.706	-0.0297	-0.723	1.157
	[0.208]	[0.620]	[0.208]	[0.549]	[0.208]	[0.445]	[0.210]	[1.183]
Mother Hispanic	-0.276	-0.553	-0.215	-0.157	-0.265	-0.386	-0.300	-0.868
	[0.224]	[0.277]	[0.224]	[0.242]	[0.224]	[0.239]	[0.226]	[0.436]
Mother White	1.054	1.690	0.995	1.433	1.036	1.334	1.066	2.203
	[0.173]	[0.380]	[0.173]	[0.292]	[0.173]	[0.247]	[0.174]	[0.729]
Mother Asian	2.993	3.397	2.981	3.438	3.032	3.364	3.014	3.822
	[0.268]	[0.352]	[0.268]	[0.371]	[0.267]	[0.334]	[0.269]	[0.586]
Maternal education in years	0.718	0.633	0.720	0.619	0.711	0.644	0.718	0.563
	[0.0204]	[0.0495]	[0.0204]	[0.0560]	[0.0204]	[0.0439]	[0.0206]	[0.0982]
Mother has at least one risk factor	-0.681	-0.418	-0.691	-0.412	-0.680	-0.531	-0.680	-0.236
	[0.133]	[0.197]	[0.133]	[0.202]	[0.133]	[0.160]	[0.134]	[0.314]
Always free/reduced lunch	-4.346	-3.661	-4.364	-3.573	-4.300	-3.791	-4.396	-3.277
	[0.156]	[0.396]	[0.156]	[0.440]	[0.156]	[0.335]	[0.157]	[0.713]
Sometimes free/reduced lunch	-3.112	-2.713	-3.130	-2.724	-3.095	-2.825	-3.143	-2.540
	[0.127]	[0.249]	[0.127]	[0.250]	[0.127]	[0.203]	[0.129]	[0.400]
male	-4.099	-3.842	-4.112	-3.858	-4.072	-3.854	-4.103	-3.669
	[0.0879]	[0.164]	[0.0879]	[0.161]	[0.0879]	[0.155]	[0.0888]	[0.286]
Birth order	-0.808	-0.499	-0.830	-0.573	-0.807	-0.634	-0.799	-0.206
	[0.0488]	[0.170]	[0.0487]	[0.142]	[0.0487]	[0.112]	[0.0493]	[0.369]
Birth weight/100	0.121	0.0985	0.122	0.102	0.120	0.107	0.120	0.0847
	[0.00991]	[0.0156]	[0.00991]	[0.0148]	[0.00990]	[0.0128]	[0.0100]	[0.0247]
Gestation in Weeks	-0.0438	0.0149	-0.0475	0.00475	-0.0472	-0.0245	-0.0452	0.0625
	[0.0282]	[0.0427]	[0.0282]	[0.0404]	[0.0282]	[0.0316]	[0.0285]	[0.0740]
Month prenatal care initiated	-0.0666	0.0733	-0.0741	0.0602	-0.0622	0.0269	-0.0631	0.203
	[0.0403]	[0.0848]	[0.0403]	[0.0814]	[0.0403]	[0.0660]	[0.0406]	[0.171]
Observations	57,270	57,310	57,310	57,310	57,309	57,309	56,278	56,278
R-squared	0.140		0.140		0.142		0.142	

Table 8: Second Stage Estimates of Lead and Average Reading Scores from Alternative IVs

