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The Long-run Spillover Effects of Pollution: How Exposure to Lead Affects Everyone in the Classroom*

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Abstract
Children exposed to pollutants like lead have lower achievement in school and are more likely to engage in risky behavior. However, little is known about whether lead-exposed children affect the long-run outcomes of their peers. We estimate these spillover effects using unique data on preschool blood lead levels (BLLs) matched to education data for all students in North Carolina public schools. We compare siblings whose school-grade cohorts differ in the proportion of children with elevated BLLs, holding constant school and peers' demographics. Having more lead-exposed peers is associated with lower high-school graduation and SAT-taking rates and increased suspensions and absences.

Keywords: Lead Poisoning, Spillovers, Peer Effects, Human Capital

JEL Codes: Q52, I14, I24

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I. Introduction

Commonly encountered pollution sources, such as lead paint, highways, and toxic sites have been shown to affect children's academic achievement and behavior (Heissel, Persico and Simon 2021; Persico and Venator 2021; Persico, Figlio, and Roth 2020). So far, researchers have focused on estimating the effects of pollution on directly exposed children. However, pollution-exposed children interact daily with peers. Because children exposed to pollution have lower achievement and engage in risky behavior, the effects of pollution might spill over to affect everyone in the classroom. Yet, few papers credibly document the long-run impacts of childhood peers generally, and no existing studies explore the spillover effects of pollution onto peers in school. Showing that pollution exposure has spillover effects is important because it reveals the scope of the problems pollution causes. If one child's exposure to pollution causes negative long-run spillover effects onto his peers, this increases the true costs of pollution and changes our understanding of how pollution might affect long-run human capital attainment.

In this paper, we focus on one type of pollution: lead poisoning. A growing literature shows that children who are lead-poisoned in early life are also more likely to be suspended and commit crimes, and have worse academic achievement and long-run outcomes.[1] A recent UNICEF report reveals lead poisoning is a global issue: as many as 800 million children, or around one in three, have blood lead levels (BLLs) at or above 5 micrograms per deciliter (μg/dL), the reference value most commonly used to identify children who have elevated BLLs during our sample period.[2] At this threshold, at least 500,000 young children are estimated to

[1] For recent evidence on the direct effects of lead poisoning on children's outcomes, see Aizer and Currie (2019), Reyes (2015), Gazze (2016), Ferrie, Rolf, and Troesken (2012), Feigenbaum and Mueller (2016), Aizer et al. (2018), Persico, Figlio, and Roth (2020), Grönqvist, Nilsson, and Robling (2020), and Hollingsworth et al. (2020).

[2] In this paper, we use the words lead poisoning and lead exposure interchangeably.

be poisoned by lead each year in the US (Aizer et al. 2018), generating $200 billion per cohort in societal costs (Reyes 2014), including reduced tax revenues and increased expenditure on special education, crime prevention, and health care. Low-income and Black children are more likely to have elevated BLLs compared to higher-income and White children (CDC 2005).

Because pollution sources like lead paint or highways are very common, particularly in low-income neighborhoods, spillovers from pollution exposure imply that most children and public schools in the US suffer from both the direct and spillover effects of pollution exposure. Our data indicate that in North Carolina public schools between 2000 and 2017, 98.9 percent of middle school students without known lead exposure had at least one lead-poisoned child in their school cohort, 79.9 percent were in a school cohort with at least 5 percent lead-poisoned peers and 52.5 percent were in a school cohort with at least 10 percent lead-poisoned peers.[3] Thus, the spillover effects of lead exposure are a heretofore unexplored mechanism through which social context, pollution, and built environment could affect schools and children's outcomes.

Using unique data linking children's BLLs by age six to the universe of public-school records in North Carolina, we are the first to investigate the negative long-run spillover effects of lead poisoning on children who are not directly exposed to lead but are exposed to lead-poisoned school peers. We identify the spillover effects of lead-exposed peers by comparing siblings whose cohorts happen to randomly differ in the proportion of children with high preschool BLLs in their grade-cohort. Our preferred specification includes family, school, grade, birth month, birth order, and year fixed effects, and controls for a broad set of time

[3] National Childhood Blood Lead Surveillance Data from the Centers for Disease Control and Prevention suggest that lead exposure might be even more pervasive in the rest of the US. While the testing rates in North Carolina between 2012 and 2017 were similar to the national average, the percent of NC children with BLLs above 5ug/dL was 0.4-0.7% compared to 2-3% in the US overall.

varying child and cohort demographic characteristics, as well as school quality. Including family fixed effects controls for unobserved family characteristics that could be correlated with both peers' quality and a child's outcomes, such as parental traits. Controlling for peers' race and socioeconomic status suggests that our estimated effects are due to lead poisoning and not peer demographics.

We find that a ten percent increase in the share of cohort peers exposed to lead is associated with a 1.7 percentage point decrease in the likelihood that a child graduates high school, a 2 percent decrease in the graduation rate. Having more lead-exposed cohort peers is also associated with a higher likelihood of suspension from school, chronic absenteeism, dropping out of school, and a decrease in the likelihood of taking the SAT. A back-of-the-envelope calculation suggests that the lost earnings of classmates of lead-poisoned children not graduating high school amount to $9.2 billion per cohort. Lead-exposed peers disproportionally affect the outcomes of Black students, suggesting that the spillover effects of pollution could be contributing to persistent inequality in human capital accumulation. These findings are generally robust to different specifications that account for potential selection, omitted variables, and measurement error biases.

To explore mechanisms, we find that exposure to lead-poisoned peers in middle school, rather than elementary school, appears to drive long-run outcomes. We also show that students who attend school with a higher share of lead-poisoned peers are more likely to be suspended and more likely to be involved in behavioral incidents with these lead-poisoned peers. We interpret our results as suggestive that noncognitive skill development might drive the spillover effects of lead poisoning through peers' influence to engage in similar disruptive behavior.

This paper makes three main contributions. First, this is the first study to investigate the spillover effects of lead exposure on peers' academic achievement, behavior, and long-run outcomes. Furthermore, our findings have implications for

more than just lead: our estimates imply that the true costs of pollution are likely higher than the direct costs alone, especially for pollutants that affect behavior.

Second, this is among the first studies to examine the long-run impacts of peers who are disruptive (in this case due to early childhood exposure to pollution), as well as the channels through which these effects manifest. Current evidence on the long-run effects of peers is mixed. While Carrell, Hoekstra, and Kuka (2018) show that having peers exposed to domestic violence lowers wages and educational attainment, Bietenbeck (2020) finds positive long-run effects from peers who repeat kindergarten.[4] We show that exposure to lead-poisoned peers can have long-term consequences, including dropping out of high school.

Several mechanisms could link peer composition and student outcomes, including differential curricular offerings and instructional practices depending on average ability (Jackson 2013); social dynamics in a student's reference group (Hoxby 2000; Brenøe and Zölitz 2020); and low performing students not keeping up with higher-achieving peers (Imberman, Kugler, and Sacerdote 2012). Peers might also draw disproportionately on a teacher's time and influence class culture and standards. We find suggestive evidence that exposure to disruptive peers in middle school might drive some of these effects through the development of noncognitive skills. In particular, exposure to lead-poisoned peers increases suspensions and chronic absenteeism, which suggests that noncognitive skills are an important mechanism through which disruptive peers affect long-run outcomes.[5]

[4] Other papers find that peers' parental education and peers' receipt of conditional cash transfers increases college attendance, while peers with special needs lower it (Bifulco, Fletcher, and Ross 2011; Bobonis and Finan 2009; Balestra, Eugster, and Liebert 2020). Evidence on the relationship between peers' gender and long-run outcomes is more mixed (Black, Devereux, and Salvanes 2013; Anelli and Peri 2019).

[5] These findings corroborate those in Carrell, Hoekstra and Kuka (2018). Indeed, the literature on short-term peer effects is more robust than the literature on long-run peer effects. (Carrell and Hoekstra 2010; Figlio 2007; Hoxby 2000; Lazear 2001; Sacerdote 2001; Fletcher 2010). See Epple and Romano (2011) and Sacerdote (2011) for overviews of this literature.

Third, we contribute to a growing literature that document neighborhood effects on health, education, and behavior outcomes, but which is largely silent on the mechanisms behind these effects (Chyn and Katz 2021). Our findings on the long-run effects of exposure to lead-poisoned children suggest that environmental factors might contribute to the persistent effects of high-poverty and high-pollution neighborhoods in the US (Chetty, Hendren, and Katz 2016).[6]

II. Background: Lead Exposure

Ingestion or inhalation of lead causes lead poisoning, which can induce widespread brain damage (Meyer, McGeehin, and Falk 2003; CDC 2022).[7] Small children are especially exposed to lead-contaminated soil and dust from paint due to normal hand-to-mouth activity. Moreover, lead is most damaging to small children: they absorb and retain more lead than adults and their neurological development is particularly susceptible to neurotoxins (CDC 2022). Lead exposure has been associated with problems in cognition, executive functioning, abnormal social behavior (including aggression), and fine motor control (Cecil et al. 2008).

