Lead Exposure and the Black-White Test Score Gap

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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 Rhode Island, we document significant declines in racial disparities in child lead levels since 1997, due in part 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 eight in an IV framework. We find that a 5 ug/dl increase in child lead levels (the threshold at which the CDC recommends intervention) reduces test scores by 6 points or 43 percent of a standard deviation. The effects are stronger in the lower tail of the test score distribution and do not appear to fade over time. We calculate that the decline in racial disparities in lead explains roughly half of the decline in racial disparities in test scores witnessed over the past decade in RI.

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I. Introduction

Racial disparities in test scores have declined significantly over the past forty years, though a substantial gap remains. As measured by National Association of Educational Progress (NAEP) scores for 9 year olds, the black-white test score gap represented 1.25 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). 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. 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). Currie (2011) demonstrated that even within zip codes, black mothers were more likely to reside near Superfund sites or plants with toxic emissions.

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.

To examine this, we focus on the environmental toxin lead, for which there is strong neuro-biological and epidemiological evidence of a negative relationship between early exposure and future cognitive and non-cognitive outcomes. The major sources of lead in the environment today are residual lead in soil from past emissions and deteriorating lead-based paint remaining in old homes (Levin et al 2008), both of which are higher in urban areas. Because African American families are residentially segregated in older, urban areas, this results in African American children's greater exposure to lead. Using a unique dataset for the state of Rhode Island 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 and poor children in our sample. Among RI children born in 1997, (the first birth cohort of our data), African-American children had lead levels of 6 ug/dl, on average, compared with 4 for white children, and children on free lunch had lead levels of 5 on average, compared with 3.3 for those not on free lunch.

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 can lead 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 African American, 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 primarily from the imprecision in each blood test, but also from the fact that blood tests capture only recent

exposure that may or may not have been persistent, 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 two policies implemented in RI in 1997 that required landlords to ensure that their rental homes were safe of lead hazards. These policies followed new CDC policies issued in 1997 urging states to target testing and remediation of the highest risk children (Jones et al, 2009). The first state policy required the owners of any building in which a child had an elevated lead level to mitigate any lead hazard or risk prosecution of the state Attorney General. The second state policy applied to all landlords, regardless of whether a child had tested positive for lead, and required them to obtain "lead-safe certificates" in order to rent their properties. For the latter, the state targeted the oldest urban centers of the state. We document that neighborhoods with a greater share of old housing (the primary source of lead paint), and with higher initial 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.[1] These neighborhoods were also predominantly African American and poor. By including neighborhood (census 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.

[1] Previous work has documented that individual blood lead levels decline in a household after a certificate has been issued (Rogers et al, 2014)

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 6 points, or 43 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, particularly for math test scores. The IV estimated effects are larger than the OLS estimates, consistent with considerable classical measurement error in lead scores which we document.[2]

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 1.56 point reduction in the black-white test score gap (12 percent of a standard deviation), thereby explaining half 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 scores among the disadvantaged more generally. When we compare outcomes for children on free lunch or not, we find that both the baseline (1997) levels of lead were lower and more similar and that the declines in disparities in lead levels and test scores were smaller. The greater effects for African American families are likely attributable to the fact that African American families are disproportionately located in urban areas of the state even conditional on poverty (89 percent of

[2] We explore whether African American children in addition to being more likely to be exposed, 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). However, our data and identification do not allow such a breakdown.

poor blacks relative to 60 percent of poor whites) and these areas are characterized by the highest (historical) lead-contaminated emissions, oldest housing and the highest baseline lead levels. These were also the areas targeted by the state in its outreach efforts.

Our results suggest that African American children's disproportionate exposure to environmental pollutants that affect child development can potentially explain a significant 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

Jencks and Phillips (2008) provide a comprehensive review of the existing research on various factors that have been considered to explain racial disparities in test scores and their decline over time. These include family income, family structure, school segregation, cultural influences, test bias, stereotype threat, parenting strategies, the quality of school inputs.[3] 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 the resulting improvements

[3] There is a long literature on this topic which includes Brooks-Gun et al (2003), Card and Rothstein (2007),Cook and Evans (2000), Dee (2005), Figlio (2005), Fryer and Levitt (2004), Hanushek and Rivkin (2006), Krueger and Whitmore (2001), and Reber (2010) among others.

in health explain a significant share of the closing of the black-white gap in cognitive achievement 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.

Debate over the cause of environmental inequalities 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; Currie et al., 2015).[4] 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.

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.

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

B. Lead Exposure in American Children and the National Policy Response

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.[5] 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.[6] 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, but especially for historically high-risk groups of children (Jones et al, 2009).

Using laboratory data from the NHANES, we examine average BLLs by race and birth cohort for children less than seven years old. Over this period, BLLs of all children have declined, but more so for African American children (Appendix Figure 1, first panel). For comparison, we also plot racial disparities in NAEP reading test scores for 9 year olds by birth cohort in the second panel. Just as the racial gap in lead levels has fallen over this period, so too has the gap in test scores, though a substantial gap remains.

Despite the removal of lead from gasoline and household paint in the 1970s and 1980s, lead has remained a significant hazard in the environment, particularly in urban areas where

[5] 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.

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

African American families are disproportionately located (Bajari and Kahn, 2001).[7] This is due to the geographic concentration of three of the main sources of lead in urban areas: residual lead in soil near high traffic areas, lead in deteriorating paint found in old homes, and residual lead in soil at former industrial sites which were often located in central cities (Lanphear et al 1998; Levin et al, 2008). With respect to past emissions, not only are urban homes located closer to major roadways where there are high levels of residual lead found in soil, but conditional on distance to roadway, urban areas are characterized by higher lead levels due to greater (historical) traffic volume (Filipelli et al, 2005).[8] With respect to lead-based paint as another major source of exposure, urban areas are also more likely to be characterized by older housing that contains lead-based paint than non-urban areas. But even conditional on living in an old home, there may be important differences in exposure to lead by socio-economic status since the hazards associated with lead-based paint can be reduced by painting over old paint and ensuring that living areas are free of paint chips and dust. To the extent that more educated or wealthier families are more likely to take these precautions, similar exposures to old housing within an urban area are more likely to negatively affect the most disadvantaged.

While child lead levels have declined dramatically over the past 30 years, the CDC still considers exposure to lead an important public health issue. Current estimates suggest that 4.5 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). Moreover, disparities by race and income, while smaller, still remain, with African American and poor children two to three

[7] In 1990, among households living in metropolitan areas (75% of the US population), a black household was 31 percentage points more likely to live in a metropolitan area's center city than a white household (Bajari and Kahn, 2001), and this is true even conditional on poverty.

[8] High levels of residual lead found in soil near major roadways exhibit an exponential decay, declining by two thirds at a distance of 50 meters (Filippelli et al, 2005).

times as likely to have elevated lead levels, defined as above 5 (CDC, MMWR 2013). [9] Indeed, 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. The CDC has also eliminated use of the term "level of concern" when referring to blood levels and lowered the threshold for which children should receive case-management services from 10 to 5 ug/dl in 2012 based on recent research that has found that even small amounts of lead in a child's system can have deleterious effects (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).

[9] 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).

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 since at least Roman times. 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), McLaine et al (2013), 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.[10]

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

[10] 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.

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 with crime levels 15-20 years later. Examples include Masters et al. (1998), Reyes (2015), Nevin (2000 and 2007), Mielke and Zahran (2012), and Grönqvist, Nilsson and Robling (2014). This work supports a strong relationship between declines in lead exposure early in life and declines in crime later in life, but like all 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 later cognitive and behavioral outcomes, there is still uncertainty regarding the magnitude of the effect as well as its duration. This uncertainty arises from two sources: the 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 as well as proximity to high traffic roadways where residual lead in soil is found in high concentrations, and, as a result, are more likely to have elevated BLLs. While this is a concern we try to address, Bellinger (2008) argues that "Such confounding seems highly unlikely to account completely for the associations, given the wide range of circumstances and settings in which they have been found. Evidence that so-called "subclinical"

exposure to lead not only alters behavior but brain structure as well would make the argument of confounding even less tenable."

The second estimation challenge is measurement error in child lead levels obtained from 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, with greater error characterizing capillary measures. According to the CDC, the "ratio of imprecision to measurement value, particularly at BLLs <10ug/dL, is relatively high" (MMWR, 2002).[11] 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.[12] 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 error, though with ambiguity with respect to any bias that may result.[13] Below we discuss our strategy for addressing these potential sources of bias in estimates of the impact of BLLs on later child cognitive outcomes.

[11] 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)

[12] "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

[13] 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.

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 gained 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.

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.[14] 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.

[14] 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.

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. Educational outcomes come from the RI Department of Education (RIDE) and include NECAP[15] 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 third source of data are birth certificate data for the child. These 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 care. The birth certificate data include a mother identifier so siblings can be identified in the dataset.