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Avg lead level	-1.763	-1.457	-0.823	-0.947	-1.082	-1.347	-1.029
	[0.778]	[0.631]	[0.528]	[0.526]	[0.904]	[0.825]	[0.775]
Married at Birth	0.521	0.595	0.748	0.718	0.686	0.621	0.698
	[0.227]	[0.196]	[0.176]	[0.176]	[0.250]	[0.235]	[0.223]
Maternal age at birth	0.0523	0.0611	0.0794	0.0758	0.0719	0.0643	0.0734
	[0.0246]	[0.0207]	[0.0181]	[0.0180]	[0.0278]	[0.0258]	[0.0244]
Mother African-American	0.381	0.151	-0.325	-0.231	-0.130	0.0690	-0.170
	[0.624]	[0.520]	[0.448]	[0.447]	[0.710]	[0.655]	[0.618]
Mother Hispanic	-0.567	-0.507	-0.383	-0.408	-0.434	-0.486	-0.424
	[0.281]	[0.262]	[0.248]	[0.249]	[0.288]	[0.281]	[0.273]
Mother White	1.694	1.562	1.287	1.341	1.400	1.514	1.377
	[0.382]	[0.326]	[0.287]	[0.287]	[0.428]	[0.398]	[0.378]
Mother Asian	3.415	3.328	3.149	3.184	3.222	3.297	3.207
	[0.358]	[0.329]	[0.308]	[0.309]	[0.374]	[0.361]	[0.349]
Maternal education in years	0.632	0.649	0.686	0.679	0.671	0.656	0.674
	[0.0501]	[0.0423]	[0.0369]	[0.0369]	[0.0565]	[0.0524]	[0.0496]
Mother has at least one risk factor	-0.417	-0.472	-0.585	-0.563	-0.538	-0.491	-0.548
	[0.197]	[0.177]	[0.164]	[0.164]	[0.210]	[0.201]	[0.193]
Always free/reduced lunch	-3.662	-3.804	-4.097	-4.040	-3.977	-3.854	-4.002
	[0.396]	[0.333]	[0.290]	[0.290]	[0.447]	[0.414]	[0.392]
Sometimes free/reduced lunch	-2.715	-2.797	-2.968	-2.934	-2.898	-2.827	-2.912
	[0.248]	[0.215]	[0.191]	[0.191]	[0.275]	[0.257]	[0.245]
male	-3.838	-3.892	-4.004	-3.982	-3.958	-3.911	-3.967
	[0.165]	[0.143]	[0.128]	[0.128]	[0.182]	[0.171]	[0.163]
Birth order	-0.493	-0.558	-0.693	-0.666	-0.637	-0.581	-0.649
	[0.173]	[0.143]	[0.122]	[0.122]	[0.199]	[0.182]	[0.172]
Birth weight/100	0.0983	0.103	0.113	0.111	0.109	0.105	0.109
	[0.0157]	[0.0140]	[0.0128]	[0.0128]	[0.0170]	[0.0161]	[0.0154]
Gestation in Weeks	0.0153	0.00311	-0.0222	-0.0173	-0.0119	-0.00128	-0.0140
	[0.0429]	[0.0385]	[0.0353]	[0.0354]	[0.0460]	[0.0439]	[0.0421]
Month prenatal care initiated	0.0801	0.0500	-0.0125	-0.000222	0.0131	0.0392	0.00785
	[0.0875]	[0.0747]	[0.0659]	[0.0659]	[0.0979]	[0.0911]	[0.0865]
Observations	57,310	57,310	57,310	57,310	57,310	57,310	57,310
Certificates at birth*Original Lead	X	X	X	X			
Certificates squared		X		X			
Certificates birth-age5*Original Lead			X	X			
Certificates at birth/Prewar housing					X	X	X
Certificates at birth/Prewar housing squared						X	X
Certificates birth to age 5/Prewar housing							X

Table 9: Disparities in Lead and Disparities in Test Scores - Tract and County Level

	(1)	(2)	(3)	(4)
	White-Black Reading Scores			
	Tract Level Disparities		County Level Disparities	
	OLS	IV	OLS	IV
White-Black Lead Scores	-0.746	-2.101	-1.422	-1.321
	[0.127]	[1.487]	[0.822]	[2.179]
Observations	1,736	1,695	40	40
R-squared	0.146	0.077	0.853	0.853

Each observation is a tract (county) year. Also included are county (tract) and year of birth fixed effects.