Federal guidelines mandate that all children on Medicaid are screened for lead poisoning at ages one and two. In addition, North Carolina mandates universal screening in some zip codes based on estimated lead exposure risk. We use these universal testing zip codes, where testing is much higher (about 60% of children living there are tested), in a robustness check later in the paper. Consistent with guidelines, most children in our sample who are ever tested are first tested by 13 months of age, and 75 percent are tested by 25 months of age. Usually, testing is done preventatively, and not in response to symptoms. Indeed, lead poisoning can

[6] Besides a higher likelihood of lead poisoning, low-income children are more likely to live near sources of toxic waste (Banzhaf, Ma, and Timmins 2019) and have higher asthma rates (Alexander and Currie 2017).

[7] Specifically, lead causes the axons of nerve cells to degenerate and lose their myelin coats (Brubaker et al 2009; Naffaa, Laprevote, and Schang 2021).

be difficult to detect initially because most lead poisoning is asymptomatic and does not cause distinctive symptoms in early life (CDC 2014) until dangerous amounts of lead have accumulated, which is rare (CDC, 2021; Mayo Clinic 2022).

During our study period, children with two consecutive BLL tests measuring 10 µg/dL or more were eligible for an intervention that included education for caregivers on nutrition and reducing exposure in the home, a home inspection, and a referral to lead remediation services. We use the more recent CDC reference value of 5 µg/dL to define lead poisoning based on current scientific understanding. To the extent that these interventions made students with BLLs at or above 10 µg/dL less disruptive for their peers, our estimates represent a lower bound of the spillover effects of lead poisoning, and we test the robustness of our estimates to different thresholds defining lead poisoning below.

III. Data Description

Education Data

We use population-level data from 1997-2017 on every child attending public school in North Carolina, including charter schools, linked to the universe of blood lead test records from 1992-2016. These unique data include home address identifiers that enable us to match siblings. To our knowledge, this is the first state-level data set linking individual BLLs to schooling records that allow the matching of siblings and locating students in classrooms. It also tracks both short- and long-run outcomes over 20 years for the same students in a large state, which allows us to investigate the long-run spillover effects of pollution for the first time.[8]

While we use the entire sample to calculate the number of children per school-grade-year cohort who have elevated BLLs (as well as all our cohort controls), for our main analysis we drop children who do not have siblings, as well

[8] Previous data sets from Rhode Island only included contemporaneous outcomes (Aizer et al. 2018; Aizer and Currie 2019).

as children who live in large buildings, since we cannot reliably identify families in those buildings. Our main analysis also drops students who themselves have an elevated BLL and estimates the spillover effects of lead exposure on children without known lead poisoning. In Section V, we perform extensive robustness tests using different samples and specifications. The Data Appendix provides more information on the linkage performed by the North Carolina Education Research Data Center, our sibling identification algorithm, and variable construction.

For our long-run outcomes, we use indicators for high school graduation, dropping out,[9] community and four-year college intentions in 12th grade, and whether the student took the SAT in high school from 2005-2017 (the period over which we can match preschool BLLs of elementary and middle school peers to long-run outcomes). For our contemporaneous outcomes, we use the average of standardized mathematics and reading end-of-grade (EOG) test scores administered in grades 3-8, indicators for being absent for more than 21 days,[10] and having at least one out-of-school (OOS) suspension,[11] as well as the number of days the child was suspended out-of-school each year in grades 6-12. We also construct indicators for being suspended on the same day and for being involved in a behavioral incident with a lead-exposed cohort peer. Because exams changed multiple times over the sample period, we limit our analysis to exams taken between 1996-1997 and 2004-2005, which were administered to all children and had a similar structure.[12]

We construct various individual, cohort, and time-varying school covariates. Individual-level covariates include indicators for gender, race, being economically disadvantaged in a year, having a blood lead level test, birth month,

[9] Dropping out of school is distinct from school switching, death, moving, promotion, graduation, and other confounding factors, and specific reason codes are given for dropping out.
[10] Absences are grouped into 0-7, 8-14, 15-21, and more than 21 days, that is chronic absenteeism.
[11] We focus on OOS suspensions because the reporting requirements for these did not change during the sample period, while in-school suspension reporting became more stringent over time.
[12] During our sample period, the scale for the math EOG exam changed in 2001-2002. The reading EOG exam scale changed in 2002-2003.

and birth order. Our cohort level covariates include the share of cohort peers that are non-white, economically disadvantaged, and tested for lead. The school-year covariates include the share of teachers with a Master's degree, school size, and the stability rate which is defined as the percentage of students from the October membership count who are still present in the second semester (90 days later).

Blood Lead Levels Data

We obtained the universe of individual blood lead test records for children up to age six from the North Carolina Department of Health and Human Services for the years 1992-2016. Test records include the date of blood draw, test result in μg/dL, and the child's identifier. We define a child as having an elevated BLL (EBLL) if their highest BLL is ≥5 μg/dL, the upper reference interval value per the 2012 guidelines by the Centers of Disease Control and Prevention (CDC 2013).[13]

Because childhood lead screening is targeted at high-risk children and neighborhoods in North Carolina, we expect screening to be higher among low-income children (who also must be screened under the Medicaid mandate).[14] We construct indicators for children missing blood lead tests and include these children in our analysis. We compute the share of a child's peers with EBLLs using all children in the cohort or classroom as the denominator, independently of whether they have a blood lead test. Figure 1 plots the share of children with blood tests and the share of children with EBLLs by birth cohort in our sample, showing that as lead screening increases over time, the incidence of lead poisoning decreases. Still, our identifying variation is not likely to be driven by differences in outcomes

[13] This value is the 97.5th percentile of BLLs in U.S. children aged 1–5 years from the combined 2007–2008 and 2009–2010 cycles of the National Health and Nutrition Examination Survey. Starting in 1991 and prior to 2012, CDC defined BLLs ≥10 μg/dL as the "level of concern" for children aged 1–5 years. In robustness checks, we vary the definition of elevated BLL.

[14] According to the NC Department of Health and Human Services "NC Childhood Lead Testing and Follow-up Manual", blood lead testing is required for children participating in Medicaid, Health Choice and/or the Special Nutrition Program for Women, Infants and Children (WIC).

between older and younger siblings, as Figure A1 shows that first-born children have only 1.5 percentage points more lead-exposed peers than their younger siblings. Since earlier-born siblings typically have better outcomes (see, e.g., Black et al., 2005; Conley and Glauber, 2006; Price, 2008; and Booth and Kee, 2009), these birth order effects would go in opposite direction to the secular trends depicted in Figure 1. Moreover, we generally control for birth order fixed effects.

Sample Description

Since our blood lead level data begin in 1992 and include children tested up to age six, we restrict our sample to children born after 1986. Table 1 presents summary statistics for the sample of all children attending public schools in North Carolina (3.3 million children, Column 1) and our analysis sample of siblings (1.3 million children, Column 2). 39.6 percent of children in our analysis sample have a blood lead test, and 10.9 percent have at least one test greater or equal than 5 µg/dL, slightly higher shares than in the full sample in Column 1. Overall, children with siblings are fairly similar to the full sample, and our results are very similar when we include all children in a model using school-grade and grade-year fixed effects, which lends support to the external validity of our results.

As expected based on screening guidelines, children with blood lead tests are more likely to be Black, be economically disadvantaged (ED) as measured by an indicator for having ever received free or reduced-price lunch, and have teachers without Master's degrees (Column 3), as are children with EBLLs (Columns 4 and 5). Consistent with findings in the literature, early childhood lead exposure is also strongly associated with worse outcomes in our sample (Figure 2). The average cohort in our sample includes 225 children. Children who spend at least one elementary school year in a cohort with above median share of lead-exposed children (or >10.1 percent of cohort peers) have worse outcomes, are more likely to be Black, be ED, and have a blood lead test themselves (Columns 6 and 7). Our

identification strategy controls for family background with family fixed effects, assuaging concerns of omitted variable bias due to these differences.

IV. Identification Strategy

There are several challenges to identifying peer effects. First, because peers influence each other simultaneously, it could be *a priori* unclear whether a disruptive lead-exposed child causes their classmates to misbehave, or vice versa. This is called the reflection problem (Manski 1993). Using a measure of lead poisoning taken prior to school entry avoids the reflection problem because a child cannot affect the BLLs of their peers, but lead poisoning affects children negatively, which in turn could affect peers.

Second, peer groups are not randomly assigned; they are selected based in part on unobserved characteristics (Angrist 2014). For example, attentive parents might remove their children from classrooms with more disruptive peers. Because of this self-selection into groups, it is challenging to determine whether the outcome is a causal effect of the peers or the reason the individuals joined the peer group. Our preferred specification addresses this issue with a family and school fixed effects design that holds constant students' family and neighborhood background, and we test for endogenous moves in response to peers' composition. Moreover, over most of our study period there were relatively few options for choosing public schools.[15] Thus, as we will show, selection into schools was minimal.

