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Lead and Juvenile Delinquency: New Evidence from Linked Birth, School and Juvenile Detention Records

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ABSTRACT

Using a unique dataset linking preschool blood lead levels (BLLs), birth, school, and detention data for 120,000 children born 1990-2004 in Rhode Island, we estimate the impact of lead on behavior: school suspensions and juvenile detention. We develop two instrumental variables approaches to deal with potential confounding from omitted variables and measurement error in lead. The first leverages the fact that we have multiple noisy measures for each child. The second exploits very local, within neighborhood, variation in lead exposure that derives from road proximity and the de-leading of gasoline. Both methods indicate that OLS considerably understates the negative effects of lead, suggesting that measurement error is more important than bias from omitted variables. A one-unit increase in lead increased the probability of suspension from school by 6.4-9.3 percent and the probability of detention by 27-74 percent, though the latter applies only to boys.

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Lead exposure in early childhood has been linked to diminished cognition, poor impulse control, inattention, and aggressive behavior (Wilson and Petersilia (1995), Needleman, Schell, Bellinger, et al. (1990), Needleman and Gatsonis (1991), Banks, Ferretti and Shucar (1997)). More recent studies seek to identify the effects of childhood lead exposure on crime by exploiting the de-leading of gasoline (Gronquist, Nilsson and Robling, 2014; Mielke and Zahran, 2012; Wolpaw Reyes, 2007 & 2015; Nevin, 2000 & 2007).[1] These studies suggest that reductions in exposure to lead in early childhood could explain up to 90% of the sharp downward trend in crime in the U.S. that started in the mid-1990s. However, there are many competing hypotheses for the large decreases in crime including abortion legalization (Donohue and Levitt, 2001), the decline of the crack-cocaine epidemic, changes in the availability of handguns, demographic shifts, changes in the demand for unskilled labor, improvements in policing, and increases in the prison population (see Blumstein and Wallman, 2004 for a discussion). Because so many of these factors co-vary, it can be difficult to distinguish the independent effect of any individual factor in a cohort analysis.[2]

In this paper we shift away from an exclusive focus on crime, which is a rare child outcome (especially among girls). We examine school disciplinary problems in addition to juvenile incarceration. Disciplinary problems are of interest in their own right as a precursor to school failure and drop out, as an outcome that can be observed in even relatively young children, and as an indicator that may be predictive of future criminal behavior (Bernberg and

[1] Other work has exploited the addition of lead pipes to municipal water systems (Feigenbaum and Muller, forthcoming; Ferrie, Rolf and Troesken, 2012) as a source of variation in lead exposure to estimate the impact of lead exposure in early childhood on homicide rates and adult IQ, respectively. See Rice (1992) for a summary of research on lead and aggression based on animal experiments.

[2] For example, California, in addition to de-leading gasoline ahead of the federal schedule, was also an early legalizer of abortion, and witnessed the early rise and decline of the crack-cocaine epidemic in Los Angeles.

Krohn, 2003; Mowen, 2016; Snyder and Sickmund, 2006).[3] In our data, children who have been suspended from school are ten times more likely to be detained or incarcerated as adolescents or young adults. Juvenile incarceration is one of the more extreme consequences of juvenile crime, and arguably has its own negative impact on the trajectories of affected adolescents. Youth who are detained for even a short period are less likely to graduate from high school and more likely to recidivate as adults (Aizer and Doyle, 2015). By examining school disciplinary problems as well as juvenile incarceration we provide a more nuanced picture of the effect of lead on anti-social behavior.

A second contribution is the use of unique individual-level data on all children born in Rhode Island between 1990 and 2004. Preschool blood lead levels are linked to birth records, school disciplinary records, and data on juvenile incarcerations. The relationship between lead exposure and juvenile anti-social behavior is likely to be confounded both by omitted variables and by measurement errors in blood lead levels. Omitted variables might be expected to lead to overstatements of the effects of lead since disadvantaged children are more likely to be exposed to lead. Measurement error could be expected to either attenuate or exaggerate the estimated effects of lead, depending on its form.

A third contribution is to develop two distinct identification strategies. Our first strategy exploits the fact that there are multiple measures of blood lead for the same child for 70-80% of our sample, or sometimes for multiple children in a family. Hence it is possible to instrument one blood lead measure with another for either the same child or a sibling, while at the same time

[3] This relationship could reflect differences in underlying behavior, a causal relationship through a labeling effect, or an "incapacitation effect" of being in school. Monahan et al. (2014) compares outcomes for the same child over time and finds that during periods when children are suspended from school, they are more likely to be arrested compared with periods during which they are in school.

controlling for child, family and neighborhood characteristics. Under the null hypothesis that the errors are uncorrelated (as they would be, for example, if they originated mainly from common random laboratory measurement errors) this procedure provides an effective way to deal with attenuation bias due to measurement error in blood lead levels.

Our second identification strategy can be used with all children (not just those with multiple measures or siblings), and focuses on the children most affected by exposure to residual lead from busy roads within a neighborhood. Before the de-leading of gasoline, the lead in car exhaust fell within 25-50 meters of the road, such that the soil near roads was historically more contaminated with lead than soil further from roads (Milberg et al, 1980; Fu et al, 1989). Consistent with this observation, we show that for children born in the early 1990s, there is a strong relationship between child blood lead levels and traffic on roads within 25-50 meters of the child's residence. However, the amount of lead in soil has declined over time following the de-leading of gasoline between 1979 and 1986, and so too has the relationship between road proximity and child blood lead levels. We find that for those born in 2004, lead levels vary little with proximity to traffic.

We adopt an instrumental variable approach that exploits the greater decline over time in child lead levels for those with more exposure to traffic within a neighborhood. Our instrument is an interaction between birth year and proximity to high traffic roads within a neighborhood. We show that over this time period there was little change in the background characteristics of children living near high traffic roads, providing some support for the assumed exogeneity of the instrument. Nor do conditions appear to be improving more generally for those near busy roads: Birth weight, which is very sensitive to maternal conditions and air pollution, does not change over time with exposure to traffic.

We find that both school disciplinary infractions and juvenile incarceration rise with preschool blood lead levels. Surprisingly, all our IV estimates indicate that simple ordinary least squares (OLS) estimates considerably understate the negative effects of lead. Since lead exposure is associated with other measures of disadvantage that tend to predict worse child outcomes, one might have expected OLS estimates to overstate lead's effects. Thus, our results point to mismeasurement of lead exposure as an important source of bias that has been largely ignored in previous research.

The IV estimates suggest that a one unit increase in blood lead levels (BLLs) increased the probability of suspension from school by 6.5 to 7% for boys and by 6.4 to 9.3% for girls, depending on the instrument and specification. For incarceration, we find significant effects only for boys. The estimates for incarceration are less precise (given low levels of detention in the sample) and vary from a 27% to a 74% increase for each one unit increase in BLLs. Nevertheless, our results are consistent with the large estimated effects of lead on crime in earlier cohort-based studies and support the hypothesis that reductions in blood lead levels may indeed have been responsible for a significant part of the observed decrease in anti-social behavior among youths and young adults in recent decades.

II. Background

Lead mimics calcium in the body and interferes with all systems that require calcium to function effectively including the renal, cardiovascular, and nervous systems. Children absorb lead via ingestion or inhalation and once absorbed, lead impairs brain development and disrupts neurotransmitter function in ways that negatively affect cognition, attention and short term

memory, and reduce impulse control. Children are more susceptible to lead than adults: Whereas nearly all of the lead taken into an adult body will leave in waste within weeks, for children only a third of the lead taken in will leave in waste (US Dept. of Health and Human Services, 2007).

Once lead is either ingested or inhaled, it enters the blood stream and is deposited in soft tissues and organs including the brain. It does not remain in the blood for long (the half life of lead in blood is 36 days), suggesting that a single blood lead measure could fail to capture all but recent exposures. Sampling or laboratory errors also add random noise to lead measurement that could lead to under-estimates of the effects of lead exposure, as discussed further below.

Historically, the two main sources of lead in the environment were gasoline and household paint.[4] Beginning in the mid-1920s, lead was added to gasoline in order to improve engine functioning. With the increase in traffic over the 20th century, the amount of lead from automobile exhaust increased dramatically from 0.3 tons per 1000 people in 1937 to 1.3 tons by 1974 (Nevin, 2000). By 1971, mounting evidence of the negative health effects of lead culminated in the U.S. Surgeon General's pronouncement that lead and childhood lead poisoning was a public health hazard (U.S. Surgeon General, 1971). In response, the federal government banned the addition of lead to household paint starting in 1978 and required the phase-out of lead from gasoline over the period 1979 to 1986.[5] As a result, the amount of new lead in the environment fell back to its 1937 level within a few years (Laidlow and Filipelli, 2008).

Lead in automobile exhaust, because of it's weight, generally fell within 25-50 meters of the automobile/road. As a result, because of the greater (historical) traffic in urban areas,

[4] A third historical source is lead smelters, with significant but localized exposure. The last lead smelter in the U.S. was closed in 2013.
[5]Gasoline was mostly de-leaded by 1986 and completely de-leaded by 1996.

concentrations of lead in soil are highest in urban areas, and within urban areas, the soil is most contaminated near roadways (Batelle, 1998; Lejano and Ericson, 2005; Filipelli et al, 2005).

Children absorb this residual lead from soil via inhalation and/or ingestion. Sometimes children eat soil (pica) but inhalation through roadside soil resuspension is more common (Laidlaw and Filipelli, 2008). Resuspension is positively related to the amount of turbulence generated by weather and by vehicles passing (Lough et al, 2005). This resuspension of lead in the air outside of homes is responsible for roughly half of lead found inside homes.[6] Open doors and windows are the primary ways in which lead bearing dust enters, but in old homes even closed, "leaky" doors and windows can represent a major pathway for lead dust (Sayre and Katzel, 1979). Lead is also frequently tracked into homes via shoes and clothing (Hunt et al, 2006).

In a review of the literature on the sources of lead poisoning among urban children, Laidlaw and Filippelli (2008) conclude that "interior paint AND pb-enriched soils are both harmful sources of Pb to children, with paint the likely culprit in cases of acute Pb poisoning, and soil an important source of Pb in the myriad examples of chronic Pb poisoning of urban children" (page 2023). As we will see, there are many cases of low level lead exposure in our data and relative few cases of what appears to be acute lead poisoning leading us to focus on lead deposited in soil in this analysis.

Fortunately, the amount of lead in soil has been declining over time as a result of physical and geochemical transformations. Over time, lead in the soil near roadways gets washed out, blown or tracked away, and/or complexed onto soil particles, posing less of a threat to children

[6] One study found that half of the lead in house dust in 10 homes in Jersey City came from sources outside of the home such as soil (Agate et al., 1998a).

(Ming et al, 2012; Datko-Williams, Wilkie and Richmond-Bryant, 2014). The decline in bioavailable lead in soil over time has been greater in areas near roads than in areas far from roads, since the former started with much higher levels which then decayed over time. This convergence in lead levels between areas near and far from busy roads provides one source of identifying variation that we will use to estimate the causal impact of lead on future behavior, as described below.

Lead paint remains a threat in many older homes. Aizer et al. (2017) examine the effect of a Rhode Island program that aimed to reduce children's exposure to lead paint by requiring landlords to obtain lead-safe certificates in order to rent their homes beginning in 1997. The younger cohorts in our study are likely to have benefitted both from the de-leading of gasoline and from the certificate program. However, the certificate program targeted particular Census tracts, rather than distance to roads. We show below that controlling for the number of certificates that had been issued in the Census tract where the child lived as of the time of their first lead test (a proxy for the intensity of the lead paint clean up efforts) has little impact on the estimates reported below which are identified by variation in lead in soil near roads.