All regressions weighted by tract (county) population

Note that the White-Black lead score difference is negative, generally, since white lead levels are lower than black lead levels

Fig 1: Certificates and 1997 Tract Characteristics

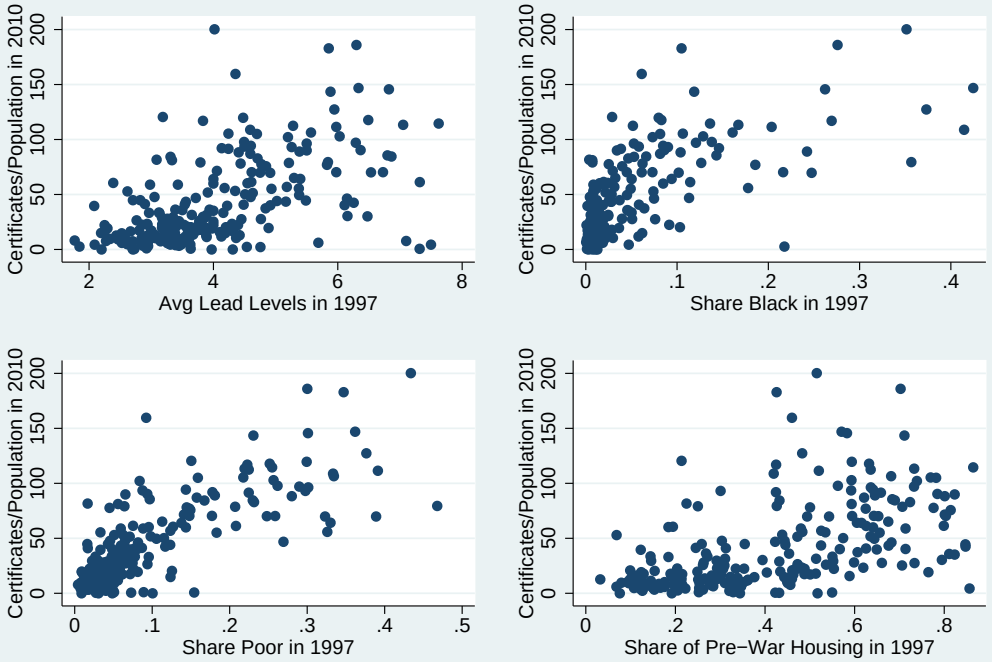
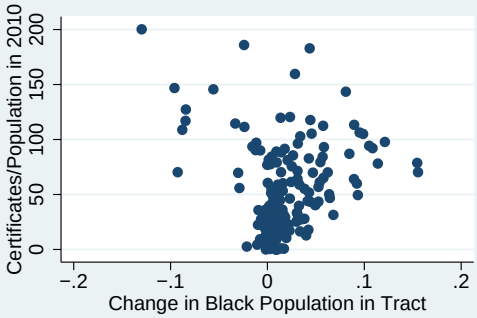
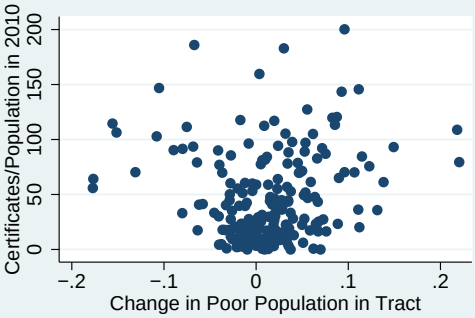
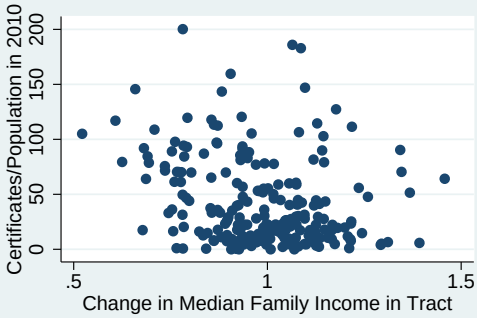