Third, unobserved factors might simultaneously cause students and their peers to perform poorly. For example, a child's lead exposure could be correlated with socioeconomic status, which in turn has been associated with peers' learning

[15] North Carolina had no statewide voucher program (until quite recently) and relatively few charter schools, which accept students independently of catchment areas (and whose students we observe). Thus, the only way to attend a different school than the one assigned by catchment zone in most places was by moving or attending, and fully paying for, private school. Only 5.3 percent of all North Carolina children attended private school over this time period (NC DPI 2020).

disruptions (Hoxby and Weingarth 2006; Hoxby 2000). Thus, to causally identify the spillover effect of a child's lead exposure on their peers we control for the share of cohort peers who are non-White or economically disadvantaged. We also control for the share of the student's peers who have been tested for lead exposure. Because screening rates are higher among low-socioeconomic status students, additionally controlling for screening rates mitigates concerns about selection into testing.

We first examine how lead exposure affects long-run outcomes, that is graduation from high school, dropout, 4- and 2-year college intentions, and SAT taking of peers without known EBLLs. We compare students who attend the same school but whose grade cohorts randomly happen to have different proportions of children with EBLLs. This specification closely follows the one used by Carrell, Hoekstra, and Kuka (2018) and includes school-by-grade and grade-by-year fixed effects. The school-by-grade fixed effects control for unobservable characteristics of students who attend the same school and grade. Grade-year fixed effects account for common shocks to a cohort. This estimating equation is as follows:

$$(1) \quad Y_{isgt} = \beta_1 \frac{\overline{\sum k \neq \iota \, PeersEBLLs_{ksgt}}}{n_{sgt}-1} + \pi X_i + \eta_{sg} + \phi_{gt} + \varepsilon_{isgt}$$

where Y_{isgt} is some outcome for child *i* who either has not been screened for lead exposure or has always tested below 5 µg/dL, attending school *s*, in grade *g* and in year *t*. $\frac{\overline{\sum k \neq \iota \, PeersEBLLs_{ksgt}}}{n_{sgt}-1}$ is the average share of the student's peers with *known* EBLLs across elementary and middle school cohorts not including the student themselves. The coefficient β_1 captures the effect of having 100 percent of peers with *known* EBLLs in elementary and middle school. X_i includes gender, race, birth month fixed effects, economically disadvantaged status, an indicator for whether a child was tested for lead, the average share of non-White peers across years, the average share of economically disadvantaged peers, the average share of peers tested for lead, as well as the average school size, school stability rate and share of

teachers with Master's degrees over elementary and middle school. For each student, we use grade *g* and year *t* from the most recent (that is the last) observation we have for that student, to maximize sample size. η_{sg} is a school-by-grade fixed effect to account for school-by-grade-specific shocks. ϕ_{gt} is a grade-by-year fixed effect to account for secular cohort-level trends. We cluster standard errors at the school level to account for arbitrary correlation in the error terms.

However, this specification does not account for the fact that families might select into schools. Thus, in our preferred specification, we compare *siblings* whose grade cohorts randomly happen to have different proportions of children with EBLLs. Including family fixed effects mitigates the selection problem by controlling for unobserved family characteristics that could be correlated with both peer quality and child's outcomes. Including school fixed effects further controls for students' characteristics that are common to the school's catchment area. Remaining idiosyncratic variation in the BLLs of siblings' cohorts offers plausibly exogenous variation to identify the spillover effects of lead and the effects of peer quality more broadly.[16] Our main estimation equation is thus given by:

$$(2)\ Y_{ijsgt} = \beta_1 \frac{\overline{\sum k \neq \iota\ PeersEBLLs_{ksgt}}}{n_{sgt}-1} + \pi X_i + \theta_j + \delta_s + \tau_g + \sigma_t + \varepsilon_{ijsgt}$$

which is identical to equation (1) except for the fact that we substitute the school-by-grade (η_{sg}) and grade-by-year (ϕ_{gt}) fixed effects with family (θ_j), grade (τ_g), school (δ_s), and year (σ_t) fixed effects, and include birth order fixed effects in X_i.

[16] 97.9 percent of sibling groups in our sample present variation in the share of lead-poisoned peers, suggesting that selection into treatment might not be a concern in our sample (Miller, Shenhav, and Grosz 2019). Moreover, schools in our siblings sample have on average 53 percent of students who go to a different school as their siblings, meaning a majority of students in the sample contribute to estimating the school fixed effects. Figure A2 shows the distribution of our regressor of interest, the average share of a student's peers with EBLLs over elementary and middle school, as well as the distributions of residuals obtained from regressions of this variable on our preferred set of controls and the fixed effects in equations (1) and (2).

To identify the effect of lead-exposed peers on student *i*'s outcomes in this specification, four conditions must hold. First, conditional on our controls, the share of lead-exposed peers in a school-grade-year must not be correlated with other student *i*'s characteristics that could affect student *i*'s outcomes. Second, school characteristics and common shocks at the school-cohort level that affect student *i*'s outcomes must not correlate with the share of cohort peers who are lead-exposed. Third, peers' lead poisoning must be uncorrelated with other characteristics of these lead-exposed peers that could affect student *i*'s outcomes (except for through lead poisoning), like socioeconomic status. Fourth, conditional on our controls, testing for lead must be random so that there is no selection into testing.

Our preferred specification includes family and school fixed effects, as well as student-specific school and cohort characteristics that address many concerns about identification related to these first three conditions. In addition, we perform a battery of tests to address remaining potential violations of these conditions in Sections VD and VE. In Section VD we also show that the fourth identifying assumption holds: conditional on our controls, testing for lead does not appear to be associated with any observable characteristics of children.

V. Results

A. Long-run Effects of Peers Exposed to Lead

Figure 3 shows that the share of a child's peers with EBLLs is negatively correlated with the child's contemporaneous and long-run outcomes. We next provide evidence that these patterns are causal.

Panel A of Table 2 shows estimates of β_1 from equation (1), which includes school-grade and grade-year fixed effects. We find that a 10 percent increase in the average share of elementary and middle school EBLL peers decreases the likelihood of graduating and taking the SAT by 1.6 and 2.8 percentage points, respectively, and increases the likelihood that a student drops out of school by 1.5

percentage points. Panel B of Table 2 presents estimates of β_1 from equation (2), which exploits within-sibling variation and is our preferred specification. Within-siblings comparisons generally estimate slightly smaller effects than comparisons within a school-grade, suggesting that family fixed effects better control for endogenous selection.[17]

Our preferred specification in Panel B shows that a child whose average cohort in elementary and middle school has 10 percent more lead-poisoned peers has a 1.7 percentage point lower likelihood of graduating high school – a 2 percent decrease on the mean graduation rate of 89 percent. We also find that having 10 percent more lead-poisoned peers increases the likelihood of dropping out by 0.5 percentage points and decreases the likelihood of taking the SAT while in high school by 2.3 percentage points, or a 4.3 percent decrease on the mean rate of 53.2 percent.[18] While a higher share of lead-poisoned peers decreases the likelihood that a student intends to attend a four-year college in Panel A, this result is not statistically significant at conventional levels in our preferred specification. Finally, after controlling for the share of lead-poisoned peers, we find little evidence of any effect of economically disadvantaged or non-White peers on graduation and dropout rates, and an effect of economically disadvantaged peers on SAT taking that is less than half the effect of lead-poisoned peers.

We estimate effects on college going that are similar in magnitude to those obtained by Carrell, Hoekstra, and Kuka (2018). Those authors find that adding one

[17] Because we identify siblings based on home addresses, our sibling-matching algorithm could lead to error. Table A1 Panel B estimates the same specification as in Panel A of Table 2 on the sibling sample and finds similar results using the sibling vs. all-children samples. Further, Column 1 of Table 7 shows results on the sample of Census tracts where the majority of homes are single family homes, where sibling attribution is more precise.

[18] We also report p-values corrected for multiple hypothesis testing, which are similar to baseline estimates. We use Stata command *rwolf2* based on Clarke, Romano and Wolf (2020), which allows for dependence among p-values by bootstrap resampling.

male peer exposed to domestic violence to a classroom decreases four-year college going by 1.4 percentage points. Using our cohort results and assuming that there are 25 students in a class, we calculate that one additional lead-poisoned peer in each class, a 4 percent increase in the share of lead-poisoned peers, would lead to a 0.92 percentage point reduction in the likelihood of taking the SATs, a proxy for college intentions, and a 0.67 percentage point reduction in graduating high school.

Next, we show that spillovers of lead poisoning increase both with the share of lead-exposed peers and with the severity of peers' lead exposure. Panel A of Figure 4 plots estimates from equation (2) using bins for different percentages of cohort peers with elevated BLLs (0-5, 5-10, 10-15, 15-20, 20-100), where the 0-5 percent bin is the omitted category. We also test whether estimates are statistically different from the 5-10 percent bin coefficient and find a statistically significant stronger effect of lead-poisoned peers on graduation rates as the percentage of peers with elevated BLLs increases above 15 percent. In Panel B, we show the effects of the average share of lead-poisoned peers on the likelihood of graduation using different thresholds to define peers' lead-poisoning (e.g., a BLL ≥2 μg/dL, ≥3 μg/dL, etc.). As the EBLL threshold increases, so does the negative effect on graduation.[19] Estimates with a lead poisoning definition of BLL ≥7 μg/dL or higher are significantly larger than our main estimates (defined as BLL ≥5 μg/dL). These results suggest that BLLs drive these peer effects, rather than other potentially correlated characteristics of children.