The final sample includes roughly 57,000 RI children born between 1997 and 2004 with linked BLL and educational test score data. Details on construction of the sample can be found in Appendix Table 1. Three things are worth noting. First, compared with a national lead screening rate of 25 percent, 80 percent of all children with a RI birth certificate are screened at least once and the screening rate remained constant throughout the study period, suggesting that the types of children screened also likely remained constant. Second, using vital statistics data to compare the maternal characteristics (race, education, and marital status) of children with a lead level (80%) against those without a lead level (20%), we find that those with a lead screen are

[15] 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.

slightly more disadvantaged along most measures with the exception of birth weight which is the same for the two groups. Third, among those with a lead level, those matched with RIDE data are slightly more disadvantaged than those who we were not found in RIDE data (presumably because they attended private school). However, children with matched lead-RIDE data are representative of all children in the RIDE data.

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.[16] To construct a single measure we calculate the arithmetic mean of all samples, but also consider alternative measures: The maximum level, the geometric mean, the median BLL, a sum of all BLLs and an approximation of the "area under the curve" for which we assume a linear trend between two lead levels and calculate the mean over all 72 months.

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 7,084 students and of those, we have 8^{th} grade test scores for 6,756 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 1997-2010 in an instrumental variable (IV) framework.

[16] 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 (43 percent) built prior to WWII, and therefore containing the greatest concentration of leaded paint. 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.[17] This disproportionate exposure is due largely to the residential segregation of the poor, and especially African Americans, in the core urban areas of the state located within the county of Providence. US Census data reveals that 81 percent of the homes in Providence county were built prior to 1978 and 49 percent before WWII (Appendix Table 2). Comparable numbers for the rest of the state are 68 and 27 percent. Moreover, low income and African American families are much more likely to live in Providence than elsewhere: 86 percent of African Americans live in Providence compared with 51 percent of whites, while 77 percent of the poor live in Providence compared with 55 percent of the non-poor. The disproportionate exposure of African American children does not just reflect poverty: Even conditional on poverty, African Americans are more likely to live in Providence (89 percent of poor African Americans compared to 60 percent of poor whites).

[17] 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 two programs to encourage lead hazard mitigation in old homes. The first required all owners of homes in which an elevated lead level was reported to the DOH mitigate such hazards. Once the home was mitigated, an inspection would be performed and the Department of Health would issue a certificate (DOH certificate). Non-compliant landlords were referred to the state Attorney General for presecution. The second was administered by the RI Housing Resources Commission (HRC) and required all landlords to mitigate lead hazards in the homes they rent – regardless of whether an elevated lead level was reported. In particular, the state 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 (HRC certificate) for homes they plan to rent. Section 8 voucher rentals and family day care centers were prioritized. There were no resources dedicated to enforcement and though non-compliance was illegal, landlords are typically not penalized for non-compliance. However, low-cost loans were made available to landlords to help finance mitigation efforts. Moreover, landlords could be 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.

Over the period 1997-2010, the total number of lead-safe certificates issued to landlords increased from 333 to over 41,000 (Appendix Table 3). 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).[18] This growth in the number of lead safe certificates within a neighborhood over time 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, median family income or housing prices over this period than tracts that witnessed no or small changes in certificates per capita (Figure 2). Regression results bear this out: increases in the number of certificates are not associated with significant (statistically or economically) changes in race, maternal education, marital status or income of families who live in the neighborhoods (Appendix Table 4).[19]

Our preferred specification relies only on the certificates issued by the RI DOH as instruments as there is less discretion in these certificates since they are required after an elevated lead level and the state's Attorney General threatening prosecution for non-compliance. We do however also use the RI HRC certificates and the amount of low interest rate loans made

[18] Certificates are scaled by 100

[19] We can examine a limited measure of mobility in the first five years of life directly with our data since the census tract of residence is recorded at each blood draw. Seventy two percent of the sample moves at some point during the first 72 months of life and movers are disproportionately disadvantaged with higher blood lead levels, 4.1 vs. 3.2 (Appendix Table 5). However, they appear to move to geographically proximate and very similar areas in terms of the underlying BLLs of children living there, the share poor in the neighborhood and the number of certificates issued (Appendix Table 5). That is, while there is a lot of mobility, movers seem to move to areas that are close and nearly the same.

to landlords to help with remediation efforts as instruments in extensions shown below and the results are very similar.

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 calculated: the number of certificates issued by the DOH in the past 5 years (recall that these are required if a child tests above a certain threshold), the number of certificates issued by the HRC in the past five years, the total number of certificates (DOH and HRC) that had been issued, and the loan amount. Based on this information, we constructed two measures to account for the fact that tracts differed in their lead burden and thus need for certificates. The first measure scales the number of certificates or the loan amount by the number of housing units in the tract built before 1978 (Certificates/Housing Units Built Pre 1978). The second multiplies 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).[20] 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:

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

[20] The average lead level is actually the leave-out average (ie, removing the focal child from the calculation of the average).

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 such as 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, and the share housing built post 1978. [21] Year of birth and tract fixed effects are also included.

The estimates reveal a strong negative relationship between the number of DOH certificates issued in the five years prior to birth over the number of old housing units and child lead levels (Table 1, column 1). Moreover, the effects are concave in nature (Table 1, column 2). The results are also strong when we interact the number of certificates with the baseline lead level in the tract in 1997 (column 3). We present additional first stage estimates based on the number of certificates issued by the HRC (columns 4-6) and total certificates (columns 7-10). Finally, we include the amount of loans provided for lead hazard mediation and find a negative relationship between the loan amount and average lead levels in the neighborhood (columns 11 - 12). The F statistics on the excluded instruments range from 41 to 133 depending on the specification. We focus on results using the first instrument (column 2) because it is the most arguably exogenous, but also present second stage estimates based on the other instruments for comparison in the robustness section.

To interpret the magnitude of the coefficients, we calculate the relationship between lead certificates and average lead levels in the tract based on the average increase in certificates from 1997 to 2004, and the increase in the lead certificates among the top 10% of tracts, because of the skewed distribution of certificates. An average increase in DOH certificates reduced the

[21] Values for intercensal years from linear interpolations between census years.

average lead levels by 0.36, while a large increase in DOH certificates reduced lead levels by 0.69 on average. For HRC certificates, the equivalent reductions are 0.30 and 0.85 respectively.

IV. Results

A. Lead Levels and Child, Family and Neighborhood Characteristics

In RI, disadvantaged children are characterized by higher lead levels, as they are nationally. Among those 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). 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).[22] But these aggregate measures mask significant heterogeneity in declines over time, which we characterize in the next section.

B. Trends in Lead Levels Over Time

Overall, average lead levels have fallen dramatically since the 1997 birth cohort (Figure 3A). The average blood lead level for those born in 1997 was 4.25, falling to 2.5 for those born

[22] 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.

in 2004.[23] 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 not 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 stratifying by race and then interacting certificates with the following child characteristics: Race, free-lunch status, maternal education and child gender. The results suggest that in addition to certificates being disproportionately issued in neighborhoods with a high share of black families (Figure 1), 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, columns 1-3). Interactions with free lunch (column 5) suggest that poorer children are also disproportionately affected, but interactions with gender and maternal education are smaller and imprecisely estimated (columns 6 and 7).

[23] Changes in the distribution of average lead levels for the 1998 vs. 2004 birth cohort are in Appendix Figure 2.

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 (36% 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. This can be indicative of confounding, but could also potentially be another source of exposure if low-income mothers have higher lead levels that they pass on to their children in-utero (Dort, Hurlbut and Hoyer-Hassen, 2004; Bellinger, 2005).

D. Lead Levels and Future Child Cognitive Achievement – OLS and 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:

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

Where Y_i 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. In the IV regressions, 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 3 through 8). 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 4A, column 1). Adding a set of controls that includes race, maternal education, marital status, free lunch, gender, birth order, birth weight, gestation, and prenatal care reduce the estimated coefficient from -1.029 to -0.283 (column 2), adding neighborhood characteristics from census data (column 3) reduces the coefficient only slightly to -0.264 and the inclusion of tract FE does little to the estimated coefficient (column 4).

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 6 points, or 46% of a standard deviation (Table 4A, column 5). This larger IV estimate is consistent with considerable measurement error in lead levels. We explore the potential attenuation bias inherent in a mis-measured 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 6), the relationship between lead levels and test scores is significantly lower (-0.161) than the estimate based on a lead level constructed from an average of multiple test scores (-0.275). These results are consistent with considerable attenuation bias from mismeasurement of lead levels, which we explore further in a later section.[24]

We consider two additional specifications that include school FE and mother FE. The school FE results (columns 7 and 8) are similar in magnitude to the tract FE. When we include

[24] Though the average is itself a noisy measure, if we consider it to be the true measure and the randomly drawn measure to be the noisy measure, this would suggest a reliability ratio of 0.58 and downward attenuation bias of 42%.

mother FE, we limit our comparison to differences in lead levels across siblings which are considerably smaller than cross-sectional differences in lead levels. Including a mother FE significantly reduces the coefficient estimate to -0.105 (Table 4A, column 9). This estimate is consistent with both omitted variable bias in the estimates that include only tract FE, but also with previous work showing that the inclusion of a maternal FE can exacerbate the attenuation bias from measurement error (Griliches and Hausman, 1986). When we include the maternal FE and instrument for lead levels, the estimate is considerably larger than the maternal FE estimator, -2.3 (Table 4A, column 10). Given the potential for exacerbating measurement error in the estimates and the loss of precision with mother FE, we focus on IV with tract FE in what follows.