Fig 2: Certificates and Changing Tract Characteristics







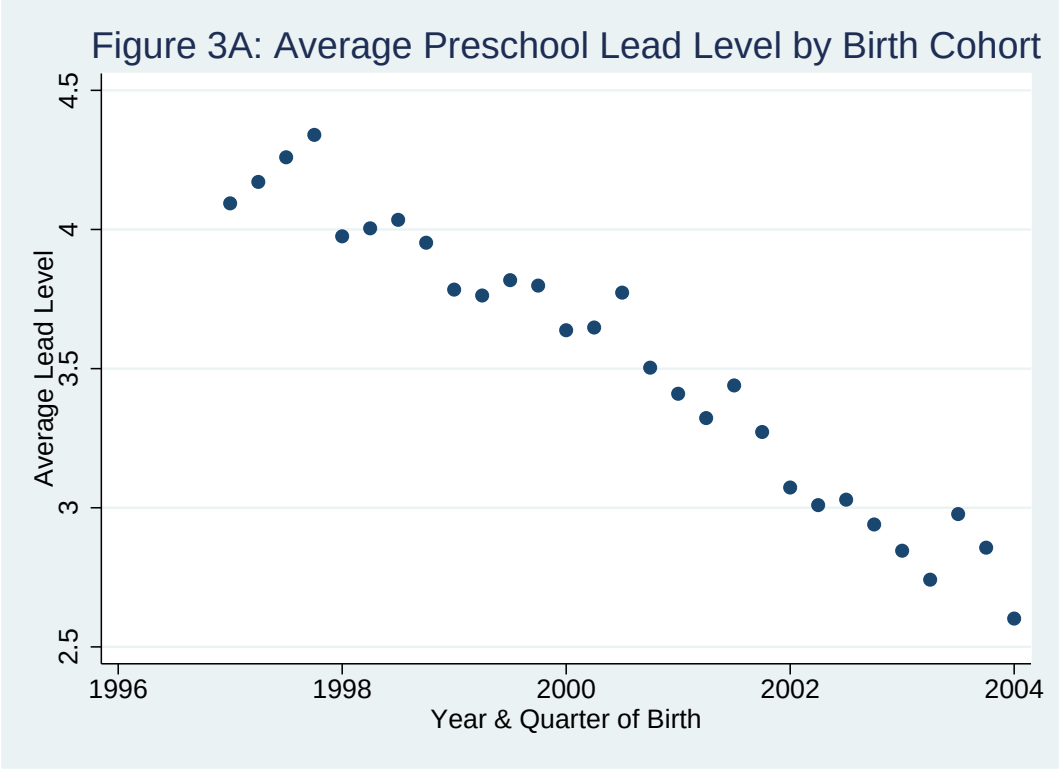


Figure 3A: Average Preschool Lead Level by Birth Cohort
Average Lead Level
2.5
3
3.5
4
4.5
1996
1998
2000
2002
2004
Year & Quarter of Birth