Because peers might impact each other differently at different ages, in Panel C of Table 2 we show the long-run effects of lead-poisoned peers from elementary and middle school cohorts *separately*. Peers in middle school could be especially impactful for long-run outcomes if middle school is when students decide whether to remain in school. Recent interventions in middle school have been very effective

[19] The effects of having more peers with at least 10 μg/dL flatten out, possibly due to interventions triggered when a child has BLLs above the 10 μg/dL threshold.

at reducing crime, suspensions, and dropping out of school, suggesting that students' outcomes can be strongly affected beyond early childhood.[20] Indeed, we find that our estimated long-run effects appear to be largely driven by middle school peers.[21] Yet, we note that elementary and middle school peers are highly correlated.

B. Mechanisms: The Contemporaneous Effects of Peers Exposed to Lead

To understand the mechanisms through which lead-poisoned peers might affect long-run outcomes, we next examine the effects of peers with elevated BLLs on contemporaneous test scores, out-of-school (OOS) suspensions, and absences. To do so, we estimate analogs of Equations (1) and (2) at the student-year level:

(3) $Y_{isgt} = \beta_1 \frac{\sum k \neq i \; PeersEBLLs_{ksgt}}{n_{sgt}-1} + \pi X_{it} + \omega S_{sgt} + \eta_{sg} + \phi_{gt} + \gamma_e + \varepsilon_{isgt}$

(4) $Y_{ijsgt} = \beta_1 \frac{\sum k \neq i \; PeersEBLLs_{ksgt}}{n_{sgt}-1} + \pi X_{it} + \omega S_{sgt} + \theta_j + \delta_s + \tau_g + \sigma_t + \gamma_e + \varepsilon_{ijsgt}$

where $\frac{\sum k \neq i \; PeersEBLLs_{ksgt}}{n_{sgt}-1}$ is the share of students in a child's school-grade-year cohort (or school-classroom-grade-year cohort) with *known* EBLLs, not including the student themselves. The coefficient β_1 on $\frac{\sum k \neq i \; PeersEBLLs_{ksgt}}{n_{sgt}-1}$ captures the effect of having 100 percent of a child's peers in a given year with *known* EBLLs. Equation (3) mirrors equation (1) by including school-by-grade (η_{sg}) and grade-by-year (ϕ_{gt}) fixed effects. X_{it} is a vector of child-specific control variables, including gender, race, birth month fixed effects, economically disadvantaged status in each year, and an indicator for whether a child was tested for lead. The vector S_{sgt} controls for time-varying characteristics at the school-grade-year level:

[20] See, for example, Guryan et al. (2021) and Heller et al. (2017).
[21] While the coefficients on middle school peers are always larger in magnitudes than those on elementary school peers, we only detect a statistically significant difference (at the 10 percent level) for dropout and SAT taking.

the percent non-White students, the percent economically disadvantaged students, and the share of students who have been tested for lead exposure. We also control for school time-varying characteristics: annual school size, the share of teachers with Master's degrees and the school-level stability rate. In equation (4), θ_j, δ_s, τ_g and σ_t are family, school, grade, and year fixed effects as in equation (2). In addition, when we look at test scores, we include γ_e, an exam fixed effect that restricts our comparison to children who took the same test.

Panel A of Table 3 presents the results for the effect of additional cohort peers who are lead-poisoned on a child's outcomes using equation (3), while Panel B uses our preferred specification in equation (4).[22] The two panels show very similar results: a higher share of peers with EBLLs is associated with a higher likelihood of, and longer, OOS suspensions, as well as a higher likelihood of absences. Again, within-siblings comparisons generally estimate slightly smaller effects than comparisons within a school-grade.[23]

In Panel B, we find that a ten percent increase in the proportion of cohort-level peers with elevated BLLs in a given year leads to a 0.2 percentage point increase in the likelihood of OOS suspensions, a 2.1 percent increase over the mean of 9.4 percent, and increases the suspension duration by 40 minutes based on a 6-hour school day. Moreover, these increased suspensions appear to be driven at least in part by suspensions on the same day as suspensions for lead-poisoned children and behavioral incidents including lead-poisoned children.[24]

[22] The sample size is smaller than in Column 1 of Table 1 due to singletons and missing outcomes.
[23] Because we only observe long-run outcomes for early cohorts of students, students move out of state, and some data only becomes available in later years, the numbers of students differ for short and long-run outcomes. However, in Panel A of Table A1, we estimate our primary specification on only students for whom we observe outcomes consistently. The results on this sample are very similar to our main results in Tables 2 and 3.
[24] Placebo estimates in Figure A3 show that our estimated effects of higher shares of peers with EBLLs on the likelihood of being suspended or involved in an incident with a student with EBLL are 154-165% of what the mere proportion of children with EBLLs in a given cohort implies.

Increased suspensions for peers of lead-poisoned children could be due to more punitive policies at the cohort-level, like teachers' responses. To disentangle peers' behavior from school policies, we look at the effects of lead-poisoned peers on absences, which should not be driven by school policies. We find that a 10 percent increase in the proportion of cohort-level peers with elevated BLLs increases the likelihood of chronic absenteeism by 0.4 percentage points, or 8 percent on a base of 5.2 percent, suggesting that our results are not driven by school policies. Finally, we find little evidence of lead-poisoned students affecting their peers' test scores, with estimates changing sign across specifications. This finding suggests that the effects of lead-poisoned peers on the long-run outcomes described in Section VA may operate through noncognitive skills and behavior, rather than a learning channel.[25] Yet, we cannot fully disambiguate between these channels.

While we use cohort-level variation in our primary specification to avoid the issue of selection into classrooms by students, Table A2 presents the estimates of the effect of having more lead-poisoned peers in the same *classroom.*[26] Classroom peers have a larger effect on suspensions and absences than cohort peers, which could be due to stronger connections within or selection into classrooms.

C. Heterogeneity of Estimated Effects by Own and Peers' Characteristics

Because exposure to lead-poisoned peers could interact with a child's background to shape their outcomes, we next study heterogeneity in peer effects by demographic subgroups. For example, students of different socioeconomic status might have differential access to resources, such as academic help outside of

[25] Several recent papers have shown that noncognitive skills are very important to long-run outcomes over and above test scores, such as Chetty et al. (2011) and Jackson (2018).

[26] We only have classroom-level data for a subset of the children in the sample from 2006 to 2017, whereas the cohort level variation is available from 1997-2017. Since we restrict test scores to 1997-2005, we cannot estimate the effects of classroom peers on test scores. Children in grades 6 and up usually switch classrooms, so they appear as many times as the number of classes they take with each student.

school, that could mitigate the effects of peers with EBLLs. Table 4 presents our preferred estimates by race/ethnicity (White, non-Hispanic in Panel A, Black students in Panel B, and Hispanic students in Panel C), by economically disadvantaged status (never economically disadvantaged in Panel D, sometimes economically disadvantaged in Panel E, and always economically disadvantaged in Panel F), and by gender (girls in Panel G and boys in panel H).

We find some evidence of heterogeneous effects of lead-poisoned peers on graduation by race and gender. Black students see the largest decrease in high school graduation from lead-poisoned peers. A 10 percent increase in the average share of lead-poisoned peers in elementary and middle school decreases the likelihood Black students graduate high school by 3 percentage points, compared to 1.5 percentage points for White students. Boys also seem more affected than girls by lead-poisoned peers, although the difference is not statistically significant. Importantly, Black and male students have lower graduation rates to start with, suggesting that peers might exacerbate existing educational disparities. However, we find little systematic evidence of heterogeneity by socioeconomic status.[27]

We also hypothesize that friends' groups might drive peer effects.[28] As we lack data on friendship networks, we use the fact that children likely sort into groups with similar characteristics (Jackson 2010). Table A4 presents both the effect of exposure to a higher share of lead-poisoned peers and the *additional* effect of exposure to a higher share of lead-poisoned peers of the same gender (Panel A), race (Panel B), and same gender and same race (Panel C). We find that same gender peers, but not same race peers, with EBLLs have an additional effect on high school graduation on top of the general effect generated by all lead-exposed peers, though

[27] Table A3 shows similar patterns when we estimate the effects of lead-poisoned peers for children in schools with different levels of poverty.

[28] Given that long-run outcomes are the focus of the paper, we only examine the likelihood of high school graduation and SAT taking for the rest of the paper since these outcomes are those for which we find the most consistent spillover effects.

the results are only statistically significant at the p<0.1 level. Moreover, Panel A of Table A5 shows that lead-poisoned boys have larger negative effects on their peers than lead-poisoned girls for SAT taking. Together, these results suggest that peer effects are mediated by assortative matching of peer groups.