Disadvantaged children are more likely to live in urban settings which in turn have more lead as a result of greater traffic congestion and an older housing stock. Even within urban settings, disadvantaged children have higher blood lead levels because they disproportionately reside in neighborhoods characterized by both greater road density and the oldest homes (Jacobs et al, 2002; Rowangould, 2013). As we will show below, these patterns also hold in Rhode Island and hence OLS estimates may be biased by unobserved confounders that are correlated with both lead exposure and worse child outcomes.

While disadvantaged children are more likely to be exposed, it is also possible that the same lead burden has a greater effect on poor and minority children. Billings and Schnepel (2015) show for example, that intensive intervention is helpful in reversing the effects of lead poisoning. To the extent that more advantaged children are more likely to receive such interventions they may suffer less harm than other children. Poor nutrition (especially iron and zinc deficiency) increases the absorption of lead in a child's system (Landrigan et al, 1976), and poor children may be more likely to have such deficiencies. In addition, recent research based on animal experiments suggests a strong interactive effect between stress (which is more prevalent among the disadvantaged) and exposure to lead.[7] Hence, in what follows we will ask whether the estimated effect of a unit exposure to lead appears to be different for more disadvantaged children.

However, it is possible that disadvantaged children who display the same behaviors as other more advantaged children are more severely punished via suspensions or juvenile incarceration (Rocque, 2010; Kinsler, 2011). So while we can observe whether some children suffer a greater burden from lead exposure in terms of suspensions and incarcerations, we will not be able to directly attribute observed differences to differences in a child's underlying behavior rather than to differential responses of society to the child's behavior.

III. Data

A. Lead and Child Outcomes in Rhode Island

[7] Two animal experiments include Amos-Koohs et al, 2016 and Cory-Slechta et al, 2010.

In order to estimate the relationship between preschool child blood lead levels and later problem behavior, we have created a unique data set for Rhode Island children born between 1990 and 2004. Data on each child's preschool blood lead levels from the state Department of Health's (RIDOH) screening program (1994-2010) is linked with information from the Rhode Island Department of Education (RIDE) on in-school disciplinary infractions resulting in suspension from school for school years 2007-2008 through 2013-2014. This data is then linked to information from the Rhode Island Training School, the state's juvenile detention facility (which houses juveniles up to age 17) as well as to data from all state correctional institutions (which house individuals age 18 and older). Data on detentions is available for 2004-2014.[8]

The state of RI is an ideal place to study the effects of lead on future outcomes because of the state's aggressive lead screening program. Reports from 2001 to 2011 indicate that over 70 percent of all children had at least one lead screen by 18 months and the screening rate was roughly constant over this time period, ranging from 71 to 75 percent (RIDOH, 2011). Children on Medicaid and children living in Providence have slightly higher screening rates.[9]

In contrast, the national screening rate ranged from 22 (in 2002) to 30 percent (in 2010) (Raymond, Wheler and Brown, 2014). In many jurisdictions children are screened only if there is some reason to suspect lead poisoning, so that the sample of children who are tested is not representative of all children in the jurisdiction. Given the high screening rates in RI, the sample with lead tests is much more similar to the state-wide population of children. Moreover, because

[8] The number of years for which we have outcome data for individuals in our sample varies. Year of birth is strongly predictive of years of outcome data available and therefore including year of birth fixed effects in our analysis implicitly controls for the number of years of outcomes data. However some children might leave the state or repeat a year in school and this would change the number of years for which we have outcome data. In our analyses, we also control for the number of years of suspension data that we have for each individual.

[9] In Providence, nearly 90% of all children have a BLL by kindergarten entry (McClaine et al, 2013) and 80% of children on Medicaid in the state had been screened for lead by 36 months (Vivier et al, 2001).

children without obvious symptoms were tested, we have a large sample of children with relatively low lead levels and can examine the effects of these low lead levels on outcomes.

Blood lead levels (BLLs) were measured multiple times for most RI children between birth and the age of 6. Seventy-three percent of children with at least one BLL have more than one BLL. The data for each test include the BLL, the age of the child in months, method of collection (capillary vs. venous), and the child's address at date of measurement. Blood lead is not normally measured for children over 6 years old so in all cases we are examining the relationship between preschool BLLs and later juvenile or young adult outcomes.

There is significant measurement error in blood lead levels that derives from three sources. The first two concern mismeasurement in each individual blood test. The CDC sets an acceptable range for measurement error at 4 ug/dL or 10 percent, whichever is greater (Parsons and Chisolm, 1997). At BLLs below 10 ug/dL, this margin represents a considerable amount of noise. Moreover, the sample can be contaminated, though this is less common. Contamination, if it occurs, is more likely in blood drawn via a finger prick (capillary method) than a needle (venous method). For this reason, venous measures are typically considered more accurate measures of child BLLs.[10] The third source of measurement error relates to the fact that the half life of lead in blood is approximately 36 days. As such, lead levels in whole blood indicate mainly recent exposure, although "there can be variable (but not dominant) input to total blood lead concentration from past exposure" (National Research Council,1993).

[10] A review of the research comparing measures of BLL from capillary and venous sampled drawn on the same day suggest a strong correlation between capillary and venous measures, and while capillary measures tend on average to be slightly higher, it does happen that venous measures can be higher than capillary measures (Parsons, Reilly and Essernio-Jensen, 1997).

These sources of noise or error in measures of blood lead levels can lead to either over or under estimates of a child's true lead burden in OLS regression. Random errors of the type implied by the CDCs margins of error would tend to cause understatement of the estimated effects of lead. The short half life of lead in blood would tend to lead to understating the extent of lead exposure, and thus overstating the estimated effect of lead on outcomes in OLS regressions. As we will show below, the evidence suggests that in practice measurement error leads to considerable understatement of the effects of lead.

Because we have on average 3 tests per child, we will have a noisy measure of lead burden over the child's first 72 months of life, but one that we will show is considerably more informative than a single measure of blood lead would be. To generate a summary measure of blood lead levels for each child, we calculate the geometric mean over all tests, a procedure that minimizes the influence of outliers and is consistent with the existing literature.

The data on disciplinary infractions includes the reason for the disciplinary infraction, the type of discipline, a scrambled school ID, year of infraction, student race, gender, ethnicity and free lunch status for school years 2007/8 to 2013/14.[11] For each child we have, on average, five years of infractions data. With these data we construct a single summary outcome measure: Whether the child was ever suspended. Roughly 20% of children in our sample were ever suspended compared to 24% who ever had any sort of disciplinary infraction suggesting that most students who ran afoul of school authorities were eventually suspended. Our second outcome is whether the child was ever detained in the state's juvenile detention center or state

[11] For all infractions: 37% are for disorderly conduct or disrespectful behavior, 10% for fighting/assault, 28% for skipping school or detention, 4% for threats/harassment and 1% for drugs. For infractions leading to suspension, 36% are for disorderly conduct/disrespectful behavior, 15% for fighting/assault, 21% for skipping school/detention, 6% for threats/harassment, and 2% for drugs. Fifty-five percent of infractions resulted in suspension.

correctional facility. In our sample, 1.1% of all children were ever detained or incarcerated (1.8% of boys). When we examine this measure, we limit our data to children born between 1991 and 1999 because few younger children are detained or incarcerated.

For a subset of these records, those born in the state of RI after 1996, we also have linked vital statistics natality data (birth certificates). The natality data include child birth weight, birth order, maternal marital status, age and education at time of birth. They also include a maternal identifier that allows us to identify siblings in the data.

Table 1 provides an overview of our data. There are significant disparities in blood lead levels by race and income, just as one sees in national data (Raymond, Wheeler and Brown, 2014). For all children, the average preschool BLL of children in our sample is 3.8 micrograms per deciliter (ug/dl); for white children, the average is 3.4 ug/dl, while Black and Hispanic children have levels of 5.3 ug/dl and 4.5 ug/dl respectively. If we categorize the sample by free lunch status (i.e. children who were ever observed to receive free school lunch), the average BLL of free lunch children is 4.5 ug/dl compared with an average BLL of 3.0 ug/dl among students who always paid for their lunch. Note that BLLs of less than 5 ug/dl (10 in the earlier years of our data) were unlikely to be treated in any way over most of our sample period. Treatment consists of chelation, which involves removing circulating lead from blood, but does not remove lead that has already been deposited to organs such as the brain.

These overall means mask important trends in both lead levels and disparities over time. Over time, both average lead levels and disparities by race and income have declined considerably (see Appendix Figures 1 and 2). Among children born in 2004, the average lead

level had declined by 3.2 ug/dl, the black-white gap had shrunk from 3.6 to 0.8 ug/dl and the income gap (free lunch vs paid) had shrunk from 2.7 to 0.9 ug/dl.

The dramatic declines in lead levels were accompanied by large declines in disciplinary infractions that were likewise greater for African-American children and free lunch children. Table 1 shows that overall, about 24 percent of children had a disciplinary infraction over the time period that we observe them, and 20 percent were suspended. These rates are more than double for African-American compared to white children. Appendix Figures 3A and 3B show that rates of infractions were falling over the time period, though they are quite different for different grade levels. Interestingly, middle school students have the highest rates of disciplinary infractions, though this may reflect high rates of drop out in higher grades (assuming that the most disruptive students drop out). Rates of juvenile detention are very low in the full sample at only 1.1 percent, but rise to 3.5 percent among African-Americans, and 5.9 percent among African-American males. One sees similar gaps in juvenile detention between free lunch and paid lunch children with rates of 3.2 percent vs. .3 percent among males in these two groups, respectively.

The rest of Table 1 traces out the familiar pattern of disadvantage that one might expect. In addition to higher lead levels, African-American and free lunch children tend to have less educated mothers, younger mothers, single mothers, lower birth weight, and more siblings as reflected in a higher birth order. These differences highlight the challenges involved in separating the effects of lead from the effects of other disadvantages.

B. Lead and Exposure to Traffic

In order to measure a child's potential exposure to residual lead in soil near roads, the data on child blood levels were matched, based on the child's geocoded address at the time of each test, to data on Rhode Island roads from the RIGIS E-911 data for the period 2001-2014.[12] For each child and blood test, we measure the number of meters of road by road type within a radius of 25 and 50 meters of the child's home. We multiply the number of meters of each road type with measures of average traffic per road type based on 1980 national traffic data. We then calculate a measure of "average traffic" over the child's multiple addresses. The appendix describes the construction of this measure in greater detail.

To address changes in the road network over time, we compared satellite images from the 1980s with current data. Few differences can be seen with one exception: the expansion of roads in suburban subdivisions. In the robustness section we describe analyses that remove these areas with newer roads from our sample in order to test the sensitivity of our results to this source of mismeasurement of road density.

There is considerable variation in road density and traffic volume across the state even within a neighborhood. The median coefficient of variation in traffic within a census block group is 0.64 and ranges from 0.29 to 2.3. Figure 1 shows a map of a single RI census tract made up of five census block groups to illustrate how this measure of traffic exposure varies even across homes within a census block group. In the figure, each circle represents a home with a child. Circle radius indicates traffic volume within a 50 meter radius of the home, with larger radii indicating greater traffic volume. The traffic volume for each home is a function of the number

[12] Because the matching process required access to address, the match was performed by the Providence Plan which created and maintained the linked dataset.

of meters of road within 50 meters and the type of road, with highways generating the most traffic, town streets the least, and state and county roads somewhere in between.

Table 2 shows that disadvantaged children are more likely to live in high traffic neighborhoods. When we compare the race and free lunch status of children who live in high traffic (top quartile) vs. low traffic (bottom quartile), we find that high traffic areas are 59% white and 33% paid lunch relative to low traffic areas which are 87% white and 64% paid lunch, a difference of 28 (white) and 31 (paid lunch) percentage points (Table 2, column 1). Much of this difference is due to the fact that disadvantaged children live in more urban parts of the state: When we condition on municipality, the difference in racial and income composition by traffic exposure declines from 28 and 31 percentage points, respectively, to 6 and 13 percentage points (Table 3, Column 2).[13] If we condition on census tract or census block group, the differences decline further still to 3 and 9 percentage points, respectively, as shown in columns 3 and 4. Clearly conditioning on neighborhood significantly reduces differences in child characteristics across high and low road dense areas, though it does not entirely eliminate the differences.