We also consider one falsification test: estimating whether childhood lead levels in the first 72 months of life affect birth weight in OLS and IV settings. The results (Table 4B) suggest a negative relationship between childhood BLLs and birth weight in an OLS regression with tract FE and a full set of controls. This is consistent with negative selection into high lead levels. However, once we instrument for child BLLs, the estimated effect is essentially zero and imprecise. We interpret this as supporting evidence of a valid identification strategy based on an instrument that meets the exclusion restriction.

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 39 percent more likely to be substantially deficient in reading and 20 percent more likely to be substantially deficient in math. They are 60 percent less likely to 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 test scores (Table 6 Panel A) as well as reaching proficiency in 3rd and 8th grade (Table 6 Panel B). When we reduce the sample size for this analysis to children with third and eighth grade test scores (birth cohorts 1997 and 1998), the estimates become less precise in the IV setting. But the point estimates show that the negative effects of lead either remain the same or increase slightly over this period.

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 too imprecisely estimated (particularly for the IV estimates) to say definitively whether the results vary by race or school lunch status.

In the next section we consider additional specifications to assess the robustness of our results.

F. Robustness

Alternative Measures of Lead

First we consider alternative measures of lead. To rule out that the possibility that the results are being driven by outliers, we trim observations with lead levels in excess of 25 and the results are unchanged (Table 7 columns 1 and 2). We try to account for the timing of the lead measures in a second alternative measure. To do so 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 over imputed values for all 72 months (we refer to this measure as the area under the curve). These results (columns 3 and 4) are smaller but still large. We also consider the sum of all lead levels which is designed to capture persistence (column 5 and 6). A fourth measure is 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 7 and 8). We also calculate the geometric mean of lead as is sometimes done in this literature, which yields somewhat larger point estimates in both the FE and IV specifications (columns 9 and 10). Finally we consider the median lead level (column 11 and 12) which reduces the influence of outlier values and the results are similar to the average lead level. In sum, regardless of how we measure lead levels, the estimated relationship between lead levels and test scores is unchanged.

Alternative Instruments

We also present IV estimates based on alternative sets of instruments: certificates at birth*initial(1997)lead level (Table 8 column 1), the number of certificates/pre-war housing units (column 2), the number of HRC certificates/old housing (columns 3 and 4), HRC and DOH certificates (columns 5 and 6) and the total number of combined certificates (columns 7 and 8). Finally we include as instruments the number of certificates/all homes and the share of all homes that are old (column 9) as well as the amount of loans offered to landlords for remediation (column 10). Estimates across all specifications range from -1.32 to -1.623 and the precision also varies. Even the smallest point estimate suggests a strong relationship.

Mean Reversion/Convergence

An alternative explanation for our finding that areas with the greatest number of certificates issued witnessed the largest declines in lead is one of mean reversion or convergence. More specifically, since areas (or groups of children) that received the greatest number of certificates were also the areas or groups with the highest lead levels, one would expect that over enough time, the high lead levels would eventually converge to the average lead levels, regardless of the number of certificates. In other words, even in the absence of the certificates, one would expect the higher lead levels of African American and poor children to eventually converge to the lower levels characterizing other children.

To address this possibility, we consider a number of alternative specifications. The first addresses convergence by race by includin race*year FE in the econometric specification to account for different time trends by race (Table 9 column 1-2). To rule out that convergence of high lead levels to low lead level areas is driving the results, we consider another specification includes an interaction between 1997 lead level in the tract and year of birth. The results (Table

9, columns 3-4) show that the inclusion of different (linear) time trends for low vs. high lead areas does not alter the findings.

We also consider limiting our sample to those areas with the highest lead burden in 1997 (defined as the top quartile). The idea underlying this test is to take areas that have initially similar high lead levels and show that lead falls more where there are more certificates. In contrast, mean reversion would suggest that the places with the highest initial lead levels would experience large declines with or without the certificate program. The IV point estimates are actually greater in magnitude but not statistically distinguishable from the full sample (Table 9, columns 5 and 6). Finally, we predict the number of certificates that a tract should receive based on 1997 lead levels as well as demographic characteristics of the tract and then limit our sample to those tracts predicted to receive the greatest number of certificates. Again, the results are very similar (Table 9, columns 7 and 8).

Measurement Error in Lead Levels and Attenuation Bias

We assess measurement error in lead levels by comparing OLS estimates based on more and less noisy measures of underlying lead levels. As noted previously, lead levels from capillary tests are noisier than venous tests. For our subsample of children for which we have both capillary and venous test results (n=10,235), we present two sets of estimates: one based on an average of all capillary measures and another on the average of all venous measures. This exercise is similar to the previous results using the randomly drawn measure that resulted in a significant downward bias on the order of 50 percent (Table 4A column 6, reproduced in Table 10, column 1). We find that the "noisy" capillary measures yield an estimated effect of -0.137 (Table 10, column 2) whereas the less noisy venous measure yields an estimate of -0.328 (Table

10, column 3), nearly three times greater. These results are consistent with significant measurement error in lead levels leading to attenuation of OLS estimates.

Finally, we follow the work of Ashenfelter and Krueger (1994) in which they assess the extent to which measurement error in reports of schooling biases estimates of the returns to schooling using a sample of twins in an IV framework. To do so, they exploit multiple measures of educational attainment: One self-reported and the other reported by the twin. With these two measures, they calculate reliability ratios and use a twin's report of his sibling's educational attainment as an instrument for the self-report. Other similar work includes Chalfin and McCrary (forthcoming) in which the authors use multiple measures of police force size to estimate the impact of policy on crime in an IV framework.

Like Ashenfelter and Krueger (1994) and Chalfin and McCrary (forthcoming), we have multiple measures of our variable of interest – lead exposure in early childhood. Our exercise differs not only because we typically have more than 2 measures for each child, but also because these multiple measures were often taken at different times and therefore the underlying child lead levels may have differed. However, each measure can still be interpreted as an (imperfect) measure of the underlying level of lead exposure during early childhood.

Following these two papers, we present estimates of the impact of child lead exposure on future academic achievement based on a single BLL (not the average) and instrument for that measure with other BLL measures for the same child. More specifically, we limit our sample to those with at least two BLLs (n=45,663), draw a single BLL at random, then instrument for it with either a second randomly drawn BLL or with an average of all other BLLs for that child.

The first finding is that the correlation between two randomly drawn lead levels is roughly 0.463 (the coefficient on the second randomly drawn BLL in the first stage regression). This can be interpreted as the reliability ratio and suggests that nearly 54 percent of the measured variance in a single BLL is error. Interestingly, when we repeat the first stage instrumenting for randomly drawn BLL with an average of all other BLLs, the coefficient increases to 0.65, consistent with the average of multiple measures being a more reliable (less noisy) measure.

As expected, the estimate of the impact of the randomly drawn BLL increases from -0.168 in the tract FE specification to -0.384 in the IV specification that relies on a second randomly drawn BLL as an instrument (Table 10, column 4), representing an increase of 139%, and to -0.395 when the average of the remaining BLLs is used as an instrument (column 5).

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 (1997 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 our causal estimates of the impact of lead on test scores, we calculate that the falling disparity in lead levels would correspond to a 1.56 point decline in the racial gap in test scores, explaining 54 percent 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.[25]

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,

[25] 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.[26] 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 11, columns 1 and 3).[27] When we instrument for that black-white gap in lead levels with the number of certificates (Table 11, columns 2 and 4), the resulting estimates are slightly larger, but imprecise and not statistically significantly different from the OLS estimates.

V. Conclusions

The racial gap in family income has, depending on the measure considered, either held steady or increased over time. [28] 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 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

[26] This generates a dataset of 40 observations – 8 birth cohorts for each of the five counties in RI.
[27] We do not present the first stage estimates, but the instrument does significantly predict reductions in lead levels within tract and county over time.
[28] 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 increased from 1.8 to 1.9 over the period 1983-2010; however, the wealth ratio has increased from 5.3 to 5.7 (Urban Institute, Interactive Race Graphic).

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 (2008) 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, when targeted to those children at greatest risk, is effective in reducing environmental exposure to toxins and that declines in racial disparities in exposure, test scores, and, ultimately, economic outcomes, are likely to follow.