D. Addressing Potential Measurement Error in BLLs and Omitted Variable Bias

We do not observe lead exposure for all children and there may be selection in who is tested for lead. Since we compute the share of lead-poisoned peers over all students in a cohort, irrespective of whether they have a blood lead test, *unknown* lead-poisoned peers could attenuate our results if measurement error is random. The direction of the bias depends on selection, if instead measurement error is not random. To address this potential measurement error as well as concerns about other potential correlates of peers' lead poisoning, we flip our main specification and regress the average outcomes for a student's peers on an indicator for that student having an EBLL using block group-by-birth year fixed effects. We estimate this specification only on the sample of tested children, for whom lead poisoning status is known. The estimating equation for this specification is as follows:

$$(5)\ \overline{Y}_{isg} = \beta_1 LeadPoisoned_{isg} + \pi X_i + \eta_{bt(i)} + \delta_s + \tau_g + \varepsilon_{isg}$$

where $\overline{Y}_{isg}$ represents the average outcomes of student *i*'s peers in a cohort in school *s* as of grade *g*, that is the last grade we observe a student. $LeadPoisoned_{isg}$ is an indicator for student *i* attending school *s* in grade *g* having an EBLL by age six. Thus, the coefficient of interest, β_1, estimates the effect of child *i* being lead poisoned on the average outcomes of their peers relative to other screened children who tested negative. We include block group-by-birth year fixed effects ($\eta_{bt(i)}$), as well as grade (τ_g) and school (δ_s) fixed effects. X_i includes gender, race, economically disadvantaged status, birth month and birth order fixed effects, the share of non-White peers, the share of economically disadvantaged peers, the share

of peers tested for lead, the share of peers with EBLLs, the student's school size, school stability rate and share of teachers with Master's degrees in the last grade we observe student i. ε_{isg} represents the error term.

We use block group-by-birth year fixed effects to absorb any time-varying neighborhood characteristics, including gentrifying patterns that could be correlated with home renovations and time-varying pollution that could correlate with lead poisoning. Thus, we identify the effect of lead poisoning by comparing peers' outcomes of children living in the same small neighborhood and time but who happen to have different EBLLs. Abbasi, Gazze, and Pals (2020) suggest that even within a Census block, the age of the housing is highly predictive of blood lead levels, so we aim to capture this source of variation with this specification. In Table 5, we find that one lead poisoned student in a cohort of 220 is associated with a 0.09 percentage point decrease in the share of peers who graduate high school or take the SAT when using our full sample of blood lead tests in Panel A. This is very similar to what we obtain if we scale our main result in Table 2 Panel B.[29]

Table A6 further investigates whether our results might be driven by characteristics of the lead poisoned student that are correlated with that student's lead poisoning, such as family background. We note that adding individual controls (gender, race, economic disadvantage) in Column 2 decreases our estimates by about a quarter, in line with our suggestive findings of potential homophily by gender, for example. Yet, adding additional controls for parental education, family composition (number of children in family and siblings' gender mix) and school controls (share non-white, share economically disadvantaged, school size, the stability rate, and the percent of teachers with an MA degree) does not alter our

[29] As 1 in 220 students is a 0.46% increase in the share of peers with elevated BLLs, we multiply that by our estimate of the effect of 100% of peers with elevated BLLs on graduation (-16.71 percentage points) to obtain the impact of one child with EBLLs through elementary and middle school on graduation rates: -0.077 percentage points, or a decrease in the probability of 0.00077.

estimates from equation (5) significantly (Columns 3 and 4). Finally, Column 5 shows that controlling for family fixed effects in this specification decreases our sample size by two thirds and our effective sample size by 85%. Consistently, estimates in Column 5 are noisier, with smaller point estimates in the whole sample (Panel A) but much larger in the samples of children tested by 37 and 25 months (Panels B and C, respectively).[30]

To address potential selection into screening due to manifested behavior at older ages, in Tables 5 (Panels B and C) and A7 we limit our variation to blood lead tests taken by 25 or 37 months of age. Table A7 presents estimates from our main estimating equation (2) on this subsample, which are largely similar to our main results in Table 2.

Next, we directly test whether screening appears random conditional on our preferred set of controls and fixed effects. In Figure A4, we find that as the share of tested children increases by 10 percent in a school cohort, the share of children testing positive for lead decreases by only 0.006 percentage points – essentially zero. This finding is consistent with the same law of small numbers that we exploit for identification. In other words, conditional on neighborhood and family time-invariant observable factors that predict lead exposure risk, which children actually get tested within a cohort is plausibly exogenous. In Appendix C, we also simulate selection under different testing regimes and estimate biases in the range of -2.3 to 8 percent of the true effect. Thus, selection into testing is unlikely to bias our findings in an economically significant way.

[30] Screening at an early age is consistent with being on Medicaid or living in older housing, so we suspect that our estimates are larger because there is less measurement error in BLLs among siblings who are all tested early. Moreover, Gazze 2022 shows that families are more likely to test a second sibling if the first one has an EBLL but less likely to test a second sibling if the first one is tested and does not have an EBLL. Thus, we do not use this much smaller and potentially selected sample as our main estimate for the "flipped" analysis.

Finally, we find no evidence of selection into screening when we regress child, family, and school characteristics on an indicator for whether or not a child was tested for lead, controlling for all of the same controls and fixed effects in our primary specification. The results, presented in Table A8, show that on average, being tested for lead is not correlated with parental education, race or school characteristics. However, children tested for lead are 2 percentage points more likely to be female. Nevertheless, we control for gender in all regressions. Thus, we conclude that measurement error plausibly attenuates our estimates.

To further assess the extent of bias from measurement error, we perform a bounding exercise in which we assign different shares of untested children to have EBLLs. Specifically, we assign the 90th or 10th percentile of the distribution of observed shares of students with EBLLs within schools or districts to peers with missing BLL data. Then, we average these imputed estimates for untested children with the observed percentage of peers in a cohort with EBLLs, weighting by share untested and tested, respectively, as follows:

$$ImputedShareEBLL_{sgt}^{p} = \left(\frac{\sum k \neq i \ PeersEBLLs_{ksgt}}{NTested_{sgt} - 1}\right)\left(\frac{NTested_{sgt} - 1}{N_{sgt} - 1}\right) + p_s\left(\frac{\sum k \neq i \ PeersEBLLs_{ksgt}}{NTested_{sgt} - 1}\right)\left(1 - \frac{NTested_{sgt} - 1}{N_{sgt} - 1}\right)$$

where $p_s\left(\frac{\sum k \neq i \ PeersEBLLs_{ksgt}}{NTested_{sgt}-1}\right)$ is the pth percentile of the distribution of observed shares of students with EBLLs within school s (or the district the school belongs to), $NTested_{sgt}$ is the number of tested children in the school-grade-year, and N_{sgt} is the total number of students in the school-grade-year.

In Panels A and B of Table 6, we use the 90th and 10th percentile of the empirical distribution of observed BLLs by school for missing BLL data, respectively. Imputing the 90th percentile of the distribution of children with EBLLs

reduces the magnitude of our estimates on graduation and SAT taking by 25-40%, but both coefficients remain negative and statistically significant at the $p<0.01$ level.[31] Imputing the 10th percentile, instead, produces estimates that are very similar to our preferred ones in Panel B of Table 2. Panels C and D repeat this exercise using the 90th and 10th percentile within school districts, respectively, and yield similar outcomes albeit attenuated likely due to wider averaging. Finally, Panel E presents results where we predict BLLs out of sample for the unscreened children based on our rich school and census data and set of fixed effects and use the predicted shares of EBLLs for the share of missing BLLs. Again, we obtain similar estimates to those in Table 2. We interpret these findings as suggestive that missing BLL data are not causing us to attribute spurious effects to lead-poisoned peers. As an additional check, Column 2 of Table 7 shows the effects of lead-poisoned peers on children in universal screening zip codes, where screening rates are 16 percent higher than average.[32,33] We find only a slightly larger effect on graduation than in the full sample.

To address further concerns on measurement of lead poisoning, in Panels B, C and D of Table A5, we show that our results are largely robust to using different measures of lead-exposed peers. Moreover, missing information on a student's own lead poisoning status does not bias our estimates: when we estimate the effects of lead-poisoned peers only on students who have tested negative for lead poisoning (Panel E), we obtain coefficients that are very similar to those in

[31] There is also reason to believe untested children are less likely to have EBLLs. As shown in Table 1, they are less likely to be minority and economically disadvantaged, which is correlated with lead poisoning. Gazze (forthcoming) and Abbasi, Gazze, and Pals (2022) also find that children who are not tested for lead are less likely to have EBLLs than those who are tested for lead.

[32] These zip codes cover at least one block group with 27 percent or more homes built prior to 1950 and areas with high prevalence of elevated BLLs (Hanchette 1999).