Because conditioning on neighborhood doesn't eliminate average differences in child characteristics between high and low traffic exposure homes, we do not simply use spatial variation in traffic exposure within neighborhoods for identification. Rather, we exploit changes over time in high vs. low traffic areas within a neighborhood. More specifically, the identifying assumption is that traffic exposure is more predictive of a child's blood lead levels early in the sample (i.e., for those born in the early 1990s), than for those born later in the sample (in the early 2000s).

[13] There are 39 cities.

Figure 2 shows the relationship between traffic exposure (measured in percentiles) and child preschool blood lead levels for children born in 1990 and children born in 2004. In order to focus on differences in traffic exposure within a neighborhood (defined as a census block group) we first regress lead levels and traffic measures on neighborhood fixed effects and then plot the residuals from these two regressions in Figure 2. The residuals capture variation in both lead levels and traffic within a neighborhood. The relationship between within-neighborhood traffic volume and child preschool BLLs (the slope) is steeper for the older children than the younger children in our sample.

For this identification strategy to be valid (that is, for the exclusion restriction to hold), it must be the case that the variation in lead exposure over time within high traffic areas is uncorrelated with underlying changes in the characteristics of children living in these areas that might also affect outcomes. In other words, the declines in children's blood lead levels cannot be correlated with changes in the underlying composition of the children that would predict probabilities of suspension or incarceration.

To provide evidence on this point, Figure 3 plots time trends in lead levels and child characteristics by birth cohort and traffic volume (top quartile vs. bottom quartile of traffic volume). The difference in the average lead levels of children with the highest vs. lowest traffic exposure are initially large, but converge over time (top left panel of Figure 3). In contrast, while the underlying characteristics of children (race, income and maternal education) differ in high traffic areas relative to low traffic areas, both the levels and the differences between high and low traffic areas are fairly constant over time (top right and bottom panels of Figure 3). We conclude that the convergence in lead levels for children living in high vs. low traffic areas is not driven by changes in the underlying characteristics of the children living in these areas.

Another possible explanation for the steeper declines in lead levels among children living in high traffic areas is that conditions are simply improving generally for disadvantaged children, who happen to live in high traffic areas of the state. To explore this possibility, we examine time trends in lead levels for advantaged children living near vs. far from traffic. If the declining lead levels are attributable to reductions in lead in soil and not to overall improvements in conditions among the most disadvantaged, then we should see that even among children from advantaged families, traffic exposure predicts child lead levels.

We explore this idea in Figure 4. In order to generate this figure we calculated a summary measure of child disadvantage: The predicted lead level of the child based on coefficients obtained from a regression of lead levels on child race, ethnicity, free lunch status, and neighborhood fixed effects using data from the 1990 and 1991 birth cohorts. Children with higher predicted lead levels are more disadvantaged and would have higher BLLs in 1990. We define the advantaged population of children as those with the lowest predicted lead (the bottom quartile). We plot the trends in lead levels for the most advantaged children in our sample separately for high traffic and low traffic areas. Figure 4 shows that even among the advantaged children, those in high traffic areas start with higher levels of blood lead which converge over time to the BLLs of advantaged children in low traffic areas. Thus, our results are not driven by contemporaneous policies mainly benefitting disadvantaged children.

Finally, could the declines in lead levels that we observe simply reflect more general improvements in environmental conditions over this period? To rule out this possibility, we show below that birthweight, an indicator of child health that is sensitive to pollution (Aizer and Currie, 2014) does not differentially improve over time for children near roads.

IV. Estimation

We first present estimates of the relationship between preschool BLLs and the probability of school disciplinary infractions and juvenile detentions based on the following OLS equation:

$$(1)\ Y_i = \beta_0 + \beta_1 Lead_i + \beta_2 X_i^1 + \beta_3 X_i^2 + \beta_4 Years\ of\ Infraction\ Data_i + \beta_5 Year\ of\ Birth * Race_{it} + \beta_6 Year\ of\ Birth^2 * Race_{it} + \beta_7 Year\ of\ Birth * Free\ Lunch_{it} + \beta_8 Year\ of\ Birth^2 * Free\ Lunch_{it} + \tau_n + \tau_t + \varepsilon_i$$

Each observation is a child, and *Y* is an indicator for ever suspended or juvenile detention/incarceration; Lead is the geometric mean of the child's multiple BLLs measured before the start of school, $\boldsymbol{X}^1$ is a vector of child characteristics that we have for all children (gender, race, ethnicity and free lunch status); $\boldsymbol{X}^2$ is a vector of child characteristics that we have only for children born in RI after 1996 (maternal education, birth weight, birth order, maternal age and marital status at birth); Years of infraction data is a vector of indicator variables for the number of years for which we have infraction data for the child which ranges from 1 to 8, with an average of 5. We also include a vector of neighborhood fixed effects, $\boldsymbol{\tau}_n$, year of birth fixed effects, $\boldsymbol{\tau}_t$, and race and free lunch specific quadratic time trends to address the concern that any improvements in outcomes could simply reflect improving circumstances over time for the most disadvantaged children generally.

As discussed above, OLS estimates may be biased by confounding, and are likely to be attenuated by random measurement error. Hence, we estimate two types of instrumental variable models. The first takes advantage of the fact that we have multiple measures for most of the

children in our sample.[14] Thus, we can estimate the impact of a single, randomly drawn measure of a child's BLL (a noisy measure) on behavior in an OLS setting and compare it with an estimate based on a less noisy measure of child lead that's based on the average of all the other lead measures. Not only should the noisier measure of lead result in a smaller estimated relationship between lead and suspensions, but once we instrument for the single measure with the average measure, the IV estimate should be considerably larger than the OLS.

For the subset of the data for which we also have sibling lead levels, we can use a sibling's average lead level as an instrument for the child's own average lead level. The rationale for using sibling lead levels as an instrument for own lead levels is that preschool lead levels are mostly a function of one's residence. Given that siblings usually co-reside, a sibling's lead level should reflect the same (or at least very similar) lead exposure. Note that we can only perform this last exercise for the school suspension outcome, not the detention/incarceration outcome due to data limitations—there are too few sibling pairs with incarceration data, given that sibling information is not available until 1997.

Our second instrumental variables strategy exploits declines in the residual lead in soil near roads to identify the estimated impact of preschool child BLLs on future behavior. Our instruments are interactions between quartiles of traffic volume and a linear time trend. The main terms (quartiles of traffic volume and year of birth) are included as controls in both the first and second stage. We use quartiles of traffic volume in order to allow for any non-linearity in the relationship between traffic volume and BLLs. The first stage equation is:

[14] Other papers that discuss this type of approach include Bound et al. (1994), Bound and Krueger (1991), and Ashenfelter and Krueger (1994).

$$(2)\ Lead_i = \alpha_0 + \alpha_1 X_i^1 + \alpha_2 X_i^2 + \alpha_3 Traffic\ Volume_i + \alpha_4 Traffic\ Volume * Year\ of\ Birth_{it} + \alpha_5 Years\ of\ Infraction\ Data_i + \alpha_6 Year\ of\ Birth * Race_{it} + \alpha_7 Year\ of\ Birth^2 * Race_{it} + \alpha_8 Year\ of\ Birth * Free\ Lunch_{it} + \alpha_9 Year\ of\ Birth^2 * Free\ Lunch_{it} + \tau_n + \tau_t + \mu_i$$

where Lead is the child's average BLL, $\mathbf{X}^1$ includes child gender, race, ethnicity and free lunch status, $\mathbf{X}^2$ includes maternal education, birth weight, birth order, maternal age and marital status at birth. Traffic volume is a vector of three indicator variables for the top three quartiles of road density (the lowest quartile is omitted), Traffic volume*Year of Birth is the interaction between each quartile of road density and a linear year of birth term. In the robustness section, we allow for a non-linear time trend for traffic volume by including quadratic terms as well (Traffic volume* year of birth^2). We include race and free lunch specific quadratic time trends as well as indicators for the number of years for which we have infractions data for each child, year of birth FE ($\boldsymbol{\tau}_t$) and census block group fixed effects ($\boldsymbol{\tau}_n$).

The second stage model is given by the following equation:

$$(3)\ Y_i = \gamma_0 + \gamma_1 Predicted\ Lead_i + \gamma_2 X_i^1 + \gamma_3 X_i^2 + \gamma_4 Years\ of\ Infraction\ Data_i + \gamma_5 Year\ of\ Birth * Race_{it} + \gamma_6 Year\ of\ Birth^2 * Race_{it} + \gamma_5 Year\ of\ Birth * Free\ Lunch_{it} + \gamma_6 Year\ of\ Birth^2 * Free\ Lunch_{it} + \gamma_7 Traffic\ Volume_i + \tau_n + \tau_t + \vartheta_i$$

where all the terms are as defined above except Predicted Lead which is the prediction created from equation (2).

V. Results

A. OLS Results

Table 3 shows OLS estimates of equation (1) with increasing numbers of controls as one moves across the columns. Comparing columns 1 and 2 shows that once we control for town or municipality, adding Census block group fixed effects has almost no impact on the estimated effect of lead on the probability of a suspension. The estimate of 0.0095 implies that each 1unit increase in average blood lead increases the probability of a suspension by about one percentage point on a baseline of 20 percent. Column 3 adds fixed effects for Census block groups and schools. This addition reduces the estimated effect of lead slightly. Finally, column 4 shows the effect of adding additional controls for family background and birth weight. This model is estimated using the sub-sample for whom we have this information. The estimate of 0.0089 is very little reduced from the column 1 model, suggesting that adding further demographic controls would not be likely to have much effect.

Columns 5 to 7 of Table 3 perform the same exercise for detentions and incarcerations on the smaller sample for whom we have this outcome. Once again, the results are quite stable when additional sets of controls are added suggesting that a fairly basic set of background controls may be sufficient to control for potential confounding. The point estimate here of 0.0014 on a baseline of 0.01 suggests that an increase of one unit in average blood lead levels increases the probability of detention or incarceration by about 10 percent.

A second way to test for the importance of omitted variable bias in the OLS estimates exploits the decline in negative selection into lead witnessed over this period. Assuming that observables and unobservables are positively correlated, as negative selection on observables declines over time, so too should negative selection on unobservables. If negative selection on unobservables is driving OLS estimates of the relationship between lead and suspensions, then the negative estimated relationship between lead and suspensions in OLS should likewise decline over time. We explore this conjecture in Appendix Table 1. We first present evidence of the decline in negative selection over time in elevated BLLs. This sample is limited to children born 1993-1998 so that we can examine a single uniform outcome for all: the number of infractions in 9^{th} grade. We regress lead levels on race, free lunch, gender, year of birth (linear), and census block group FE, as well as interactions between free lunch and year of birth and race (African American) and year of birth. The interaction terms are intended to capture any changes over time in selection on race and income into elevated lead. The results (column 1) show that being poor and African American is strongly predictive of elevated BLLs, but this relationship has been declining over time, as evidenced by the negative and significant interaction terms.

Next, we present estimates of a regression of the number of ninth grade infractions on child lead levels, lead interacted with year of birth and all demographic controls. While the coefficient on lead is positive and significant, the coefficient on the interaction between lead and year of birth is small and imprecise (column 2). Column (3) shows similar results for a specification that includes race and free lunch-specific time trends. This table suggests that although disparities in lead exposure are falling the relationship between a unit of blood lead and behavior is not changing over time which is inconsistent with the estimated effect being driven by omitted variables bias.