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Table 1: First Stage Regression Estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
DOH Certificates at birth/Old Housing	-10.80	-22.89										
	[1.270]	[3.037]										
DOH Certificates at birth/Old Housing squared		128.3										
		[29.28]										
DOH certificates at birth*1997 tract lead levels/100			-0.165									
			[0.0266]									
DOH certificates at birth*1997 tract lead levels/100 squared			0.00490									
			[0.00201]									
HRC certificates/Old Housing				-12.63	-17.01							
				[1.092]	[2.149]							
HRC certificates/Old Housing squared					36.14							
					[15.27]							
HRC certificates at birth*1997 tract lead levels/100						-0.168						
						[0.0186]						
HRC certificates at birth*1997 tract lead levels/100 squared						0.00366						
						[0.000843]						
Total Certificates at Birth/Old Housing							-7.402	-11.03				
							[0.654]	[1.364]				
Total Certificates at Birth/Old Housing squared								17.73				
								[5.851]				
Total certificates at birth*1997 tract lead levels									-0.0576	-0.103		
									[0.00562]	[0.0115]		
Total certificates at birth*1997 tract lead levels/100 squared										0.00142		
										[0.000317]		
Loan Amount in $/Old Housing											-2.145	-4.915
											[0.269]	[0.607]
Loan Amount in $/Old Housing Squared												4.392
												[0.863]
Married at Birth	-0.239	-0.239	-0.239	-0.238	-0.238	-0.238	-0.238	-0.238	-0.238	-0.238	-0.229	-0.228
	[0.0264]	[0.0264]	[0.0264]	[0.0264]	[0.0264]	[0.0264]	[0.0264]	[0.0264]	[0.0264]	[0.0264]	[0.0270]	[0.0270]
Maternal age at birth	-0.0287	-0.0287	-0.0287	-0.0288	-0.0288	-0.0288	-0.0288	-0.0288	-0.0287	-0.0288	-0.0284	-0.0286
	[0.00214]	[0.00214]	[0.00214]	[0.00213]	[0.00213]	[0.00213]	[0.00213]	[0.00213]	[0.00213]	[0.00213]	[0.00218]	[0.00218]
Mother African-American	0.712	0.712	0.715	0.720	0.721	0.724	0.718	0.718	0.720	0.721	0.722	0.723
	[0.0453]	[0.0453]	[0.0453]	[0.0453]	[0.0453]	[0.0453]	[0.0453]	[0.0453]	[0.0453]	[0.0453]	[0.0465]	[0.0465]
Mother Hispanic	-0.253	-0.247	-0.247	-0.248	-0.246	-0.242	-0.249	-0.245	-0.249	-0.241	-0.258	-0.253
	[0.0481]	[0.0481]	[0.0481]	[0.0481]	[0.0481]	[0.0481]	[0.0481]	[0.0481]	[0.0481]	[0.0481]	[0.0489]	[0.0489]
Mother White	0.361	0.362	0.362	0.369	0.369	0.367	0.368	0.368	0.365	0.366	0.350	0.350
	[0.0352]	[0.0352]	[0.0352]	[0.0352]	[0.0352]	[0.0352]	[0.0352]	[0.0352]	[0.0352]	[0.0352]	[0.0360]	[0.0360]
Maternal education in years	-0.0587	-0.0585	-0.0587	-0.0585	-0.0585	-0.0586	-0.0585	-0.0584	-0.0587	-0.0586	-0.0564	-0.0562
	[0.00450]	[0.00450]	[0.00450]	[0.00450]	[0.00450]	[0.00450]	[0.00450]	[0.00450]	[0.00450]	[0.00450]	[0.00458]	[0.00458]
Mother has at least one risk factor	0.177	0.177	0.176	0.171	0.171	0.172	0.174	0.174	0.173	0.174	0.165	0.165
	[0.0293]	[0.0293]	[0.0293]	[0.0293]	[0.0293]	[0.0293]	[0.0293]	[0.0293]	[0.0293]	[0.0293]	[0.0299]	[0.0299]
Always free/reduced lunch	0.455	0.457	0.455	0.458	0.459	0.458	0.458	0.459	0.455	0.458	0.475	0.477
	[0.0344]	[0.0344]	[0.0344]	[0.0344]	[0.0344]	[0.0344]	[0.0344]	[0.0344]	[0.0344]	[0.0344]	[0.0354]	[0.0354]
Sometimes free/reduced lunch	0.266	0.266	0.266	0.267	0.267	0.266	0.266	0.266	0.266	0.266	0.262	0.262
	[0.0281]	[0.0281]	[0.0281]	[0.0281]	[0.0281]	[0.0281]	[0.0281]	[0.0281]	[0.0281]	[0.0281]	[0.0288]	[0.0288]
Male	0.178	0.177	0.177	0.178	0.178	0.178	0.178	0.178	0.178	0.177	0.167	0.168
	[0.0194]	[0.0194]	[0.0194]	[0.0194]	[0.0194]	[0.0194]	[0.0194]	[0.0194]	[0.0194]	[0.0194]	[0.0198]	[0.0198]
Birth order	0.213	0.213	0.213	0.213	0.213	0.213	0.214	0.214	0.213	0.213	0.211	0.211
	[0.0107]	[0.0107]	[0.0107]	[0.0107]	[0.0107]	[0.0107]	[0.0107]	[0.0107]	[0.0107]	[0.0107]	[0.0110]	[0.0110]
Birth weight/100	-0.0156	-0.0156	-0.0155	-0.0156	-0.0155	-0.0155	-0.0156	-0.0156	-0.0156	-0.0155	-0.0153	-0.0153
	[0.00219]	[0.00219]	[0.00219]	[0.00219]	[0.00219]	[0.00219]	[0.00219]	[0.00219]	[0.00219]	[0.00219]	[0.00223]	[0.00223]
Gestation in Weeks	0.0407	0.0406	0.0406	0.0408	0.0407	0.0405	0.0408	0.0407	0.0407	0.0406	0.0407	0.0406
	[0.00622]	[0.00622]	[0.00622]	[0.00622]	[0.00622]	[0.00622]	[0.00622]	[0.00622]	[0.00622]	[0.00622]	[0.00635]	[0.00635]
Month prenatal care initiated	0.100	0.100	0.100	0.0999	0.0996	0.0999	0.0999	0.0995	0.101	0.0999	0.0985	0.0985
	[0.00889]	[0.00889]	[0.00889]	[0.00889]	[0.00889]	[0.00889]	[0.00889]	[0.00889]	[0.00889]	[0.00889]	[0.00911]	[0.00911]
Share of tract below poverty	0.779	1.079	0.419	0.221	0.599	-0.102	0.542	1.131	-0.478	0.303	1.182	0.699
	[1.246]	[1.247]	[1.256]	[1.244]	[1.255]	[1.258]	[1.244]	[1.259]	[1.248]	[1.260]	[1.270]	[1.273]
Median family income in tract ($1000)	0.0157	0.0127	0.0137	0.0102	0.00889	0.00811	0.0111	0.00912	0.0120	0.00891	0.0176	0.0130
	[0.00697]	[0.00700]	[0.00699]	[0.00700]	[0.00702]	[0.00703]	[0.00699]	[0.00702]	[0.00699]	[0.00703]	[0.00706]	[0.00711]
Share housing in tract built post 1979	3.865	3.815	3.437	2.964	2.940	2.560	3.173	3.141	2.810	2.756	3.719	3.259
	[0.838]	[0.838]	[0.846]	[0.845]	[0.845]	[0.853]	[0.843]	[0.843]	[0.851]	[0.851]	[0.853]	[0.857]
Observations	57,310	57,310	57,310	57,310	57,310	57,310	57,310	57,310	57,310	57,310	53,955	53,955
R-squared	0.204	0.204	0.204	0.205	0.205	0.205	0.205	0.205	0.204	0.205	0.205	0.206
F-test	72.39	45.80	40.81	133.7	69.66	64.78	128	68.59	105.2	62.64	63.51	44.73
Effect Sizes:												
Change in lead (2004-1997): tracts with average # of certific	-0.205	-0.358			-0.297							
Change in lead (2004-1997): tracts with top 10% of certifica	-0.410	-0.690			-0.850							

Notes: All regressions include fixed effects for year of birth and census tract.

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 (wh	4.15	4.18	43	43
Low Share Black Neighborhood (wh	2.94	3.08	48	47
Difference	1.21	1.1	-5	-4

Sample includes full sample of children between 1997 and 2004 in RI with valid lead levels and valid reading test scores in grade 3.

Table 3: Alternative First Stage Estimate of Certificates and Lead Levels: Heterogeneity by Child Characteristic

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Black	Non Black	White	All			
DOH Certificates at birth/Old Housing	-30.15	-19.88	-20.97	-9.787	-5.294	-14.65	-10.29
	[10.69]	[3.221]	[4.625]	[1.317]	[2.303]	[3.449]	[1.479]
Certificates*black				-5.841			
				[2.025]			
Certificates*free lunch					-6.551		
					[2.285]		
Certificates*maternal education						0.331	
						[0.276]	
Certificates*male							-0.994
							[1.457]
Observations	4,279	53,031	42,764	57,310	57,310	57,310	57,310
R-squared	0.213	0.189	0.181	0.204	0.204	0.204	0.204
F-test	8.711	27.04	12.43	40.36	40.31	18.05	36.43

Notes: All regressions include all variables listed in Table 1, and fixed effects for year of birth and census tract.