[33] Table A9 repeats the robustness exercise shown in Table 7 for SAT taking, as well as out-of-school suspensions and chronic absenteeism. Our results on SAT taking and absences are very robust to different samples and specifications. Our suspensions results are similarly robust in terms of magnitude, but noisy at times.

Table 2. The same is true when we include also children who tested positive (Panel F), suggesting that spillover effects are not mediated by one's own poisoning status.

E. Additional Threats to Internal Validity

This section discusses and tests for threats to internal validity, including spurious correlation and endogenous sorting.

Our estimates could be biased if the share of peers with EBLLs in a school-grade-year is systematically correlated with student or peers' characteristics that affect a student outcome other than those included in equation (2). In addition to the alternative specification in Table 5, Column 3 of Table 7 controls for the share of a student's peers who live in block groups with above-median income, share Black and Hispanic residents, share in poverty, and share with a high school degree. The estimate of the effects of lead-poisoned peers is virtually indistinguishable from our main estimate in Table 2. Column 4 of Table 7 adds fixed effects for the Census block group where students reside when they first appear in the school data. Both columns suggest that neighborhood characteristics, such as air pollution, do not drive the results. Column 5 of Table 7 further shows that estimates using more stringent school-grade fixed effects are similar to our main results.

We also test whether our estimated effect of lead poisoning is driven by the socioeconomic status of the lead poisoned children. To do so, we estimate the effect of lead poisoned children with higher income and who are White (Panels A and B of Table A10), and further control for peers' parental education (Panel C of Table A10). Our results are robust to these alternative specifications, which suggests that our results are not driven by socioeconomic status of children with EBLLs.

To further rule out spurious correlation, Table A11 shows the robustness of our specification to different sets of controls. When we omit all controls other than family, school, grade, year fixed effects, and the share of peers tested for lead (Panel A), estimates are similar to those in our main specification, albeit slightly larger.

Panel B shows that once we add individual and school-level controls, omitting the average share of students who are non-White and the average share of students who are economically disadvantaged does not affect our estimates compared to our main results. So, peers' characteristics other than lead poisoning do not appear to explain much of the variation in students' outcomes after including the set of fixed effects and controls that provides our identification. Relatedly, Table A12 shows limited evidence that peers' composition at the cohort level is related to school quality or resources in a way that could confound our estimates. Cohorts with higher share of students with EBLLs appear to be in school-years with higher stability rate, if anything, and larger student bodies.[34] Panel C of Table A11 shows that excluding school fixed effects yields slightly larger peer effects on suspensions compared to our main results. These results suggest that our more conservative primary specification controls for unobserved time invariant school characteristics. Our results also hold when we add school-specific linear time trends to our preferred specification (Panel D).

Figure A5 further shows that our estimates are unlikely to be due to spurious correlation. This figure plots the results from estimating 500 placebo specifications in which we assign a random share of lead-poisoned peers to each school-grade-year cohort drawn from a distribution with the same mean and standard deviation as the empirically observed peers' distribution. Our true estimates for the effects of lead-poisoned peers on graduation rates and SAT taking fall well outside the distribution of estimates from the placebo specifications.

[34] While we find no difference in school quality or characteristics between siblings in Table A12, lead poisoning is correlated with race and socioeconomic status, as seen in Table 1. Thus, we do not include these observed characteristics of peers in the table, and instead control for them in our main specification. This fact also underscores why we use family fixed effects to account for time invariant characteristics of families, such as race and economic status, that could affect outcomes. On average, these individual characteristics also do not vary within families.

Because the incidence of lead poisoning has decreased over time (Figure 1), our primary estimates might capture similarly occurring trends in outcomes, despite controlling for grade and year fixed effects. To assuage this concern, in Column 6 of Table 7, we control for school-year fixed effects and find peer effects that are larger than our main results.

Endogenous sorting into peer groups could also bias our results if high-achieving students sort out of cohorts with many lead-poisoned students, for example. Importantly, most of North Carolina did not offer school choice options for public schools up until the 2014-2015 school year, unless students switched into a charter or magnet school, which we observe.[35] Column 7 of Table 7 shows that our results are larger for children in zip codes with no charter schools or other school choice options (at the time), which are effectively no-choice zip codes. Column 8 of Table 7 controls for siblings-by-school fixed effects, effectively comparing siblings only in grades during which they attend the same school as in Bertoni, Brunello, and Cappellari (2020). We find spillover effects of lead-poisoned peers that are two-thirds the size of our main result. Finally, Table A13 formally investigates the association between a student's share of lead-poisoned peers and students or their siblings switching to public or charter schools. The results indicate that endogenous sorting is not a concern in our setting.

Finally, including siblings fixed effects could lead us to underestimate the spillover effects of lead exposure in the presence of within-family spillovers. For example, if parents respond to their children's performance by shifting resources across offspring, a student's peers could also affect the outcomes of that student's siblings. Table A14 shows no evidence of this by controlling for the share of lead-

[35] In the 2014-2015 school year, North Carolina implemented the Opportunity Scholarships program, a voucher program for low-income children. Children whose families make less than 133 percent of the qualifying amount for the federal free or reduced-price lunch program qualify for the voucher, which can be used for any school. Because the Charlotte Mecklenburg Public School district has had a school choice program from 2002 we exclude it in this robustness check.

exposed peers of a student's siblings. Our results are also similar when we add controls for sibling gender composition and family size to our preferred specification (Table A11 Panel E).

VI. Conclusion

This is the first study showing that pollution has long-run spillover effects on school peers. By comparing siblings, we show that a child's own lead exposure spills over to affect other children's long-run outcomes, including high school graduation and SAT taking. These effects suggest that the social cost of lead exposure has been underestimated so far. In addition, we reveal some mechanisms through which peer effects manifest, namely behavior shaping while in middle school likely through noncognitive skills. Thus, our findings have implications for other types of common pollution that have been linked to suspensions from school, such as traffic and industrial pollution (Persico and Venator 2020; Heissel, Persico, and Simon 2020), suggesting that the cost of pollution has been underestimated. These findings hold even though we likely underestimate the effect of lead-poisoned peers due to potential within-family spillovers and measurement error. Thus, environmental hazards appear to contribute to human capital accumulation, even for children who are not themselves exposed to these hazards.

While external validity issues make it difficult to extrapolate how lead exposure might affect labor market outcomes, we attempt a back-of-the-envelope calculation for the social cost of the spillovers of lead poisoning. We find that being exposed to one additional lead-poisoned peer in a cohort of 220 is associated with $84 in lost earnings per student from lower graduation rates alone, that is excluding the additional costs of behavioral issues and absences.[36] This estimate implies a

[36] Following Heckman, Lochner, and Todd (2006), we estimate the net present value of graduating high school to be $93,188. We estimate a schooling-experience-earnings profile non-parametrically in the 2018 March Current Population Survey data and predict earnings conditional on years of schooling at each age between 18 and 65, assuming a growth rate of real labor productivity growth

spillover effect of a lead-poisoned child of $18,368 on their 219 school peers. As half a million young children are poisoned by lead each year (Aizer et al. 2018), these spillovers total almost $9.2 billion per birth-year cohort. Reyes (2014) estimates the direct social cost of lead poisoning at $200 billion per birth-year cohort. Thus, our lower-bound estimates suggest that the social cost of lead has been underestimated by at least 4.6 percent by not including these spillover effects. Because lead-poisoned students are quite dispersed across schools, most public-school children in the US are likely affected by the spillover effects of lead.

Our results imply some important lessons for policy. Remediating lead hazards is likely to be more cost effective than previously supposed since lead exposure affects everyone in the classroom. Lead remediation efforts have shown positive impacts on children's blood lead levels and test scores (Sorensen et al. 2019). In addition, Billings and Schnepel (2018) show that offering early interventions for lead-poisoned children improves their school performance and decreases antisocial behavior, for a total benefit of $9,666 per directly exposed child. Our estimates suggest an additional potential benefit of about twice the direct benefit when accounting for the spillover effects of lead poisoning on school peers.

Finally, school segregation by race and socioeconomic status likely exacerbates these peer effects, suggesting that efforts to desegregate students might be beneficial. Low-income schools have some of the largest achievement gaps (e.g., see Reardon 2015). Lead exposure and exposure to lead-poisoned peers are both mechanisms through which poverty produces worse human capital outcomes.

of 1.9 percent and a discount rate of 3.38 (i.e., the 30-year Treasury bond rate). Thus, one child with EBLLs in a cohort decreases the net present value of lifetime earnings by 0.0009*$93,188=$84.