B. Heterogeneous Effects

As discussed above, there is considerable heterogeneity in exposure to lead, and there may also be heterogeneous effects of similar lead exposure by socioeconomic status. Heterogeneity in the effects of lead is explored further in Table 4. Panel A examines the effects of lead on the probability of any suspension. The specification is similar to that in column 2 of Table 3 except that lead is also interacted with an indicator for male. Column (1) shows that overall, the effect of lead on girls (0.0059) is roughly half the size of the effect on boys (0.0059+0.0056=0.012). But this smaller effect for girls is driven largely by white and paid lunch students (columns 2 and 5). Among whites the gender difference in the effect of lead on suspensions is even more pronounced with a point estimate of 0.0033 for girls compared to 0.0099 for boys. Among children who pay for their lunch, lead is only estimated to affect suspensions among boys.

In contrast, among African-American, Hispanic, and free lunch students, lead is estimated to have equal effects on boys and girls, that is, the interaction with male is not statistically significant. Among boys, the estimated effect of a unit of blood lead on the probability of suspension is actually rather similar for whites, African-Americans, or Hispanics. These results could be easily explained in terms of, for example, preferential treatment for white and higher income girls.[15] That is, the differences do not necessarily reflect any differences in the underlying behavior problems caused by a one unit increase in blood lead levels.

[15] Existing national data on racial differences in school suspensions shows that boys receive two thirds of all suspensions but black girls are suspended at higher rates than girls of any race and most boys (USDOE Office of Civil Rights, 2014).

Panel B shows a similar breakdown for detention/incarceration. This panel makes it clear that lead only appears to affect detention/incarceration among boys, with a clear gradient by socioeconomic status. Among boys, whites and children who pay for their lunch have much lower estimates than African-Americans, Hispanics, and children on free lunch. Again it is not entirely clear that these differences reflect differences in behavior vs. differences in punishments conditional on behavior. It should be noted that while the point estimates are larger for African American, Hispanic and free lunch children, relative to the baseline rates of detention for these groups, the effects are actually smaller. For example, a unit of lead increases the probability of incarceration for black males by only 6.6 percent compared to 16.6 percent for white males.

In what follows we will allow estimated effects to vary for males, which seems very important at least in the case of incarceration. We will also show separate estimates for the free lunch sample, as it is the group most vulnerable to suspensions and incarceration.

C. Instrumental Variables Estimates Based on Multiple Measures of Lead

The estimates in Tables 3 and in Appendix Table 1 suggest that given the rich controls available in our data, perhaps residual confounding is not a large source of bias in our estimates. However, as we will show below, there is good reason to believe that measurement error remains a problem, biasing the estimated effects of lead shown in Tables 3 and 4 towards zero. The fact that there are multiple lead measures for each child, and in some cases for each family, suggests several possible instrumental variables estimates based on using one measure as an instrument for another noisy measure of the child's underlying level of lead exposure. Table 5a shows three different ways in which we have implemented this idea.

In columns 1 to 4, we focus on the subset of children who have both a capillary and a venous BLL measure. Since capillary measures are less accurate, we would expect the use of a capillary measure to result in an estimate that is smaller than one based on a venous measure. A comparison of columns 1 and 2 shows that this is indeed the case. In column 3 we use the average of the venous tests as instruments for the average of the capillary test measures, and in column 4 we do the reverse. Both instrumental variables estimates are considerably larger than the corresponding OLS estimates, consistent with measurement error in these tests. Column 4 suggests, for example, that the probability of having ever been suspended increases by 1.2 percentage points for girls and 1.6 percentage points for boys for each one unit increase in BLLs (on a baseline of 20 percent of children who have any suspension). A drawback of this approach is that relatively few children in our sample (24,509) have both types of measures and they are unlikely to be a random sample of all children.

In columns 5 through 8 we therefore experiment with taking a random draw and instrumenting for it using the average of all the other available measures, and vice versa. This procedure is possible for any child who has more than one BLL. Columns 7 and 8 show that these IV estimates are again much larger than the corresponding OLS estimates shown in columns 5 and 6. For example, column 8 suggests that based on the instrumented estimate, a one unit increase in the average BLL increases the probability of any suspension by .96 percentage points for girls, and by 1.7 percentage points for boys, compared with .49 and .96, respectively, for the OLS.

Finally, in column 9 we show estimates where one child in the household's average BLL is instrumented using a sibling's BLL (if there are multiple siblings, we use the average of the siblings' BLLs). This estimation can only be done on the subset of children (34,252) who have a

sibling with a BLL. The estimates of 1.2 for girls and 2.98 for boys are somewhat larger than those discussed above.

Panel B of Table 5a performs the same estimations for detention/incarceration, with the exception of the sibling IV. The latter is infeasible because the sibling subsample was born in 1997 or later so that they are too young for the incarceration outcome. Again these estimates suggest that OLS estimates are biased towards zero due to considerable measurement error in BLLs. For example, column 6 shows the OLS estimate of lead on suspension using the average of all other tests as the measure of lead. The estimated effect of a one-unit increase in BLL for males is .35 percentage points. Column 8 shows the IV estimate, where the instrument is a randomly drawn BLL. The estimated effect increases to .56 percentage points, on a baseline of 2.04 percent for males, suggesting a very large effect of lead on the probability of incarceration.

Table 5b repeats these estimations using the sample of children with a much higher baseline rate of suspensions and incarceration: children who ever participated in the free school lunch program (Table 1). These results suggest larger effects for girls in this sample, but similar effects for boys (Panel A). However, given the different baseline probabilities of suspensions in this sample, the estimates for boys, for example, translate into a 4.3% increase in the probability of suspension per one-unit increase in BLL in the free lunch sample compared to a 6.5% increase in the full sample.

For the incarceration outcome, Panel B of Table 5b shows that the point estimates of the effects of lead are remarkably similar in the full and free lunch samples. Comparing the IV coefficients in column 8, panel B of Tables 5a and 5b suggests that a unit increase in BLL increases the probability of male incarceration by .56pp in the full sample and by .55pp in the

free lunch sample. Given the baseline differences in the probability of incarceration, these estimates translate into increases of 27% and 16.9% for the full sample and the free lunch sample respectively.

Overall, these IV estimates suggest that the true effect of lead on suspensions is considerably understated by OLS estimates, consistent with a large amount of measurement error in BLLs. The estimates also suggest that the underlying effects of lead may be quite similar in more and less disadvantaged samples, although a given increase in disruptive behavior results in larger percentage increases in negative outcomes among more advantaged children given their lower baseline probabilities of suffering either suspensions or incarcerations.

D. Instrumental Variables Estimates Based on Exposure to Traffic

In this section, we present estimates based on our traffic IV. These estimates measure the effect of lead on the children who were "treated" by a reduction in ambient lead from roadways near their homes. Regression models linking traffic volume with child BLLs from equation (2) are shown in Appendix Table 2. Exposure to traffic is a highly statistically significant determinant of child BLLs (see the coefficients on the indicators for Traffic volume quartile 2 to Traffic volume quartile 4). The relationship is strongest for children in the top quartile of traffic exposure. As discussed above, our instrument is not traffic volume, but the interaction between the traffic volume indicators and a linear time trend. The identifying assumption is that places with higher traffic volumes saw larger reductions in blood lead levels, due to the reduction over time in the amount of residual lead near roadways, and not some other factor that also affected children's probability of being suspended or incarcerated. Recall earlier evidence that changes in

the composition of children are not driving the decline near roadways (Figure 3), nor are general improvements in the outcomes of disadvantaged children over this period (Figure 4).

Because we are allowing the effects to differ for males, by including the interaction term lead*male, we use the predicted lead level from column 1 of Appendix Table 2 to predict lead levels for all children, and then interact this variable with an indicator for male to form an instrument for lead*male (Wooldridge, 2010). Table 6 presents the first stages for the two endogenous variables: lead (column 1) and lead*male (column 2). Column 1 of Table 6 is similar to Appendix Table 2 and like Appendix Table 2, shows a strong relationship between traffic and lead that declines over time. However, this first stage differs slightly from the Appendix regression because it also includes the instrument for lead*male. In column 2 of Table 6, the instrument, predicted lead*male, is highly statistically significant.

Estimates of the second stage regression (equation 3), including the lead*male interaction and using the traffic instrument, are shown in columns 2 and 4 of Table 7 along with the corresponding OLS estimates from column 1 of Table 4 (reproduced in columns 1 and 3 of Table 7 for convenience). The estimates in column 2 Panel A suggest that a one unit increase in her average BLL raises a girl's probability of being suspended by 1.3 percentage points on a baseline of 14 percent. The corresponding estimate for boys is 2.7 percentage points on a baseline of 25 percent. In percent terms, the effects are not very different, suggesting a 9.3% increase in the probability of suspension for girls and a 10.8% increase in the probability of suspension for boys. These results can be compared to the IV estimated based on multiple measures of lead presented in Table 5a which implied that a one unit increase in BLL would increase the probability of suspension among boys and girls by 6.4% and 6.5% respectively.

Column 4 in Panel A of Table 7 shows the traffic IV estimates of the effect of lead on the probability of incarceration in the full sample. As with the IV estimates based on alternative lead measures (Table 5a), there is no statistically significant effect for girls. For boys, the estimate implies that a one unit increase in average blood lead increases the probability of incarceration by 1.3 percentage points, on an overall baseline incarceration rate of 1.8 percent. This is a large percent effect, in part because the baseline incarceration rate in the overall population is so small.

We also present these results for the free lunch sample, given that this is the population most at risk for these outcomes. In terms of the estimated effects of BLLs on suspension, the estimates suggest once again that in the free lunch sample, lead increases the probability of suspension for both boys and girls, whereas in the full sample, the effect was only statistically significant for males. The point estimates suggest that a one unit increase in BLL increases the probably of suspension by 24% for boys and 39% for girls in the free lunch sample.

The estimated effects of lead on incarceration are also larger in the IV setting, and once again found only for boys. A one unit increase in BLL is estimated to increase the probability of incarceration by 1.8 percentage points. However, given the much higher incidence of incarceration in free lunch group, the percentage increase is smaller than in the full sample, at 57%.

Clearly, the percent changes in incarceration are very sensitive to the baseline level chosen for comparison, given the small incidence of incarceration in the population. However, regardless of the correct baseline for computing percentage effects, the traffic IV point estimates for incarceration in Table 7 are much larger than the multiple measures IV estimates in Table 5,

which is in line with the idea that the traffic IV is picking up the effect of lead in the most vulnerable population.

E. Robustness

We conducted four robustness checks (Table 8). First we include a control for the average number of infractions per year in the child's school*birth cohort to account for any changes in policy at the school*cohort level that might also influence the number of disciplinary infractions (column 2).[16] Second, we add controls to account for the Rhode Island Lead-Safe certificate program analyzed by Aizer et al. (2017). This program aimed to encourage landlords to mitigate lead hazards in homes for rent. Specifically, in column 3 we add controls for the number of certificates that had been issued in the census tract where the child lived as of the time of their first lead test (a proxy for the intensity of the lead paint clean up efforts).

As a third robustness check, we drop all census tracks for which we observe substantial changes (increases) in roads over this period. Sixty-six percent of our sample live in tracts that gain no new roads between 1980 and today. Eight percent of our sample live in tracts that gain a substantial number of new roads. These are mostly suburban subdivisions. No one lives in a tract that loses roads over this period. When we drop the sample that gain a substantial number of new roads, the sample size falls to 114,512 from approximately 125,000, and the estimates remain largely unchanged (column 4). Finally, we include quadratic trends for traffic*birth

[16] Construction of this control proceeds as follow: for all children in the RI schools in 6th grade, we include the average number of infractions in that child's school for 6th graders. For children missing information on 6th grade (because they were born early or late in the period), we include either 3rd grade averages (for those born later), or 9th grade averages for those born earlier.

cohort in the IV regressions (column 5, panel B). The results suggest that the estimates are robust to all these modifications.