Figure 3B: Trends in Lead by Child Characteristic

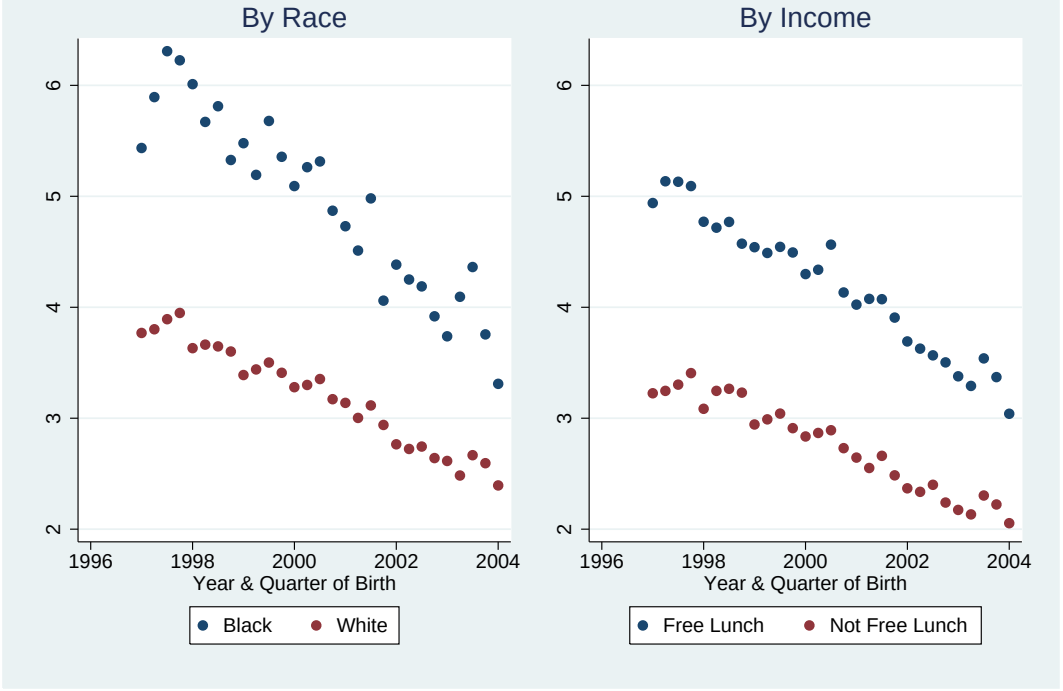


Figure 4: Lead Levels, Test Scores and Maternal Education

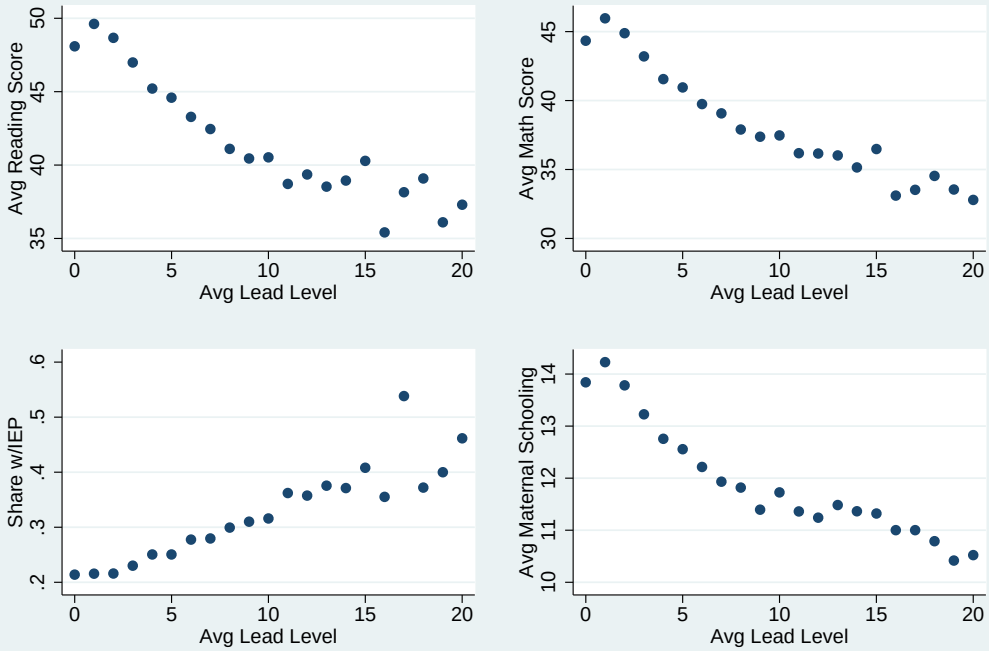


Figure 5A: Reading Scores by Race

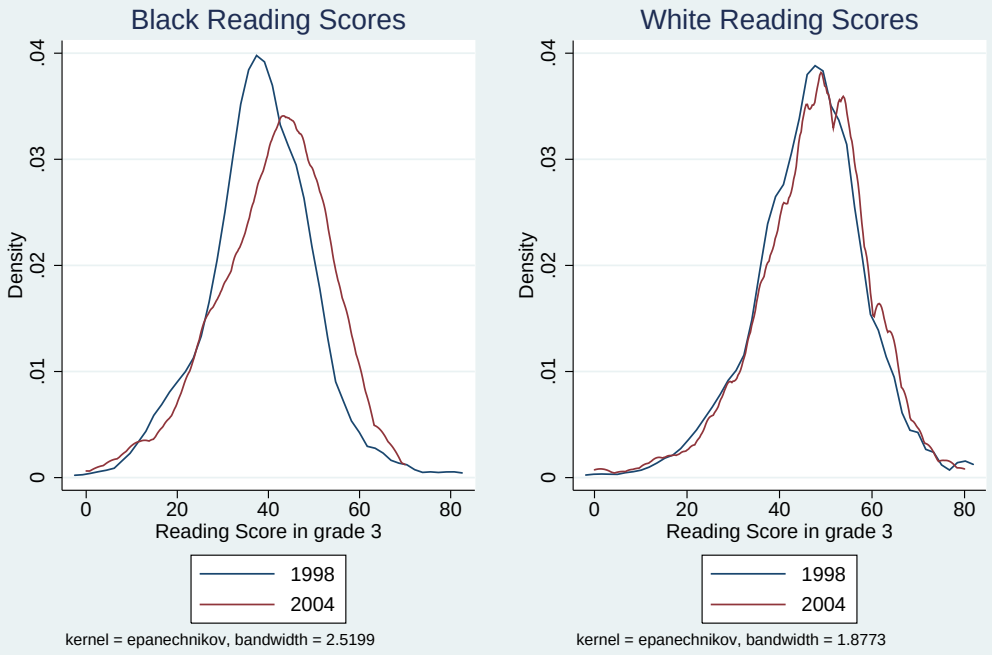


Figure 5B: Reading Scores by Year

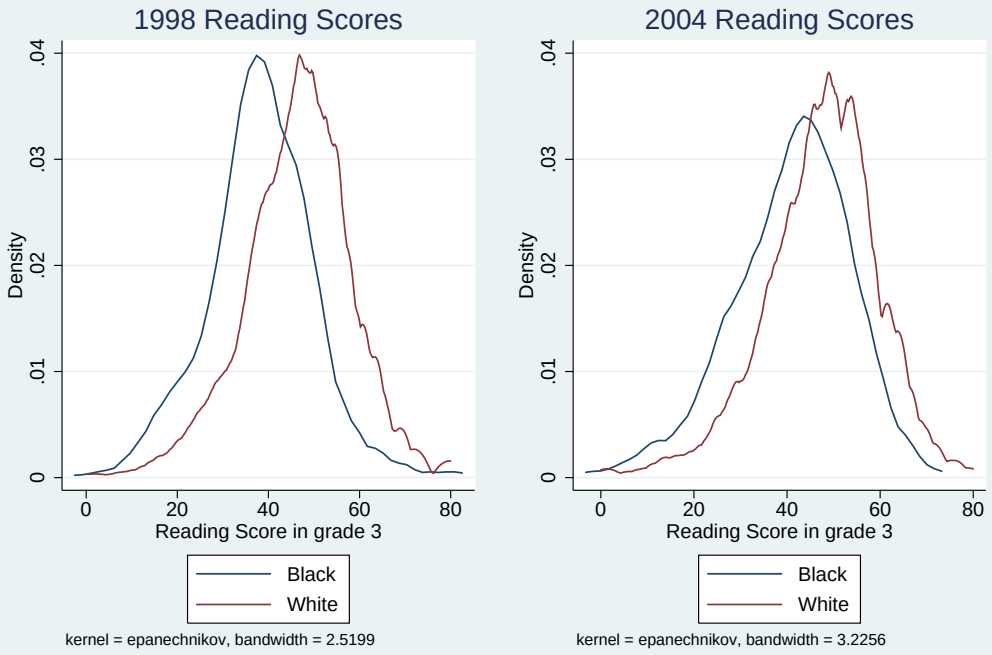
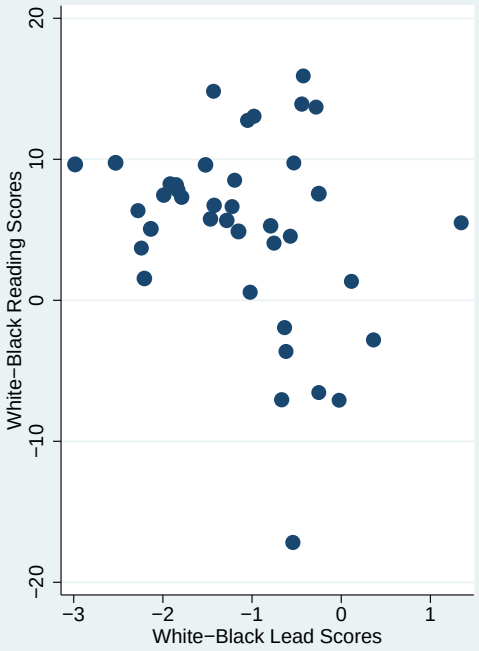
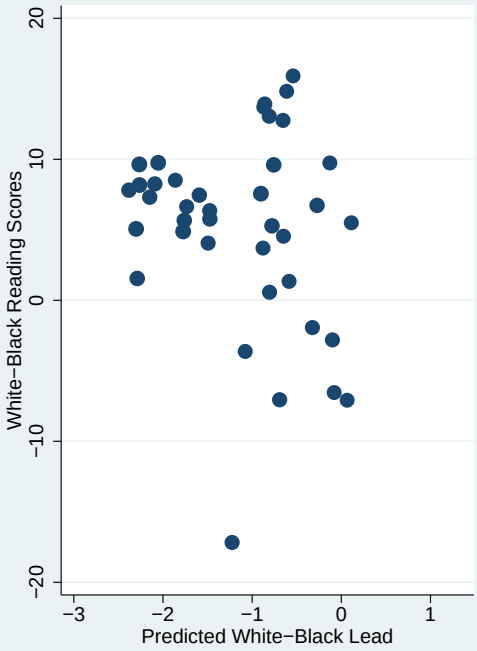