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Table 1: Characteristics of children and schools

Sample:	(1) All children attending public school in North Carolina	(2) Children in sibling sample	(3) Children with BLL test	(4) Children with EBLLs	(5) Children without known EBLLs	(6) Children with above-median share of EBLL peers in at least one elementary grade	(7) Children with below-median share of EBLL peers in all elementary grades
Average test score	0.001	0.063	-0.117	-0.288	0.128	-0.093	0.264
Any out-of-school suspension	0.265	0.258	0.305	0.404	0.238	0.314	0.202
Ever graduated	0.837	0.872	0.866	0.816	0.881	0.848	0.895
4-year college intentions	0.418	0.454	0.398	0.346	0.471	0.387	0.516
Has taken the SAT	0.434	0.466	0.411	0.366	0.482	0.405	0.521
Cohort size	220	225	199	203	229	193	262
Share of teachers with an MA degree	0.338	0.356	0.346	0.335	0.359	0.337	0.377
Share economically disadvantaged	0.438	0.441	0.512	0.521	0.429	0.528	0.343
Stability rate	0.929	0.957	0.957	0.953	0.958	0.955	0.960
Share Black	0.277	0.266	0.285	0.331	0.256	0.309	0.217
Share Hispanic	0.102	0.107	0.120	0.103	0.108	0.114	0.099
Share with a BLL test	0.338	0.396	1	1	0.322	0.533	0.256
Share with EBLL	0.097	0.109	0.276	1	0	0.165	0.052
N Students	3,334,365	1,326,622	525,535	144,957	1,181,665	670,559	656,063

Notes: The table presents summary statistics for selected variables in our sample. Observations are at the student-year level. Cohort is defined as student-grade-year. Column 1 shows the means for all children in our original sample. Column 2 shows means for children with siblings, that is our main sample. Column 3 shows means for children that have a blood lead level test. Column 4 shows means for children with elevated blood lead levels (EBLLs), and Column 5 shows means for children without elevated blood lead levels. Column 6 shows means for children whose share of elementary school peers with elevated BLLs was above the median share at the grade-year level in at least one grade, while Column 7 shows means for children whose share was below the median in all elementary grades. Test scores are standardized at the grade-year level. The stability rate is defined as the percentage of students from the October membership count who are still present in the second semester (90 days later)

Table 2: Long-run Outcomes of Exposure to Peers with Elevated BLLs by Timing of Exposure

	(1)	(2)	(3)	(4)	(5)
Dependent Variable:	Ever graduated	Ever dropped out	Intention to Attend a 4-Year College	Intention to Attend a Community College	Took the SAT
Panel A: Share of All Peers with EBLLs Over Elementary and Middle School with School-Grade and Grade-Year Fixed Effects					
Share of peers with BLLs over 5 µg/dL	-0.1558***	0.1453***	-0.1257***	-0.0112	-0.2769***
	(0.0173)	(0.0150)	(0.0360)	(0.0343)	(0.0349)
Romano-Wolf adjusted p-value	0.002 (**)	0.002 (**)	0.002 (**)	0.988	0.002 (**)
Mean of outcome	0.8491	0.0597	0.4407	0.3544	0.4589
N Students	831,147	1,155,293	666,613	665,951	657,670
Panel B: Share of All Peers with EBLLs Over Elementary and Middle School with Sibling, School, Grade and Year Fixed Effects					
Share of peers with BLLs over 5 µg/dL	-0.1671***	0.0470^{+}	-0.1171	0.0457	-0.2283**
	(0.0350)	(0.0246)	(0.0720)	(0.0785)	(0.0740)
Romano-Wolf adjusted p-value	0.002 (**)	0.040 (*)	0.118	0.926	0.002 (**)
Mean of outcome	0.8904	0.0529	0.5069	0.3288	0.5320
N Students	282,964	414,562	205,760	205,688	201,713
Panel C: Share of Elementary Versus Middle School Peers with EBLLs with Sibling, School, Grade and Year Fixed Effects					
Share of peers with BLLs over 5 µg/dL in Elementary School	-0.0611*	-0.0064	-0.0104	0.0240	-0.0024
	(0.0295)	(0.0218)	(0.0671)	(0.0685)	(0.0669)
Romano-Wolf adjusted p-value	0.016 (*)	0.988	0.990	0.986	0.990
Share of peers with BLLs over 5 µg/dL in Middle School	-0.1184**	0.0710*	-0.0545	-0.0106	-0.2214*
	(0.0414)	(0.0299)	(0.0906)	(0.0987)	(0.0872)
Romano-Wolf adjusted p-value	0.002 (**)	0.006 (**)	0.926	0.990	0.006 (**)
p-val. Elementary =middle	0.35	0.08	0.75	0.81	0.10
Mean of outcome	0.8945	0.0519	0.5108	0.3299	0.5380
N Students	248,391	354,195	182,305	182,248	178,831

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's long-run outcomes. Column 1 reports the effects on the likelihood a student ever graduates from high school, and column 2 shows the effects on the likelihood of ever dropping out of school. Columns 3 and 4 show the effects on self-reported intention of enrolling in a four-year college and community college, respectively. Column 5 shows the effects on the likelihood of taking the SAT test by grade 12. Panel A includes school-by-grade and grade-by-year fixed effects. Panels B and C instead include family, school, grade, and year fixed effects, controlling for birth order and birth month. All regressions include individual controls for gender, race, whether the student has a blood lead level test, and economically disadvantaged status measured in the highest grade a student is observed in. We also control for the average share of elementary and middle school peers that are non-White or ED, average share of children with a lead test, school size, the stability rate, and the percent of teachers with an MA degree averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. We also report p-values corrected for multiple hypothesis testing. We use Stata command *rwolf2* based on Clarke, Romano and Wolf (2020), which allows for dependence among p-values by bootstrap resampling. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table 3: Potential Mechanisms: Contemporaneous Effects of Attending School with an Increased Share of Children with Elevated BLLs

Dependent Variable:	(1) Average Test Score	(2) Out of School Suspension (OSS)	(3) Days Suspended	(4) OSS Same Day as Lead-Exposed Child	(5) Incident with Lead-Exposed Child	(6) Absent 22 or More Days
Panel A: Cohort Peers with School-Grade and Grade-Year Fixed Effects						
Share of peers with BLLs over 5 µg/dL	-0.0272	0.0677***	1.0974***	0.2250***	0.1107***	0.0234*
	(0.0351)	(0.0188)	(0.2424)	(0.0143)	(0.0118)	(0.0091)
Romano-Wolf adjusted p-value	0.196	0.020 (*)	0.020 (*)	0.020 (*)	0.020 (*)	0.020 (*)
Observations	3284720	7916670	7916670	7189301	6540081	8128020
N Students	930228	1906345	1906345	1883489	1764684	1902185
Mean of outcome	0.0572	0.1048	0.7732	0.0318	0.0202	0.0611
Panel B: Cohort Peers with Family, School, Grade and Year Fixed Effects						
Share of peers with BLLs over 5 µg/dL	0.0190	0.0205+	0.6099***	0.1929***	0.1041***	0.0434***
	(0.0361)	(0.0117)	(0.1807)	(0.0106)	(0.0082)	(0.0072)
Romano-Wolf adjusted p-value	0.373	0.020 (*)	0.020 (*)	0.020 (*)	0.020 (*)	0.020 (*)
Observations	1,409,299	4,287,750	4,287,750	3,919,448	3,672,544	4,395,695
N Students	373,801	944,335	944,335	933,508	891,259	939,430
Mean of outcome	0.1298	0.0942	0.6762	0.0285	0.0187	0.0520

Notes: The table reports the effect of a child's share of peers with EBLLs on the child's school outcomes. Both panels use the share of peers with maximum BLLs over 5 µg/dL *at the school-grade-year level* as the main explanatory variable. Panel A includes school-by-grade, grade-by-year and birth month fixed effects. Panel B instead includes family, school, grade, and year fixed effects, controlling for birth order. In Column 1, we take the average of math and reading test scores and additionally control for subject-by-type test fixed effects. In Columns 2-6 we limit the sample to grades 6 and above. All regressions control for individual and cohort controls, which include indicators for gender, race, economically disadvantaged status, whether the student has a blood lead level test, the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, the stability rate, and the percent of teachers with an MA degree. Standard errors are in parentheses and clustered at the school level. We also report p-values corrected for multiple hypothesis testing. We use Stata command *rwolf2* based on Clarke, Romano and Wolf (2020), which allows for dependence among p-values by bootstrap resampling. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table 4: Heterogeneity by Demographic Subgroups, All Long-run Outcomes