Could traffic volume and the interaction between traffic volume and birth cohort simply reflect changes in housing or air quality more generally over this period? If so, one might expect to estimate a significant relationship between traffic volume and birth weight, a measure of newborn health that is very responsive to both maternal circumstances and air pollution (Aizer and Currie, 2015). To consider this possibility, we present reduced form estimates of the impact of traffic volume and its interaction with year of birth on birth weight as well as neighborhood fixed effects and IV estimates of the impact of lead levels on birth weight in Table 9.

There appears to be no relationship between traffic volume or its interaction with birth cohort, and birth weight (Table 9, column 1). Moreover, there does not appear to be any meaningful or significant relationship between lead and birth weight (columns 2 and 3): The OLS estimate of -.00291 would suggest that a 3.5 unit decline in lead (the decline witnessed over the 14 year period studied here) is associated with a 10 gram decline in birth weight (relative to a mean birth weight of 3,300 grams) and the IV estimate is similarly small in magnitude though positive and statistically insignificant. We interpret these estimates as suggestive evidence that the decline in lead levels over time in high traffic areas does not reflect improving conditions more generally as these might also be expected to affect birth weight.

VI. Conclusions

This paper makes several contributions to the literature examining the link between lead poisoning and anti-social behavior among juveniles. First, we broaden our study away from the

exclusive focus on crime, to consider school disciplinary problems. Disciplinary problems predict school failure and drop out, are much more common, and can be observed in both boys and girls. Suspensions are also highly predictive of future criminal activity: in our data, children who have been suspended are ten times more likely to be involved in criminal activity. We also look at juvenile detention/incarceration, which is an indicator of crime but also has its own negative impacts on juveniles.

A second contribution is to add to a literature that has relied on variation in exposure to lead at the state-cohort level by constructing and examining rich individual-level data from linked administrative records for all children born in Rhode Island between 1990 and 2004 and exploiting very local variation in exposure to lead. This long time span allows us to link preschool BLLs to the outcomes of children in middle school and beyond. By including individual-level controls and also controlling for neighborhoods at the level of the Census block group, we are able to alleviate concerns about confounding due to omitted variables.

A third contribution is to develop two identification strategies which rely on different assumptions. Both identification strategies indicate that OLS estimates are considerably attenuated by measurement error in BLLs, an important result given that attenuation due to measurement error has been largely ignored in the previous literature.

We find that a one unit increase in BLLs increased the probability of suspension from school by 6.5 to 7% for boys and by 6.4 to 9.3% for girls. For incarceration, we only find statistically significant effects for boys. The estimates are less precise and suggest that a one unit increase in BLL increases the probability of incarceration by between 27% to a 74% increase for each one unit increase in BLLs. Given low baseline levels of incarceration, the estimated

probabilities are quite sensitive to the exact baseline level chosen. Still, our results support the hypothesis that reductions in blood lead levels may have been responsible for a significant part of the observed decrease in anti-social behavior among youths and young adults in recent decades.

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Table 1: Summary Statistics for Sample by Child Characteristic

	All	White	Black	Hispanic	Free Lunch	Paid Lunch
Lead (Geometric Mean per Child)	3.800	3.400	5.290	4.500	4.500	3.000
Number of tests	2.600	2.400	3.100	3.300	2.700	2.300
Share capillary	0.320	0.370	0.267	0.180	0.280	0.370
Any school disciplinary infraction	0.240	0.200	0.409	0.350	0.350	0.120
Any suspension	0.200	0.150	0.358	0.290	0.290	0.090
Any detention/incarceration	0.011	0.006	0.035	0.019	0.019	0.002
Any detention/incarceration - males only	0.018	0.009	0.059	0.033	0.032	0.003
Number of years with school infraction data	5.300	5.300	5.200	5.400	5.400	5.200
Traffic Volume (as a % standard deviation relative to mean)	0.000	-0.073	0.184	0.166	0.104	-0.120
Black	0.093				0.160	0.020
White	0.694				0.490	0.940
Hispanic	0.175				0.310	0.020
Asian	0.034				0.040	0.020
Sometimes Free Lunch	0.294	0.257	0.395	0.370	0.550	
Always Free Lunch	0.241	0.117	0.500	0.570	0.450	
Mother <HS	0.096	0.052	0.150	0.225	0.169	0.010
Mother HS	0.186	0.179	0.230	0.198	0.239	0.126
Mother College+	0.162	0.209	0.067	0.042	0.056	0.284
Maternal age	28.400	29.400	26.300	26.200	26.400	30.900
Mother married at birth	0.670	0.760	0.400	0.486	0.490	0.900
Birth Weight	3.340	3.360	3.250	3.300	3.300	3.400
Birth order	1.900	1.840	2.100	2.100	2.000	1.800
Observations (1990-2004 birth cohorts)	124579	90,813	11527	22239	66,710	57,869
Observations for birth certificate data (birth cohorts 1997+)	80078	53833	7805	15586	44732	35346
Observations for detention/incarceration (birth cohorts 1991-199	76,646	54,722	6,602	12,700	37,040	39,606

Table 2: Road Density and Child Disadvantage

	Share White			
Area of Comparison	State	Municipality	Census Tract	Census Block Group
High traffic (top quartile)	0.586	0.699	0.709	0.710
Low traffic (bottom quartile)	0.866	0.754	0.744	0.742
Difference	-0.280	-0.055	-0.035	-0.0324

	Share Paid Lunch			
Area of Comparison	State	Municipality	Census Tract	Census Block Group
High traffic (top quartile)	0.327	0.416	0.431	0.436
Low traffic (bottom quartile)	0.638	0.548	0.534	0.530
Difference	-0.311	-0.132	-0.103	-0.094

Table 3: OLS Estimates of the Effects of Lead on Disciplinary Infractions and Juvenile Detention/Incarceration

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Suspension	Suspension	Suspension	Suspension	Incarceration	Incarceration	Incarceration
Geometric Mean of Lead	0.00953	0.00954	0.00898	0.00890	0.00145	0.00149	0.00137
	[0.000408]	[0.000416]	[0.000424]	[0.000539]	[0.000133]	[0.000137]	[0.000138]
Male	0.108	0.109	0.110	0.100	0.0142	0.0141	0.0136
	[0.00206]	[0.00206]	[0.00210]	[0.00231]	[0.000764]	[0.000768]	[0.000772]
African American	0.0695	0.0505	0.0690	-0.0182	0.00720	0.00554	0.00470
	[0.0255]	[0.0255]	[0.0261]	[0.157]	[0.00421]	[0.00426]	[0.00431]
White	0.0700	0.0759	0.0865	0.0633	-0.000871	0.000179	0.00119
	[0.00586]	[0.00600]	[0.00624]	[0.00660]	[0.00227]	[0.00234]	[0.00237]
Hispanic	0.0940	0.0848	0.0876	0.0694	0.00622	0.00442	0.00280
	[0.00617]	[0.00626]	[0.00649]	[0.00689]	[0.00238]	[0.00243]	[0.00246]
Sometimes free/reduced lunch	-0.0726	-0.0829	-0.0627	0.268	-0.00745	-0.00958	-0.0114
	[0.0144]	[0.0144]	[0.0152]	[0.0935]	[0.00231]	[0.00235]	[0.00241]
Always free/reduced lunch	-0.0209	-0.0381	-0.0249	0.311	-2.55e-05	-0.000942	-0.00219
	[0.0145]	[0.0145]	[0.0153]	[0.0935]	[0.00217]	[0.00220]	[0.00225]
Black*year of birth	0.0187	0.0193	0.0172	0.0280			
	[0.00506]	[0.00507]	[0.00519]	[0.0236]			
Black*year of birth squared	-0.000808	-0.000804	-0.000725	-0.00118			
	[0.000235]	[0.000235]	[0.000242]	[0.000868]			
Paid lunch*year of birth	-0.0663	-0.0660	-0.0583	-0.00161			
	[0.00293]	[0.00293]	[0.00311]	[0.0141]			
Paid lunch*year of birth squared	0.00377	0.00375	0.00333	0.00121			
	[0.000138]	[0.000138]	[0.000147]	[0.000523]			
Birth weight in kg				0.00363			
				[0.00214]			
Mother <HS				0.0132			
				[0.00422]			
Mother HS grad				-0.0135			
				[0.00318]			
Mother College+				-0.0221			
				[0.00342]			
Married at birth				-0.0464			
				[0.00320]			
Maternal age at birth				-0.00323			
				[0.000274]			
Birth order				0.0197			
				[0.00136]			
Observations	124,579	124,579	121,290	80,079	70,681	70,681	70,533
Years	1990-2004	1990-2004	1990-2004	1997-2004	1991-1999	1991-1999	1991-1999
R-squared	0.176	0.185	0.199	0.191	0.029	0.039	0.047
Level of neighborhood fixed effects:	Municipality (n=39)	Census Block Group (n=810)	Census Block Group & School (n=1208)	Census Block Group (n=810)	Municipality (n=39)	Census Block Group (n=810)	Census Block Group & School (n=1208)

Robust standard errors in brackets, clustered at the level of the Census tract. All models also include year of birth fixed effects.

Table 4: Heterogeneity in the Relationship Between Lead and School Suspensions and Incarcerations

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Any School Suspension	ALL	White	Black	Hispanic	Paid	Free Lunch
Geometric Mean of Lead	0.00588	0.00328	0.0109	0.0100	-0.000347	0.0102
	[0.000597]	[0.000746]	[0.00170]	[0.00139]	[0.000745]	[0.000785]
Lead*male	0.00555	0.00659	-0.00208	0.00322	0.00776	0.00103
	[0.000790]	[0.00102]	[0.00219]	[0.00182]	[0.00116]	[0.00103]
Observations	124,579	86,536	11,527	22,239	57,869	66,710
R-squared	0.185	0.165	0.218	0.178	0.089	0.155
mean of dep variable - male	0.250	0.200	0.440	0.360	0.120	0.360
mean of dep variable - female	0.140	0.100	0.270	0.230	0.050	0.120
mean of dep variable - all	0.200	0.150	0.360	0.290	0.090	0.290

	(1)	(2)	(3)	(4)	(5)	(6)
Panel B: Any Detention/Incarceration	ALL	White	Black	Hispanic	Paid	Free Lunch
Geometric Mean of Lead	-0.000468	-0.000292	0.000714	0.000103	-5.05e-05	-1.37e-05
	[0.000207]	[0.000169]	[0.000792]	[0.000510]	[0.000149]	[0.000279]
Lead*male	0.00355	0.00149	0.00389	0.00481	0.000626	0.00367
	[0.000376]	[0.000328]	[0.00135]	[0.00105]	[0.000249]	[0.000525]
Observations	76,646	54,722	6,602	12,700	37,040	39,606
R-squared	0.042	0.034	0.125	0.065	0.031	0.048
mean of dep variable - male	0.0176	0.009	0.0586	0.0332	0.0018	0.019
mean of dep variable - female	0.00173	0.0022	0.0098	0.0042	0.0007	0.0057
mean of dep variable - all	0.0107	0.0057	0.0348	0.0192	0.0028	0.0315

Robust standard errors in brackets, clustered on the Census tract. Additional controls include fixed effects for the block group, year of birth, and race and free lunch specific quadratic time trends. Panel B is limited to children born between 1991 and 1999; Panel A includes cohorts born between 1990 and 2004.