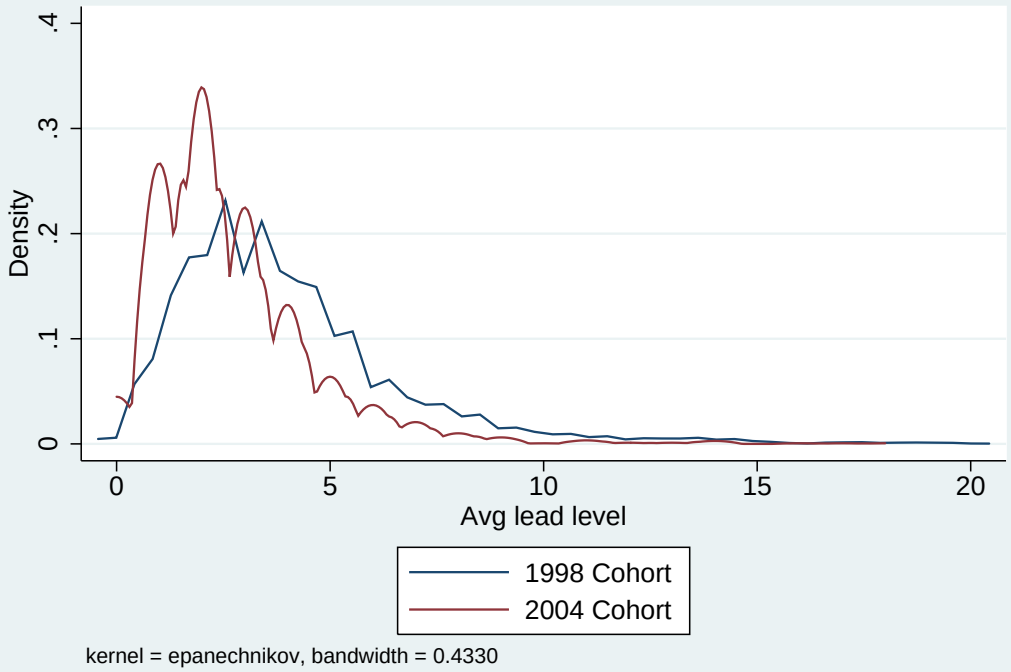


Figure 6: Racial Disparities in Lead & Test Scores





Appendix Figure 1: Average Lead Levels Over Time



Appendix Table 1: Within Tract Correlations Between Lead Levels and Child and Family Characteristics

	Avg lead level
Married at Birth	-0.244
	[0.0214]
Maternal age at birth	-0.0280
	[0.00174]
Mother African-American	0.328
	[0.0325]
Mother Hispanic	-0.554
	[0.0370]
Mother other race/ethnicity	-0.251
	[0.0292]
Mother Asian	-0.0116
	[0.0441]
Maternal education in years	-0.0526
	[0.00371]
Mother has at least one risk factor	0.172
	[0.0223]
Always free/reduced lunch	0.359
	[0.0267]
Sometimes free/reduced lunch	0.253
	[0.0239]
male	0.169
	[0.0160]
Birth order	0.207
	[0.00870]
Birth weight/100	-0.0147
	[0.00180]
Gestation in Weeks	0.0389
	[0.00510]
Month prenatal care initiated	0.0865
	[0.00730]
Observations	53,994
R-squared	0.206