Table 4A: OLS and IV Estimates of Lead Levels and Reading Test Scores

	(1) OLS	(2) OLS	(3) OLS	(4) OLS	(5) IV	(6) OLS	(7) OLS	(8) IV	(9) OLS	(10) IV
Fixed Effects:	None	None	None	Tract	Tract	Tract	School	School	Mother	Mother
Avg lead level	-1.029	-0.283	-0.264	-0.275	-1.190		-0.241	-1.320	-0.105	-2.320
	[0.0188]	[0.0186]	[0.0187]	[0.0190]	[0.483]		[0.0191]	[0.749]	[0.0412]	[0.956]
Randomly Drawn Lead Level						-0.16				
						[0.0142]				
Married at Birth		1.055	0.934	0.913	0.692	0.962	0.834	0.591	-0.697	-0.535
		[0.119]	[0.120]	[0.120]	[0.169]	[0.120]	[0.121]	[0.210]	[0.486]	[0.547]
Maternal age at birth		0.116	0.101	0.0965	0.0704	0.102	0.0942	0.0649	0.243	0.101
		[0.00964]	[0.00967]	[0.00969]	[0.0169]	[0.00970]	[0.00977]	[0.0227]	[0.280]	[0.319]
Mother African-American		-1.142	-1.138	-1.103	-0.454	-1.246	-1.087	-0.389	0.262	0.0278
		[0.203]	[0.203]	[0.206]	[0.402]	[0.206]	[0.208]	[0.530]	[0.808]	[0.907]
Mother Hispanic		-0.976	-0.747	-0.873	-1.108	-0.817	-0.603	-0.856	-1.910	-1.573
		[0.215]	[0.216]	[0.218]	[0.255]	[0.218]	[0.221]	[0.287]	[0.599]	[0.683]
Mother White		0.906	0.274	0.321	0.644	0.256	-0.0530	0.306	0.184	0.134
		[0.145]	[0.154]	[0.160]	[0.236]	[0.160]	[0.163]	[0.301]	[0.520]	[0.580]
Maternal education in years		0.767	0.725	0.710	0.656	0.722	0.673	0.613	0.158	0.0580
		[0.0201]	[0.0203]	[0.0204]	[0.0353]	[0.0204]	[0.0207]	[0.0467]	[0.0695]	[0.0888]
Mother has at least one risk factor		-0.737	-0.722	-0.717	-0.555	-0.749	-0.657	-0.484	-0.243	0.000697
		[0.133]	[0.133]	[0.133]	[0.160]	[0.133]	[0.134]	[0.182]	[0.310]	[0.362]
Always free/reduced lunch		-5.000	-4.504	-4.442	-4.030	-4.537	-4.060	-3.651	2.175	2.203
		[0.151]	[0.155]	[0.156]	[0.270]	[0.156]	[0.163]	[0.330]	[0.825]	[0.921]
Sometimes free/reduced lunch		-3.442	-3.176	-3.123	-2.879	-3.179	-2.888	-2.624	0.942	1.285
		[0.125]	[0.127]	[0.128]	[0.183]	[0.128]	[0.131]	[0.228]	[0.768]	[0.870]
Male		-4.098	-4.100	-4.090	-3.927	-4.131	-4.043	-3.860	-3.924	-3.706
		[0.0883]	[0.0881]	[0.0880]	[0.124]	[0.0881]	[0.0887]	[0.157]	[0.158]	[0.200]
Birth order		-0.843	-0.818	-0.809	-0.614	-0.849	-0.775	-0.554	-0.729	-0.778
		[0.0488]	[0.0488]	[0.0488]	[0.114]	[0.0488]	[0.0492]	[0.161]	[0.207]	[0.232]
Birth weight/100		0.119	0.118	0.117	0.103	0.120	0.116	0.100	0.0960	0.0737
		[0.00994]	[0.00992]	[0.00991]	[0.0126]	[0.00992]	[0.00998]	[0.0149]	[0.0237]	[0.0282]
Gestation in Weeks		-0.0402	-0.0404	-0.0390	-0.00201	-0.0449	-0.0433	-0.00169	-0.0655	-0.0296
		[0.0283]	[0.0282]	[0.0282]	[0.0348]	[0.0283]	[0.0284]	[0.0411]	[0.0629]	[0.0719]
Month prenatal care initiated		-0.0268	-0.0256	-0.0448	0.0474	-0.0646	-0.0166	0.0925	-0.0415	0.0483
		[0.0403]	[0.0403]	[0.0403]	[0.0637]	[0.0404]	[0.0407]	[0.0865]	[0.0856]	[0.103]
Share of tract below poverty			-1.659	15.24	15.65	15.09	0.950	1.423	1.689	2.989
			[0.769]	[5.640]	[5.758]	[5.649]	[1.015]	[1.095]	[2.388]	[2.722]
Median family income in tract ($10			0.0371	-0.0288	-0.00988	-0.0338	0.0346	0.0356	0.00367	0.00689
			[0.00371]	[0.0315]	[0.0336]	[0.0315]	[0.00469]	[0.00487]	[0.0126]	[0.0141]
Share housing in tract built post 19			-2.350	3.746	7.836	2.781	-0.584	-0.0138	-2.099	-3.539
			[0.371]	[3.785]	[4.424]	[3.791]	[0.636]	[0.765]	[1.284]	[1.561]
Observations	60,582	57,310	57,310	57,310	57,310	57,310	55,173	55,173	27,962	27,962
R-squared	0.048	0.243	0.247	0.138		0.135	0.127		0.062	
Number of tracts				239	239	239				
Number of mothers									16,127	16,127
Number of schools							225	225		

Note: for school FE regressions, additional tract*year controls from Census data included: share black, ln(population), number of housing units, share pre-war units

Table 4B: Falsification Test - Impact of Lead on Birth Weight

Dependent Variable= Birth Weight (grams)/100	(1) OLS	(2) IV
Average Lead Level	-0.0571	0.00594
	[0.00833]	[0.205]
Observations	53,955	53,955
R-squared	0.436	

Notes: These regressions include all of the variables included in Table 4A, column 4, census tract and year of birth fixed effects.

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	OLS	IV	OLS	IV	OLS	IV
Avg lead level	0.00547	0.0783	-0.00863	-0.121	-0.00275	0.0269
	[0.000537]	[0.0153]	[0.000794]	[0.0229]	[0.000728]	[0.0182]
Observations	53,978	53,978	53,978	53,978	53,978	53,978
R-squared	0.033		0.067		0.058	
Mean of Dependent Variable	21%		71%		10%	

	(1)	(2)	(3)	(4)	(5)	(6)
	Substantially below Proficient <30		Proficient >=40		with Distinction >56	
Panel B: Math Proficiency	OLS	IV	OLS	IV	OLS	IV
Avg lead level	0.00471	0.0409	-0.00471	0.000181	-0.00175	0.0206
	[0.000618]	[0.0156]	[0.000845]	[0.0207]	[0.000592]	[0.0147]
Observations	53,967	53,967	53,967	53,967	53,967	53,967
R-squared	0.031		0.065		0.033	
Mean of Dependent Variable	12%		60%		14%	

Notes: These regressions include all of the variables included in Table 4A, column 4, census tract and year of birth FE.

Table 6: Lead Levels and Test Scores Over Time

	(1)	(2)	(3)	(4)
	Score in Grade 3		Score in Grade 8	
Panel A: Continuous Reading Score	OLS	IV	OLS	IV
Avg lead level	-0.276	-1.066	-0.257	-2.286
	[0.0345]	[1.304]	[0.0348]	[1.510]
Observations	16,488	16,488	17,825	17,825
R-squared	0.103		0.143	

	(1)	(2)	(3)	(4)
	Proficient in Grade 3		Proficient in Grade 8	
Panel B: Proficient Reading Score	OLS	IV	OLS	IV
Avg lead level	-0.00800	-0.0882	-0.00776	-0.135
	[0.00136]	[0.0557]	[0.00112]	[0.0586]
Observations	16,488	16,488	17,825	17,825
R-squared	0.068		0.068	
Number of tracts	238	238	238	238

Note: These regressions included all of the variables in Table 4A column 4, census tract and year of birth FE.

Table 7: Alternative Measures of Child Lead Levels and Reading Test Scores

	(1) OLS	(2) IV	(3) OLS	(4) IV	(5) OLS	(6) IV	(7) OLS	(8) IV	(9) OLS	(10) IV	(11) OLS	(12) IV
Average lead, truncate at 25	-0.279	-1.197										
	[0.0192]	[0.486]										
Area under the curve			-0.310	-1.246								
			[0.0206]	[0.511]								
Sum of lead tests					-0.0266	-0.136						
					[0.00226]	[0.0549]						
Max lead level							-0.140	-0.793				
							[0.0107]	[0.327]				
Geometric Mean of Lead									-0.310	-1.907		
									[0.0207]	[0.926]		
Median lead level											-0.268	-1.522
											[0.0187]	[0.382]
Observations	57,310	57,310	57,297	57,297	57,310	57,310	57,310	57,310	56,278	56,278	57,310	57,310
R-squared	0.138		0.138		0.137		0.138		0.140		0.138	
Number of tracts	239	239	239	239	239	239	239	239	239	239	239	239

Note: These regressions included all of the variables in Table 4A column 4, census tract and year of birth FE.