Table 5a: Using One Measure of Lead as an Instrument for Another for Full Sample

Panel A: Dependent Variable is Any Suspension	(1) FE	(2) FE	(3) IV-FE	(4) IV-FE	(5) FE	(6) FE	(7) FE-IV	(8) FE-IV	(9) FE-IV
Instrument			venous lead & venous lead*male	capillary lead & capillary lead*male			All other draws & all other draws*male	random draw & random draw*male	sibling lead level & sibling lead level*male
Average of all capillary tests	0.00380		0.0106						
	[0.000826]		[0.00148]						
Capilary lead*male	0.00185		0.00243						
	[0.00107]		[0.00165]						
Average of all venous tests		0.00674		0.0118					
		[0.00115]		[0.00206]					
Venous lead*male		0.00206		0.00449					
		[0.00151]		[0.00231]					
First random draw					0.00341		0.00887		
					[0.000520]		[0.000879]		
First random*male					0.00407		0.00641		
					[0.000695]		[0.000991]		
Avg all other tests						0.00486		0.00957	
						[0.000598]		[0.00104]	
Avg all other tests*male						0.00470		0.00751	
						[0.000797]		[0.00114]	
Geometric Mean of Lead									0.0124
									[0.00263]
Lead*male									0.0174
									[0.00290]
Observations	24,509	24,509	24,509	24,509	91,057	91,057	91,057	91,057	34,252
R-squared	0.200	0.200			0.190	0.191			
mean of dep variable - male	0.308	0.308	0.308	0.308	0.263	0.263	0.263	0.263	0.189
mean of dep variable - female	0.181	0.181	0.181	0.181	0.15	0.15	0.15	0.15	0.098
mean of dep variable - all	0.248	0.248	0.248	0.248	0.209	0.209	0.209	0.209	0.144

Panel B: Dependent Variable is Any Incarceration	(1) FE	(2) FE	(3) IV-FE	(4) IV-FE	(5) FE	(6) FE	(7) FE-IV	(8) FE-IV
Average of all capillary tests	-0.000673		-2.99e-05					
	[0.000205]		[0.000515]					
Capilary lead*male	0.00236		0.00438					
	[0.000438]		[0.000591]					
Average of all venous tests		-0.000370		-0.00128				
		[0.000382]		[0.000718]				
Venous lead*male		0.00356		0.00579				
		[0.000708]		[0.000830]				
First random draw					-0.000423		-0.000557	
					[0.000149]		[0.000317]	
First random*male					0.00287		0.00488	
					[0.000352]		[0.000367]	
Avg all other tests						-0.000548		-0.000306
						[0.000195]		[0.000381]
Avg all other tests*male						0.00345		0.00558
						[0.000389]		[0.000431]
Observations	16,039	16,039	16,039	16,039	50,774	50,774	50,774	50,774
R-Squared	0.077	0.079			0.047	0.047		
mean of dep variable - male	0.024	0.024	0.024	0.024	0.0204	0.0204	0.0204	0.0204
mean of dep variable - female	0.0036	0.0036	0.0036	0.0036	0.0034	0.0034	0.0034	0.0034
mean of dep variable - all	0.014	0.014	0.014	0.014	0.0122	0.0122	0.0122	0.0122

All controls from table 3 column 2 included in all regressions. Samples for column 1-4 are limited to children for whom we have both capillary and venous tes For columns 5-8, the sample includes children with two or more lead tests. For column 9, panel A, the sample is limited to those children with at least one sibling with lead levels. Robust standard errors are clustered at the Census tract level and appear in brackets.

Table 5b: Using One Measure of Lead as an Instrument for Another, Free Lunch Sample Only

Panel A: Dependent Variable is Any Suspension	(1) FE	(2) FE	(3) IV-FE	(4) IV-FE	(5) FE	(6) FE	(7) FE-IV	(8) FE-IV	(9) FE-IV
Instrument			venous lead & venous lead*male	capillary lead & capillary lead*male			All other draws & all other draws*male	random draw & random draw*male	sibling lead level & sibling lead level*male
Average of all capillary tests	0.00570		0.0136						
	[0.00107]		[0.00187]						
Capilary lead*male	0.000263		-0.000354						
	[0.00137]		[0.00217]						
Average of all venous tests		0.00937		0.0153					
		[0.00147]		[0.00254]					
Venous lead*male		-0.000375		0.000671					
		[0.00190]		[0.00297]					
First random draw					0.00570		0.0135		
					[0.000665]		[0.00111]		
First random*male					0.00135		0.000747		
					[0.000877]		[0.00132]		
Avg all other tests						0.00871		0.0133	
						[0.000781]		[0.00133]	
Avg all other tests*male						0.000745		0.00249	
						[0.00103]		[0.00154]	
Geometric Mean of Lead									0.0245
									[0.0044]
Lead*male									0.0081
									[0.0054]
Observations	15,206	15,206	15,206	15,206	52,317	52,317	52,317	52,317	14,312
R-squared	0.178	0.179			0.160	0.161			
mean of dep variable - male	0.409	0.409	0.409	0.409	0.37	0.37	0.37	0.37	0.314
mean of dep variable - female	0.257	0.257	0.257	0.257	0.227	0.227	0.227	0.227	0.173
mean of dep variable - all	0.336	0.336	0.336	0.336	0.302	0.302	0.302	0.302	0.245

Panel B: Dependent Variable is Any Incarceration	(1) FE	(2) FE	(3) IV-FE	(4) IV-FE	(5) FE	(6) FE	(7) FE-IV	(8) FE-IV
Average of all capillary tests	-0.000639		0.000211					
	[0.000260]		[0.000719]					
Capilary lead*male	0.00249		0.00419					
	[0.000589]		[0.000865]					
Average of all venous tests		-0.000116		-0.00130				
		[0.000499]		[0.000994]				
Venous lead*male		0.00333		0.00582				
		[0.000908]		[0.00119]				
First random draw					-0.000256		-0.000112	
					[0.000182]		[0.000451]	
First random*male					0.00284		0.00484	
					[0.000440]		[0.000549]	
Avg all other tests						-0.000209		-9.58e-05
						[0.000264]		[0.000551]
Avg all other tests*male						0.00355		0.00550
						[0.000527]		[0.000657]
Observations	9,965	9,965	9,965	9,965	28,898	28,898	28,898	28,898
R-Squared	0.098	0.099			0.052	0.053		
mean of dep variable - male	0.0365	0.0365	0.0365	0.0365	0.033	0.033	0.033	0.033
mean of dep variable - female	0.0054	0.0054	0.0054	0.0054	0.0057	0.0057	0.0057	0.0057
mean of dep variable - all	0.0219	0.0219	0.0219	0.0219	0.0202	0.0202	0.0202	0.0202

All controls from table 3 column 2 included in all regressions. Samples for column 1-4 are limited to children for whom we have both capillary and venous test results.

For columns 5-8, the sample includes children with two or more lead tests. For column 9, panel A, the sample is limited to those children with at least one sibling with lead levels. Robust standard errors are clustered at the Census tract level and appear in brackets.

Table 6: First Stage Regressions		
	(1)	(2)
Dependent Variable	Lead	Lead*male
Traffic volume quartile 1 linear trend	-0.132	0.00805
	[0.0398]	[0.0302]
Traffic volume quartile 2 linear trend	-0.155	0.00967
	[0.0398]	[0.0302]
Traffic volume quartile 3 linear trend	-0.174	0.0127
	[0.0398]	[0.0302]
Traffic volume quartile 4 linear trend	-0.168	0.00783
	[0.0398]	[0.0302]
Traffic volume quartile 2	0.301	-0.0217
	[0.0620]	[0.0471]
Traffic volume quartile 3	0.571	-0.0539
	[0.0630]	[0.0478]
Traffic volume quartile 4	0.618	0.000117
	[0.0633]	[0.0481]
Predicted lead*male	0.0825	1.144
	[0.0125]	[0.00948]
African American	1.919	-0.117
	[0.182]	[0.138]
White	0.00932	0.0568
	[0.0427]	[0.0324]
Hispanic	-0.586	0.0853
	[0.0446]	[0.0339]
Sometimes free/reduced lunch	2.401	-0.171
	[0.104]	[0.0788]
Always free/reduced lunch	2.747	-0.240
	[0.105]	[0.0794]
Male	-0.126	-0.564
	[0.0510]	[0.0387]
Black*year of birth	-0.136	0.0221
	[0.0360]	[0.0273]
Black*year of birth squared	0.00124	-0.000846
	[0.00167]	[0.00127]
Paid lunch*year of birth	0.244	-0.0199
	[0.0209]	[0.0159]
Paid lunch*year of birth squared	-0.00647	0.000446
	[0.000979]	[0.000743]
Observations	124,579	124,579
R-squared	0.278	0.570

All controls from table 3 column 2 included in all regressions. Standard errors are clustered on the Census tract and appear in brackets. Predicted lead is the child's lead level as predicted from Appendix Table 2. The predicted lead measure is then interacted with male to serve as an instrument for lead*male.

Table 7: IV Estimates of Lead, Any Suspension, and Any Detention/Incarceration

Panel A: Full Sample	(1) OLS	(2) IV	(3) OLS	(4) IV
	Suspension	Suspension	Incarceration	Incarceration
Geometric Mean of Lead	0.00588	0.0130	-0.000468	0.00409
	[0.000550]	[0.0163]	[0.000207]	[0.00596]
Lead*male	0.00555	0.0144	0.00355	0.0131
	[0.000685]	[0.00198]	[0.000376]	[0.000757]
Sample Birth Cohorts	1990-2004	1990-2004	1991-1999	1991-1999
Observations	124,579	124,579	76,646	76,646
R-squared	0.128		0.042	
mean of dep variable - male	0.2500	0.2500	0.0176	0.0176
mean of dep variable - female	0.1400	0.1400	0.00173	0.00173
mean of dep variable - all	0.2000	0.2000	0.0107	0.0107
Panel B: Free Lunch Sample Only				
Geometric Mean of Lead	0.0102	0.0868	-0.000205	0.00807
	[0.000785]	[0.0264]	[0.000270]	[0.00907]
Lead*male	0.00103	-0.00523	0.00388	0.0180
	[0.00103]	[0.00333]	[0.000538]	[0.00150]
Sample Birth Cohorts	1990-2004	1990-2004	1991-1999	1991-1999
Observations	66,710	66,710	36,979	36,979
R-squared	0.155		0.049	
mean of dep variable - male	0.3600	0.3600	0.0315	0.0315
mean of dep variable - female	0.2200	0.2200	0.0053	0.0053
mean of dep variable - all	0.2900	0.2900	0.0188	0.0188

Standard errors are clustered at the Census tract level and shown in brackets. Regressions include al variables in Table 3 column 2.

Table 8: OLS & IV Estimates of Lead and Suspensions, Robustness Checl

Panel A: OLS Estimates	(1)	(2)	(3)	(4)
Geometric Mean of Lead	0.00588	0.00525	0.00585	0.00634
	[0.000597]	[0.000591]	[0.000598]	[0.000615]
Lead*male	0.00555	0.00553	0.00555	0.00511
	[0.000790]	[0.000782]	[0.000791]	[0.000814]
Average infractions per child in school and		0.0470		
		[0.00162]		
Certificates in tract per old housing unit			0.0269	
			[0.0381]	
Observations	124,579	124,578	124,386	114,552
R-squared	0.185	0.194	0.186	0.184
Additional Controls	None	d in School*grade	Certificates in Tract at Birth	None
Sample	Full	Full	Full	Drop tracts w/new roads

Panel B: IV Estimates	(1)	(2)	(3)	(4)	(5)
Geometric Mean of Lead	0.013	0.00720	0.0182	0.0123	0.0116
	[0.0163]	[0.0164]	[0.0217]	[0.0179]	[0.0156]
Lead*male	0.0144	0.0148	0.0141	0.0129	0.0145
	[0.00198]	[0.00198]	[0.00221]	[0.00212]	[0.00195]
Average infractions per child in school and		0.0461			
		[0.00248]			
Certificates in tract per old housing unit			0.143		
			[0.148]		
Observations	124,579	124,578	124,386	114,552	124,579
Additional Controls	None	Infractions/Chil d in	Certificates in Tract at Birth	None	Quadratic time trends
Sample	Full	Full	Full	Drop tracts w/new roads	Full

Robust standard errors clustered on the Census tract and shown in brackets.