Table 8: Alternative IV Estimates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Avg lead level	-1.411	-1.453	-1.490	-1.408	-1.320	-1.492	-1.595	-1.539	-1.623	-1.622
	[0.517]	[0.479]	[0.412]	[0.457]	[0.422]	[0.408]	[0.462]	[0.386]	[0.469]	[0.498]
Observations	57,310	57,310	57,310	57,303	57,303	57,310	57,303	57,310	57,310	53,955
DOH Certificates at Birth*1997 Lead Level of Trac	X									
DOH Certificates/Pre-war		X								
HRC Certificates at Birth*1997 Lead Level of Trac			X							
HRC Certificates at Birth/Pre-War				X						
DOH Certificates at Birth/Pre-War					X					
HRC Certificates Birth/Pre-War					X					
DOH Certificates at Birth*1997 Lead Level						X				
HRC Certificates Birth*1997 Lead Level						X				
Total Certificates at Birth*1997 Lead Level of Tra							X			
Total Certificates at Birth/Pre-War								X		
DOH Certificates at Birth/Pre-war									X	
Share Homes Pre-War									X	
Share Homes 1945-1979									X	
Loan Amount $/Old Homes										X
Loan Amount $/Old Homes Squared										X

Note: These regressions included all of the variables in Table 4A column 4, census tract and year of birth FE.

Table 9: Extensions Addressing Convergence in Lead Levels

	(1) OLS	(2) IV	(3) OLS	(4) IV	(5) OLS	(6) IV	(7) OLS	(8) IV
Avg lead level	-0.273	-1.073	-0.278	-1.238	-0.302	-2.214	-0.318	-1.627
	[0.0190]	[0.539]	[0.0193]	[0.506]	[0.0310]	[1.039]	[0.0272]	[0.895]
Observations	57,310	57,310	57,310	57,310	14,382	14,382	20,127	20,127
R-squared	0.138		0.138		0.123		0.123	
Black*year FE	X	X						
1997 Lead Levels*Year			X	X				
Sample=highest lead levels in 1997					X	X		
Sample=highest predicted # certificates							X	X

Note: These regressions included all of the variables in Table 4A column 4, census tract and year of birth FE.

Table 10: Measurement Error in Child Lead Levels

	(1)	(2)	(3)	(4)	(5)
Outcome=Reading Score	OLS	OLS	OLS	IV	IV
Randomly drawn lead level	-0.161			-0.384	-0.395
	[0.0151]			[0.0338]	[0.0296]
Average over all capillary tests		-0.137			
		[0.0294]			
Average over all venous tests			-0.328		
			[0.0378]		
Observations	45,663	10,235	10,235	45,663	45,663
R-squared	0.139	0.145	0.149		
IV=Avg of All Other Lead Levels					X
IV=Other (Different) Random Lead Le				X	
Outcome=Random Lead Level					
Randomly Drawn lead level				0.463	
				[0.00432]	
Avg of All other Lead Tests					0.653
					[0.00514]
Observations				45,663	45,663
R-squared				0.254	0.310
F-test				11478	16136

Note: These regressions included all of the variables in Table 4A column 4, census tract and year of birth FE.

Table 11: Racial Disparities in Lead and Test Scores, County and Tract Level

	(1)	(2)	(3)	(4)
	White-Black Reading Scores			
	Tract Level Disparities		County Level Disparities	
	OLS	IV	OLS	IV
White-Black Lead Levels	-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

Notes: 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

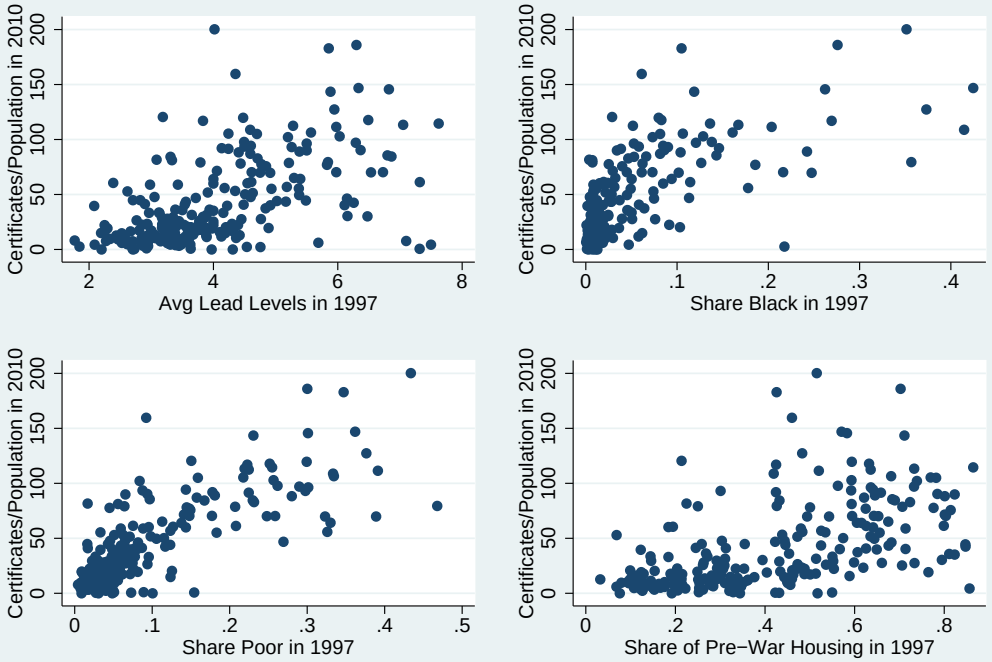
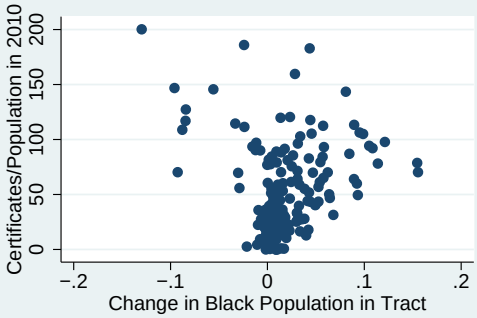
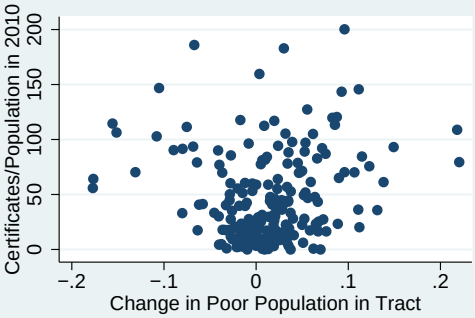
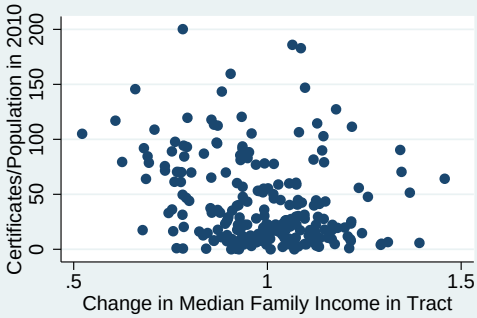