All regressions include year of birth fixed effects, birth order fixed effects, 801 census block group fixed effects, race and free lunch specific quadratic time trends and the other variables listed in Table 3, column 2.

The average infractions per child in school and grade is based on the average number of infractions in the child's grade and school (excluding his or her own) when the child was in 6th grade. If 6th grade data are not available (because the child did not attend middle school in RI), we use the average for third grade. For children who only attended high school in RI, we use the 9th grade average.

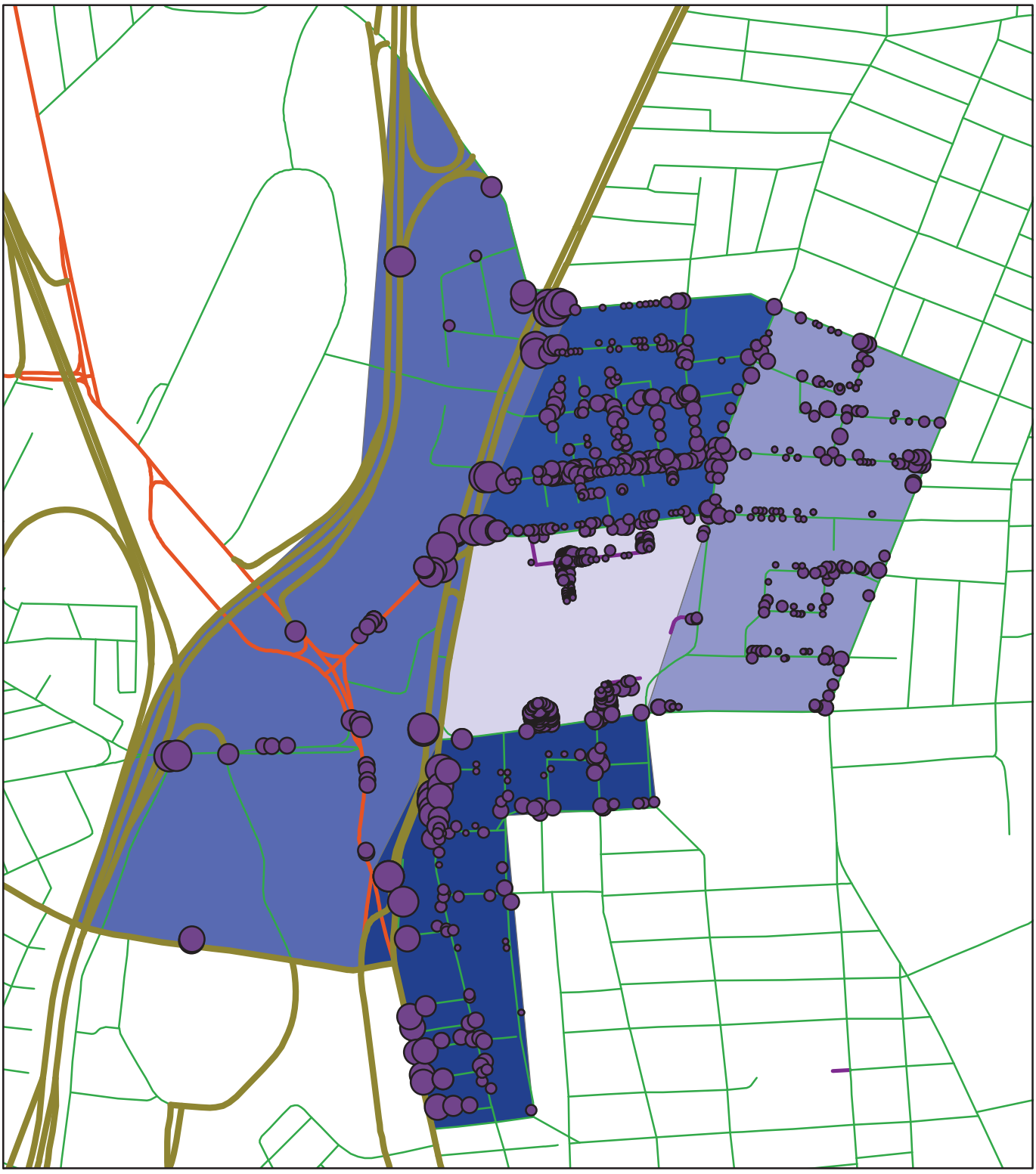
Table 9: Traffic Volume and Birth Weight

	(1)	(2)	(3)	(4)	(5)	(6)
outcome: birth weight in kg	Reduced Form	OLS	IV	Reduced Form	OLS	IV
Geometric Mean of Lead		-0.00292	0.0404		-0.00265	0.0717
		[0.000913]	[0.0597]		[0.00118]	[0.0674]
Lead*male					-0.000727	-0.00467
					[0.00156]	[0.00605]
Mother <HS		-0.0154	-0.0282		-0.0153	-0.0365
		[0.00827]	[0.0190]		[0.00754]	[0.0205]
Mother HS grad		0.0300	0.0305		0.0300	0.0310
		[0.00592]	[0.00551]		[0.00550]	[0.00566]
Mother College+		0.0676	0.0701		0.0676	0.0718
		[0.00676]	[0.00680]		[0.00589]	[0.00707]
Married at birth		0.0448	0.0533		0.0448	0.0589
		[0.00615]	[0.0129]		[0.00584]	[0.0138]
Maternal age		-0.00445	-0.00322		-0.00445	-0.00241
		[0.000599]	[0.00175]		[0.000520]	[0.00190]
Traffic volume quartile 1 linear trend	-0.00193			-0.00646		
	[0.00962]			[0.00948]		
Traffic volume quartile 2 linear trend	-0.00540			-0.00818		
	[0.00959]			[0.00948]		
Traffic volume quartile 3 linear trend	-0.00225			-0.00648		
	[0.00965]			[0.00945]		
Traffic volume quartile 4 linear trend	-0.00425			-0.00874		
	[0.00953]			[0.00947]		
Traffic quartile 1*linear trend*male				0.00188		
				[0.00335]		
Traffic quartile 2*linear trend*male				0.00135		
				[0.00340]		
Traffic quartile 3*linear trend*male				0.000455		
				[0.00337]		
Traffic quartile 4*linear trend *male				-0.000371		
				[0.00331]		
Observations	80,078	80,078	80,078	80,079	80,079	80,079
R-squared	0.028	0.028		0.047	0.047	
Number of bl	808	808	808	808	808	808

Robust standard errors clustered on the Census tract and shown in brackets.

All regressions include year of birth fixed effects, birth order fixed effects, 801 census block group fixed effects, race and free lunch specific quadratic time trends and the other variables listed in Table 3, column 2.

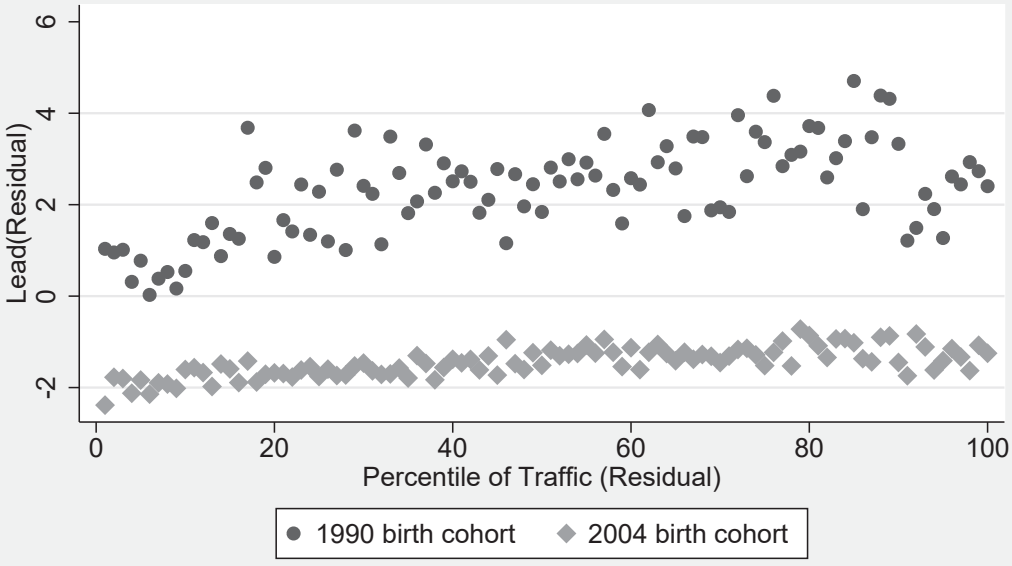
Figure 1: Traffic Volume within 50 meters



Note: each circle indicates a home. The size of the radius indicates the traffic volume within 50 meters of the home, with larger radii indicating greater traffic volume.

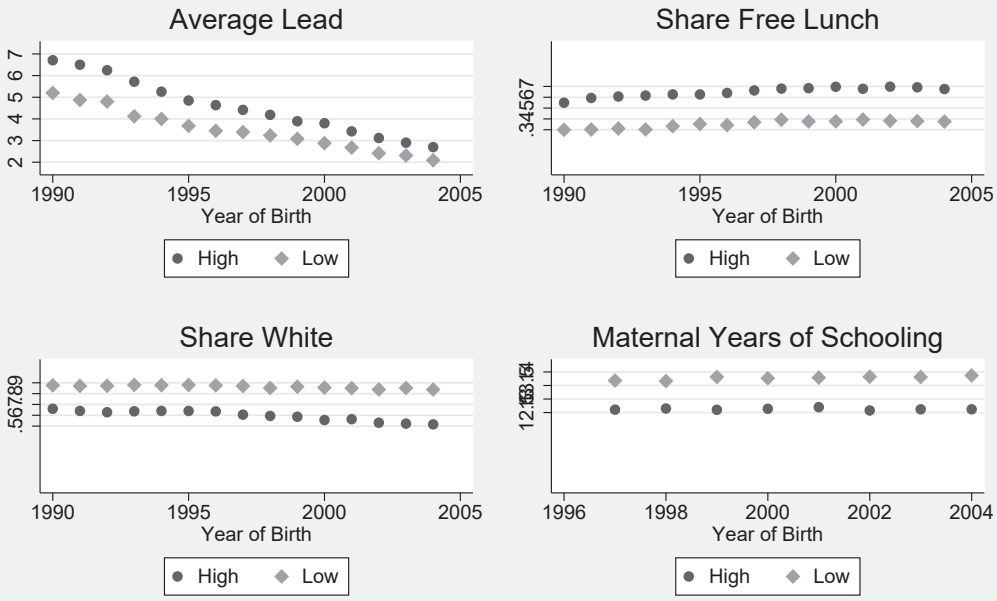
federal and interstate road
state road
town road
county road

Figure 2:
Child Lead Levels and Traffic(Residuals)
by Birth Cohort



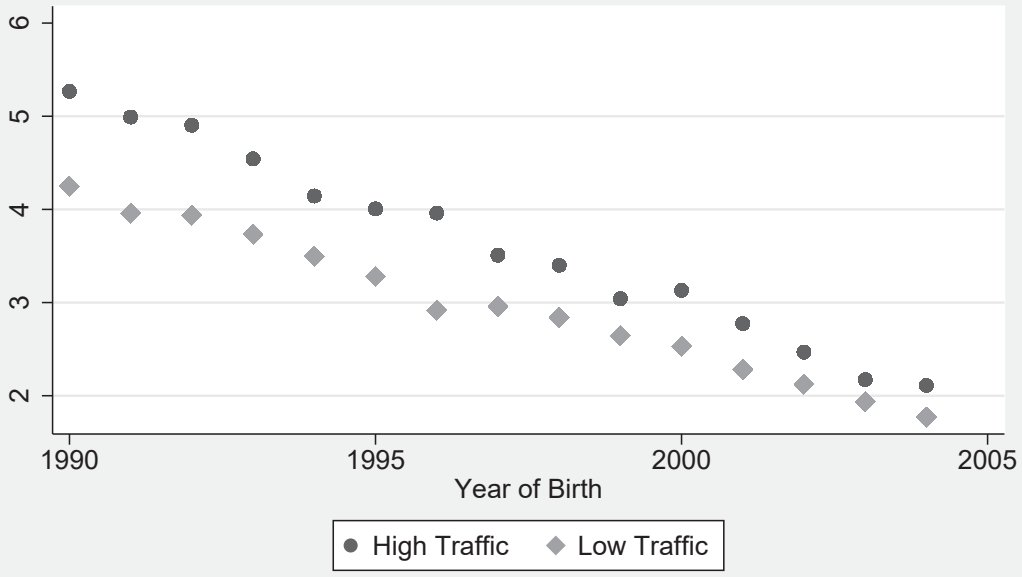
Residuals from regressions of lead and traffic on census block group FE

Figure 3: Trends in Child Characteristics by Traffic Density



High(low) traffic defined as top(bottom) quartile of traffic volume in the state.
Maternal Education only avilable for chidlren born post 1996.