Fig 2: Certificates and Changing Tract Characteristics







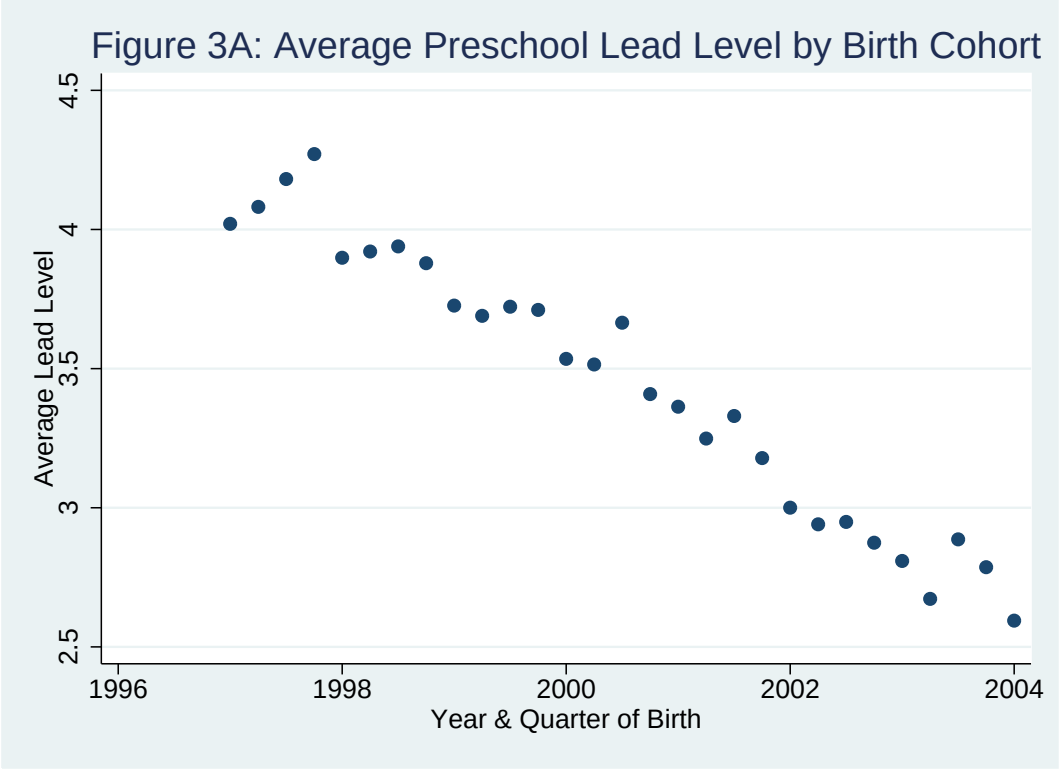


Figure 3A: Average Preschool Lead Level by Birth Cohort
Average Lead Level
2.5
3
3.5
4
4.5
1996
1998
2000
2002
2004
Year & Quarter of Birth

Figure 3B: Trends in Lead by Child Characteristic

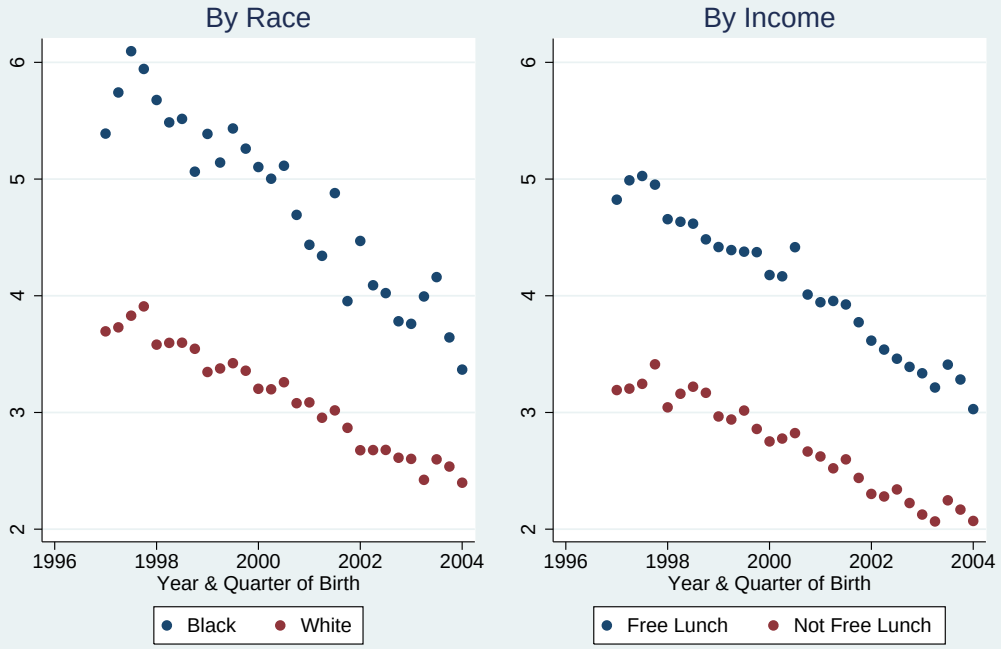


Figure 4: Lead Levels, Test Scores and Maternal Education

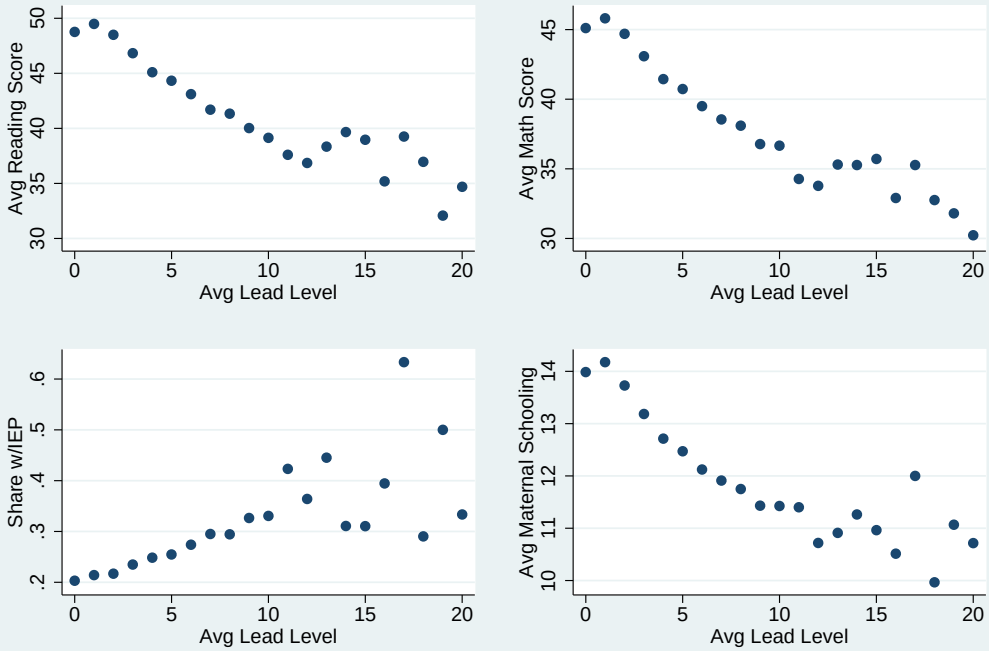


Figure 5A: Reading Scores by Race

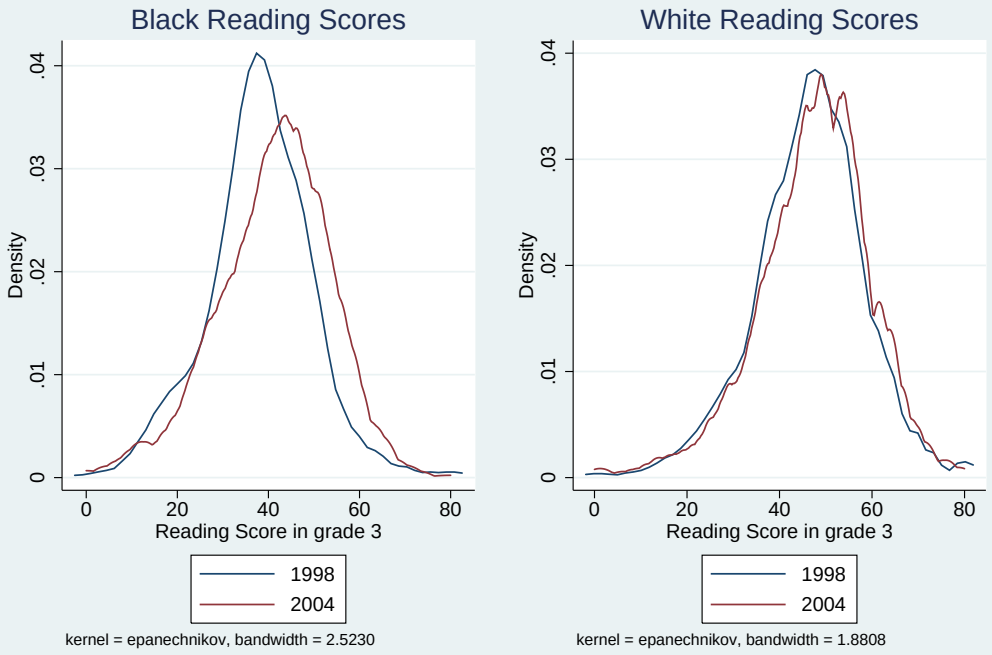
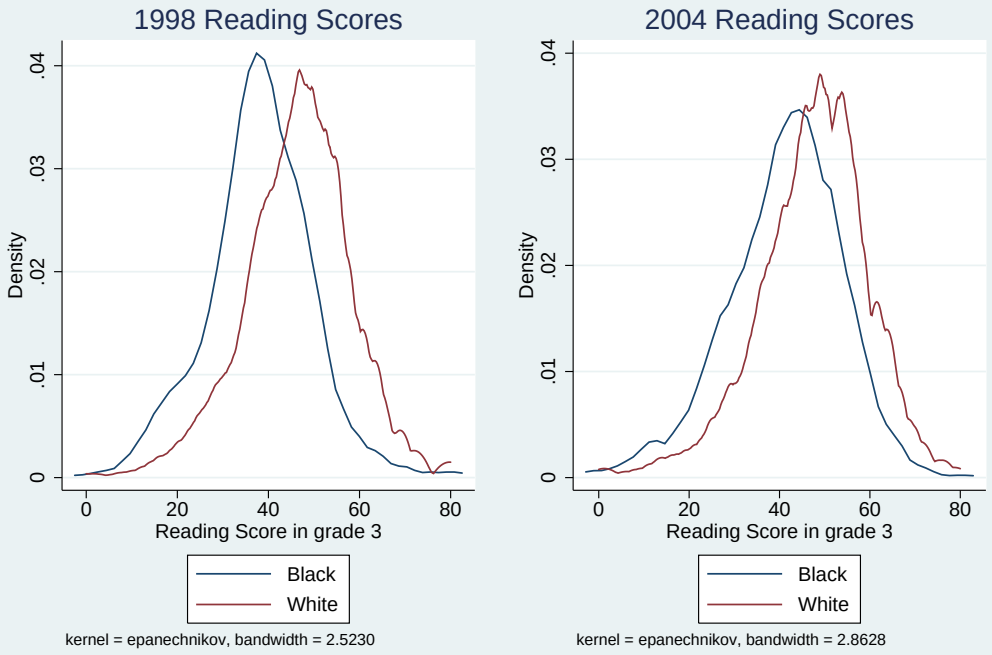


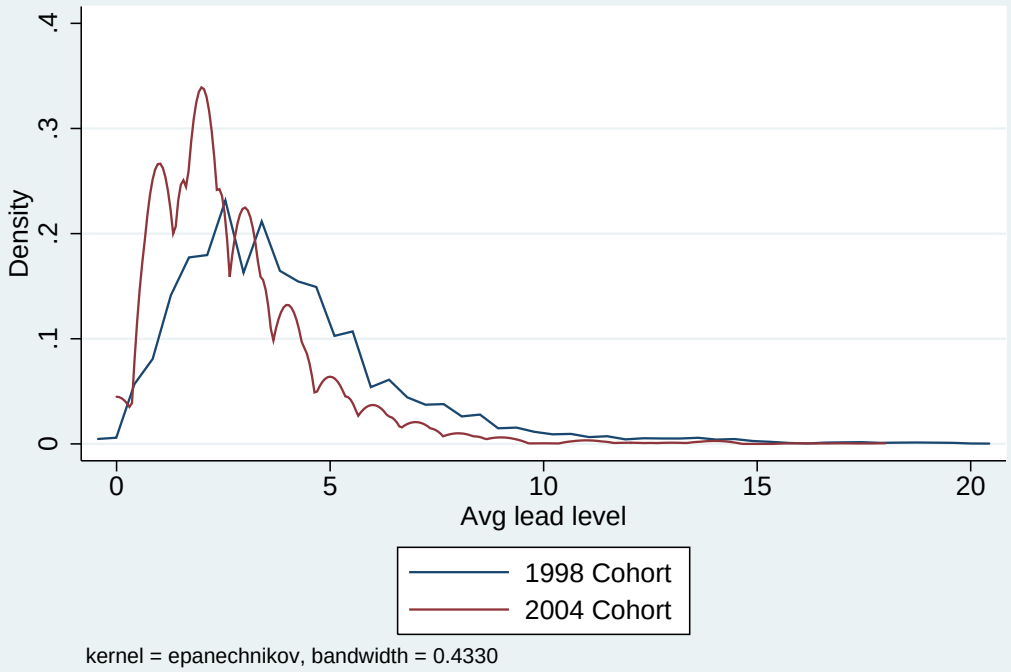
Figure 5B: Reading Scores by Year



App Figure 1: National Black−White Differences in Lead and Test Scores by Birth Cohort

Note: Lead data come from combined NHANES data and test scores from 9 Year old NAEP English tests

Appendix Figure 2: Average Lead Levels Over Time



Appendix Table 1: Sample Construction

	VS Data (n=104,467)		VS-Lead Matched data (n=84,501)		
	No Lead (n=19.966)	Lead (n=84,501)	No RIDE (n=14,776)	RIDE (n=69,725)	Overall RIDE (n=75,586)
Birth weight	3238	3344	3368	3339	3335
Black	0.063	0.079	0.075	0.08	0.078
Maternal educatior	14.1	13.4	14.2	13.2	13.1
Married at birth	0.74	0.62	0.73	0.61	0.6
Maternal age at bir	29.8	28.8	30	28.5	28.5
Birth Order	1.94	1.9	1.9	1.9	1.9
Lead - avg		3.4	3.2	3.4	
Third Grade Reading Scores				45.6	45.5
Free lunch (third grade)				0.44	0.47

Appendix Table 2: Age of Housing and Child Characteristics

Concentration of Children by Race and Income Living in Old Housing in the US and RI

		US Share in Housing Built		RI Share in Housing Built	
		Pre 1978	Pre 1945	Pre 1978	Pre 1945
Black		0.72	0.22	0.83	0.52
White		0.63	0.22	0.74	0.37
Hispanic		0.68	0.2	0.83	0.53
<=100% FPL				0.81	0.43
>=200% FPL				0.76	0.4
<200% FPL and Black				0.83	0.5
<200% FPL and White				0.84	0.48
Black					
	2010	0.58	0.18	0.82	0.58
	2000	0.7	0.21	0.85	0.47
	1990	0.81	0.26	0.82	0.52
White					
	2010	0.5	0.18	0.62	0.41
	2000	0.59	0.21	0.71	0.34
	1990	0.73	0.25	0.8	0.32

Concentration of Children by Race and Income Living in Providence

	Providence	Rest of State
Share housing built pre 1978	0.81	0.68
Share housing built pre 1945	0.49	0.27
Share black population living in:	0.86	0.14
Share white population living in:	0.51	0.49
Share population <100% FPL living in:	0.77	0.23
Share population >100% FPL lining in:	0.55	0.45
Share population <100% FPL & black livi	0.89	0.11
Share population <100% FPL & white livi	0.6	0.4

Calculations from US census IPUMS data for 1990, 2000 and 2010

Sample includes all children <=10 years old

Appendix Table 3: Certificates Issued in RI

Certificates Granted by end of 2010 by County

Panel A: Total Cumulative Certifi	Population	Old Housing	Certificates			rtificates per 1000 Populati			rtificates per Old Housing U		
			Total	DOH	HRC	Total	DOH	HRC	Total	DOH	HRC
Bristol	49,875	8,602	634	0	634	12.71	-	12.71	0.07	-	0.07
Kent	166,155	22,249	4336	3122	1214	26.10	18.79	7.31	0.19	0.14	0.05
Newport	75,400	13,514	3204	707	2497	42.49	9.38	33.12	0.24	0.05	0.18
Providence	626,667	128,225	37059	11452	25607	59.14	18.27	40.86	0.29	0.09	0.20
Washington	118,269	13,265	1501	349	1152	12.69	2.95	9.74	0.11	0.03	0.09

Panel B: Certificates in Past 5 Years	Certificates			rtificates per 1000 Populati			rtificates per Old Housing U		
	Total	DOH	HRC	Total	DOH	HRC	Total	DOH	HRC
Bristol	597	0	426	11.97	-	8.54	0.24	-	0.17
Kent	3661	2427	936	22.03	14.61	5.63	0.13	0.09	0.03
Newport	2943	577	2372	39.03	7.65	31.46	0.52	0.10	0.42
Providence	24724	7421	17483	39.45	11.84	27.90	0.06	0.02	0.04
Washington	1072	254	823	9.06	2.15	6.96	0.08	0.02	0.06

Appendix Table 4: Assessing Validity of the Instruments

	(1) Mother African-American	(2) Free/Reduced Lunch	(3) Maternal education in years	(4) Maternal age at birth	(5) Married at Birth
DOH Certificates at birth/Old Housing	-0.584	3.338	2.668	0.627	0.812
	[0.314]	[0.467]	[3.128]	[7.130]	[0.538]
DOH Certificates at birth/Old Housing s	6.314	-25.13	-12.26	-21.89	-2.967
	[3.049]	[4.543]	[30.48]	[69.34]	[5.232]
Observations	60,582	60,582	58,392	60,559	60,571
R-squared	0.001	0.011	0.002	0.002	0.001
Mean of dependent variable	0.078	0.269	13.17	28.5	0.61
Effect size at the mean of DOH certifica	-0.007	0.048	0.043	-0.001	0.014
Effect size at the top 10% of DOH certifi	-0.013	0.092	0.084	-0.007	0.027

Note: These regressions included all of the variables in Table 4A column 4, census tract and year of birth FE.

Appendix Table 5: Mobility of the Sample

Panel A: Family Characteristics	Non-Mover (28%)	Mover (72%)
Black	0.058	0.125
Maternal Education	13.6	12.1
Free lunch	0.46	0.75
Married	0.69	0.41
Own Lead Level	3.19	4.11

Panel B: Tract Characteristics	Tract of Birth	Destination Tract
Tract Lead Level in 1997	4.64	4.44
# Certificates by 2004	0.044	0.039
Share Poor (2000 Census data)	0.178	0.163

Note: Mover is defined as a child whose tract at the last BLL differs from the tract of birth