Figure 4: Lead Levels for High SES Children



Child SES is measured using the predicted lead level of the child based on estimates of a regresion of lead on gender race ethnicity free lunch status and neigbhborhood fixed effects for children born 1990-1991 and applied to all chidlren.
Our proxy for high SES children is a predicted lead level in the bottom quartile.

Appendix – Construction of the measure of traffic volume

Steps in the construction of the traffic measure:

1) For each household, we measure the number of meters of road within 25 and 50 meters by functional class (primary, secondary and tertiary roads) and urban/rural status. Primary roads refer to highways, secondary roads to state routes, and tertiary roads to streets. In total, there are 6 road types: primary-urban, secondary-urban, tertiary-urban, primary –rural, secondary-rural, tertiary-rural. See below for a more detailed description of the road types. These data come from the RIGIS Roads-E-911 data. A description of the data and a link to the data can be found here: http://www.rigis.org/data/e911Roads. "These data contain street center lines and address ranges for all highways, roads, and streets for the entire state of Rhode Island. AK Associates conflated this data from the existing road data developed by MicroDATA GIS, RI DOT, 2008 Pictometry and data provided by Municipal Agencies for Rhode Island Enhanced 9-1-1 (RI E-911). Portions of this data set were collected as early as 2001."
2) Using data from the Bureau of Transportation Statistics on traffic patterns by road type for 1980 we then generate a measure of traffic volume by multiplying each meter of road by functional class and urban/rural status with measures of vehicles miles travelled per lane-mile by functional class and urban/rural status in 1980.[1]
3) Finally, we sum up the traffic measures generated in step 2. Roads within 25 meters are given full weight, roads between 25 and 50 meters are given ¼ weight to reflect the highly localized nature of lead contamination.
4) For each child, we may have multiple addresses and therefore multiple measures of traffic exposure. Since we are estimating the impact of an average measure of lead on outcomes, we calculate an average measure of traffic exposure over the preschool years and use that measure in our analysis.

Description of road types: Primary roads are generally divided limited-access highways within the interstate highway system or under State management, and are distinguished by the presence of interchanges. These highways are accessible by ramps and may include some toll highways. Secondary roads are main arteries, usually in the U.S. Highway, State Highway, and/or County Highway system. These roads have one or more lanes of traffic in each direction, may or may not be divided, and usually have at-grade intersections with many other roads and driveways. They usually have both a local name and a route number. Tertiary roads consist of paved non-arterial streets, roads, or byways that usually have a single lane of traffic in each direction.

[1]These data are found here: http://www.rita.dot.gov/bts/sites/rita.dot.gov.bts/files/publications/national_transportation_statistics/html/table_01_36.html

Appendix Table 1: Negative Selection into Elevated Lead and Omitted Variable Bias

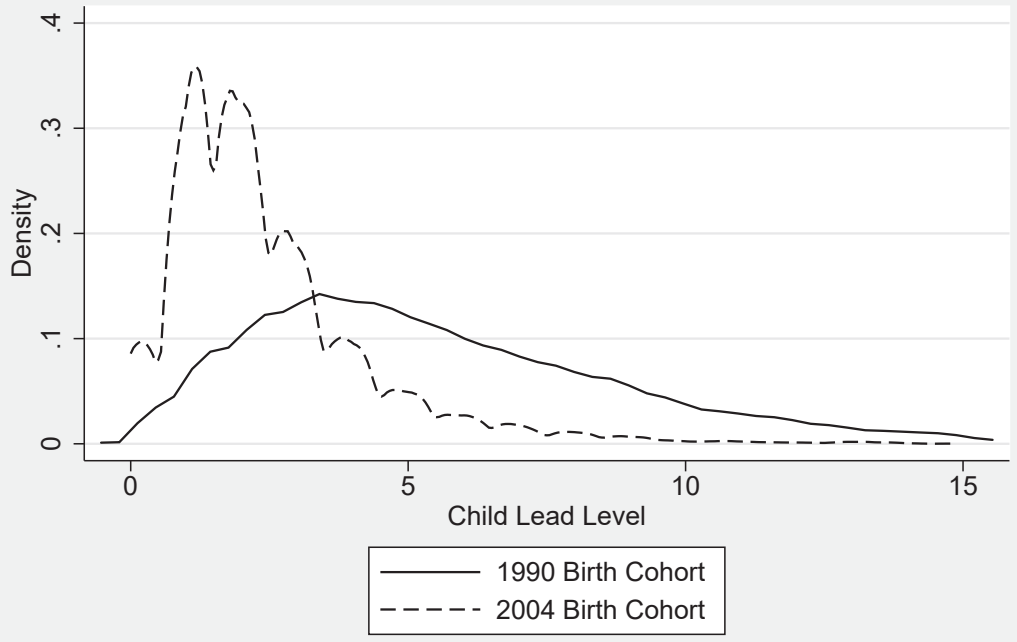
	(1)	(2)	(3)
Outcome:	Lead	9th Grade Infractions	
Geometric Mean of Lead		0.0253	0.0170
		[0.00595]	[0.00625]
Lead*year of birth		0.000121	0.00237
		[0.00152]	[0.00160]
African American	1.843	0.304	0.554
	[0.237]	[0.0537]	[0.156]
Sometimes free/reduced lunch	0.955	0.499	0.620
	[0.0696]	[0.0211]	[0.0463]
Always free/reduced lunch	1.548	0.647	0.793
	[0.0805]	[0.0261]	[0.0542]
Black*year of birth	-0.117		-0.0283
	[0.0254]		[0.0168]
Sometimes free lunch*year of birth	-0.0969		-0.0329
	[0.0165]		[0.0110]
Always free lunch*year of birth	-0.141		-0.0389
	[0.0186]		[0.0126]
Observations	49,374	49,374	49,374
R-squared	0.234	0.067	0.067

Sample restricted to chidlren born 1993-1998. These children all have 9th grade infractions data.
Also included are year of birth (linear) and neighborhood FE.

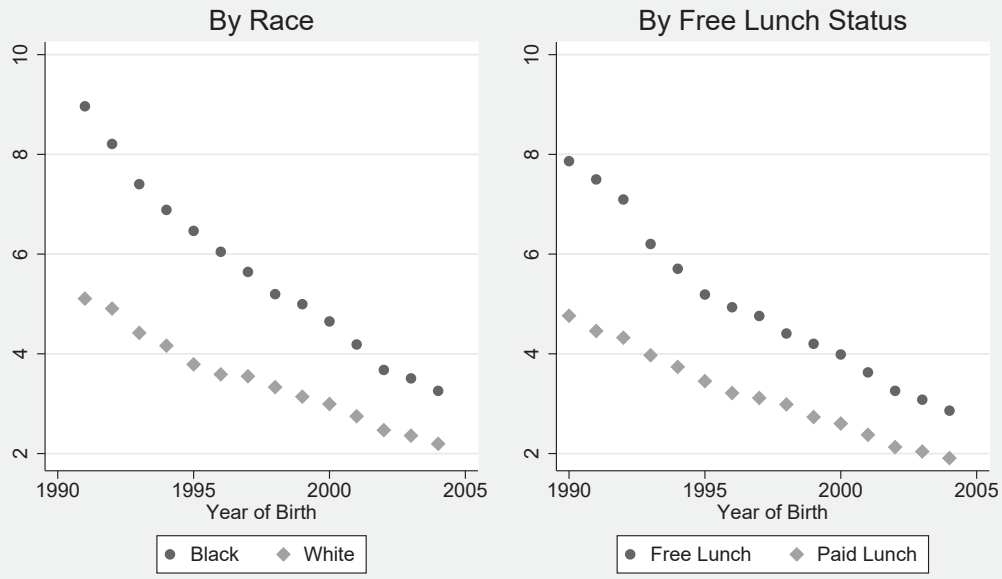
Appendix Table 2: Traffic Exposure and Child Blood Lead Levels	
	(1)
	All
Traffic volume quartile 1 linear trend	-0.137
	[0.0319]
Traffic volume quartile 2 linear trend	-0.161
	[0.0319]
Traffic volume quartile 3 linear trend	-0.181
	[0.0320]
Traffic volume quartile 4 linear trend	-0.175
	[0.0318]
Traffic volume quartile 2	0.312
	[0.0807]
Traffic volume quartile 3	0.594
	[0.0895]
Traffic volume quartile 4	0.646
	[0.0863]
Male	0.197
	[0.0157]
Observations	124,579
R-squared	0.278

Robust standard errors clustered on Census tract and shown in brackets.
All regressions include full set of controls included in the second column of Table 3, including Census tract fixed effects, year fixed effects and quadratic trends in race and free lunch.

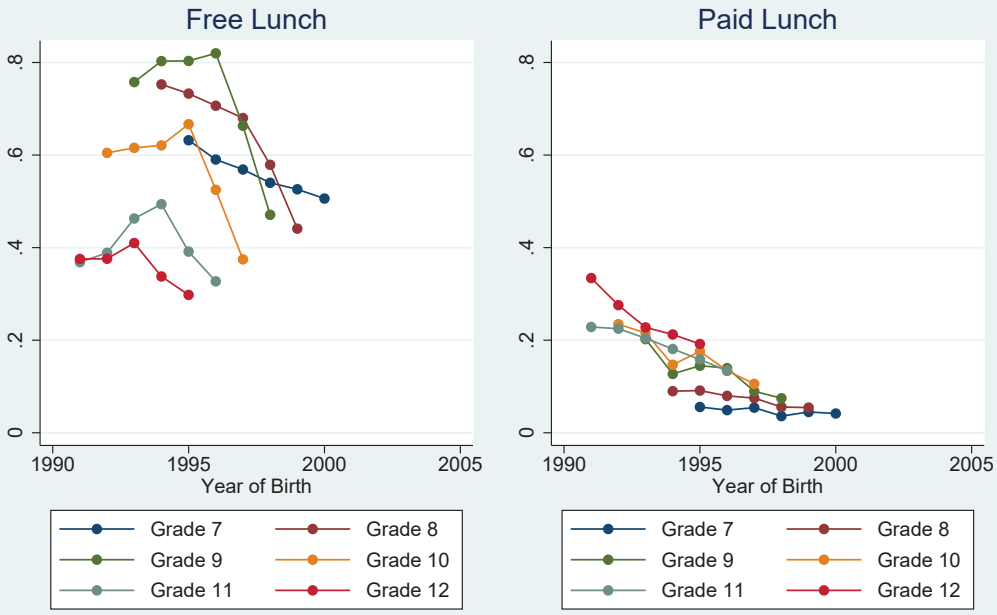
Appendix Figure 1: Child Lead Levels by Birth Cohort



Appendix Figure 2:
Trends in Lead Levels
by Race & Free Lunch Status



Appendix 3A: Number of Infractions Over Time by Free Lunch Status



Appendix Figure 3B: Number of Infractions Over Time by Race