Dependent Variable:	(1) Ever Graduated	(2) Ever Dropout	(3) Intention to Attend a 4-Year College	(4) Intention to Attend a Community College	(2) Took the SAT
			Panel A: White, non-Hispanic students		
Share of peers w/ BLLs ≥5µg/dL	-0.1467***	0.0489	-0.0952	0.0013	-0.1202
	(0.0404)	(0.0301)	(0.0890)	(0.0985)	(0.0871)
			Panel B: Black non-Hispanic students		
Share of peers w/ BLLs ≥5µg/dL	-0.3140***	0.0384	0.0465	0.0454	-0.3714*
	(0.0768)	(0.0526)	(0.1731)	(0.1588)	(0.1683)
p-val. =White	0.05	0.86	0.47	0.81	0.19
			Panel C: Hispanic students		
Share of peers w/ BLLs ≥5µg/dL	-0.1367	0.0324	-0.1250	-0.1480	-0.4038+
	(0.1250)	(0.0823)	(0.2231)	(0.2452)	(0.2403)
p-val. =White	0.94	0.85	0.90	0.57	0.27
			Panel D: Never Economically Disadvantaged students		
Share of peers w/ BLLs ≥5µg/dL	-0.0911*	0.0257	-0.0419	-0.0645	-0.0157
	(0.0401)	(0.0222)	(0.1041)	(0.1138)	(0.1106)
			Panel E: Sometimes Economically Disadvantaged students		
Share of peers w/ BLLs ≥5µg/dL	-0.1809**	0.0476	-0.1700	0.1566	-0.2764*
	(0.0682)	(0.0457)	(0.1388)	(0.1400)	(0.1361)
p-val. =Never	0.26	0.67	0.46	0.22	0.14
			Panel F: Always Economically Disadvantaged students		
Share of peers w/ BLLs ≥5µg/dL	0.0266	-0.0445	-0.1496	-0.0163	-0.1426
	(0.1032)	(0.0776)	(0.1813)	(0.2028)	(0.2019)
p-val. =Never	0.29	0.38	0.61	0.84	0.58
			Panel G: Girls		
Share of peers w/ BLLs ≥5µg/dL	-0.1105+	0.0328	-0.0546	0.0556	-0.2376+
	(0.0605)	(0.0403)	(0.1306)	(0.1428)	(0.1311)
			Panel H: Boys		
Share of peers w/ BLLs ≥5µg/dL	-0.2513***	0.0724	-0.0592	0.0276	-0.2072
	(0.0668)	(0.0486)	(0.1295)	(0.1461)	(0.1358)
p-val. =Girls	0.12	0.53	0.98	0.89	0.87

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes for children with different observable characteristics in each panel. For each outcome, results are from three regressions, one for each characteristic (race, economic status, gender). All regressions include cohort and individual controls, as well as family, birth month, birth order, school, grade, and year fixed effects. Individual controls include indicators for whether the student has a blood lead level test, gender, race, and economically disadvantaged status. Cohort controls include the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, the stability rate, and the percent of teachers with an MA degree. Cohort and school controls are averaged over elementary and middle school. Standard errors are clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table 5: Effects of Having an Elevated BLL on Peers' Average Long-run Outcomes using Block Group-by-Birth Year Fixed Effects

	(1) Ever graduated	(2) Took the SAT
Panel A: Tested by 72 months of age		
Child's maximum BLL is >=5	-0.0009**	-0.0009***
	(0.0003)	(0.0002)
Observations	338,232	322,115
Mean of outcome	0.6153	0.2740
Panel B: Tested by 37 months of age		
Child's maximum BLL is >=5	-0.0011*	-0.0011***
	(0.0004)	(0.0003)
Observations	225,382	232,965
Mean of outcome	0.6217	0.2673
Panel C: Tested by 25 months of age		
Child's maximum BLL is >=5	-0.0008+	-0.0009**
	(0.0005)	(0.0003)
Observations	190,155	198,367
Mean of outcome	0.6226	0.2636

Notes: The table reports the effect of one additional child with elevated blood lead levels on the average long-run outcomes for their peers (equation 5). Column 1 reports the effects on the likelihood peers ever graduate from high school, and Column 2 shows the effects on the likelihood of ever taking the SAT. Panel A uses all blood lead tests in our sample. Panels B and C limit the sample to children screened by 37 and 25 months of age, respectively. We control for census block group-by-birth year fixed effects, as well as school, grade, birth month and birth order fixed effects. We also control for the share of peers that have EBLLs, the share of peers that are non-White or economically disadvantaged, share of children with a lead test, school size, the stability rate, and the percent of teachers with an MA degree. Standard errors are in parentheses and clustered at the school level. $^{+}$ $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 6: Bounding our Estimates to Account for Missing BLL Data

Dependent Variable:	(1) Ever graduated	(2) Took the SAT
Panel A: Imputing the 90th Percentile of the BLL Distribution by School for Missing BLL Data		
Share of peers with BLLs over 5 µg/dL	-0.0882***	-0.1396**
	(0.0231)	(0.0514)
Mean of outcome	0.8904	0.5320
N Students	282,964	201,711
Panel B: Imputing the 10th Percentile of the BLL Distribution by School for Missing BLL Data		
Share of peers with BLLs over 5 µg/dL	-0.1370***	-0.1980**
	(0.0318)	(0.0642)
Mean of outcome	0.8904	0.5320
N Students	282,964	201,711
Panel C: Imputing the 90th Percentile of the BLL Distribution by District Cohort for Missing BLL Data		
Share of peers with BLLs over 5 µg/dL	-0.1121***	-0.1672***
	(0.0261)	(0.0420)
Mean of outcome	0.8904	0.5320
N Students	282,964	201,711
Panel D: Imputing the 10th Percentile of the BLL Distribution by District Cohort for Missing BLL Data		
Share of peers with BLLs over 5 µg/dL	-0.0908***	-0.1628***
	(0.0204)	(0.0422)
Mean of outcome	0.8904	0.5320
N Students	282,964	201,711
Panel E: Using Predicted BLLs		
Share of peers with BLLs over 5 µg/dL	-0.1216***	-0.2201***
	(0.0304)	(0.0664)
Mean of outcome	0.8939	0.5408
N Students	264,999	189,611

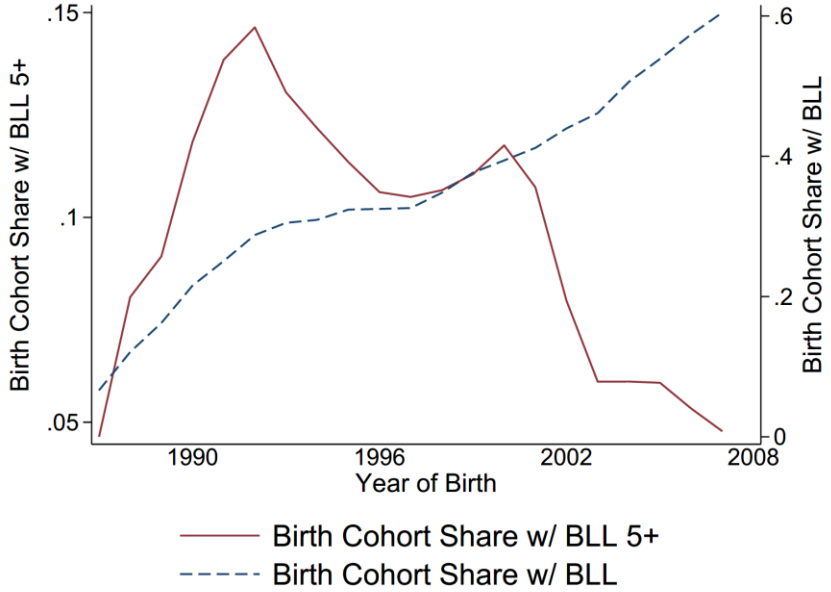
Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's long-run outcomes accounting for missing data on children's BLLs by bounding our estimates. In Panel A, we use the 90th percentile of the empirical distribution of observed BLLs by school to impute missing BLL data for a student's peers, and in Panel B we use the 10th percentile of the empirical distribution of observed BLLs by school. Panels C and D replicate Panels A and B using district-level percentiles. In Panel E, we regress an indicator for having an EBLL (of 5 or above) on the share of tested students by cohort, the share of tested students who have an EBLL, gender, individual race, racial demographics of the first school and census block group, all other census block group controls, and school, block group, birth year, birth month and birth order fixed effects. We then predict whether an unscreened child is likely to have an EBLL (out of sample) and calculate the shares of children by school-grade-year predicted to have EBLLs. We use the predicted shares of EBLLs for our share of missing BLLs and average it in with the share of student who are tested. Standard errors are in parentheses and clustered at the school level. $^{+}$ $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 7: High School Graduation Results for Alternative Samples and Alternative Specifications

	(1) >50% of Homes in Census Tract are Single Family	(2) Zip Codes with Universal Screening	(3) Adding Cohort Block Group Characteristics	(4) Adding Block Group Fixed Effects	(5) Adding School-Grade Fixed Effects	(6) Adding School-Year Fixed Effects	(7) Zip Codes with No School Choice Options	(8) Adding Sibling-School Fixed Effects
Share of peers with BLLs over 5 µg/dL	-0.1846*	-0.1851***	-0.1713***	-0.2234***	-0.1467***	-0.2293***	-0.2668***	-0.1030***
	(0.0719	(0.0453)	(0.0345)	(0.0591)	(0.0308)	(0.0449)	(0.0714)	(0.0266)
N Students	84,711	146,559	282,962	118,713	282,514	281,789	175,941	228,002
Mean of outcome	0.8830	0.8800	0.8904	0.8835	0.8913	0.8925	0.8934	0.9094
School FEs	X	X	X	X			X	
Sibling FEs	X	X	X	X	X	X	X	
Year FEs	X	X	X	X	X		X	X
Grade FEs	X	X	X	X		X	X	X

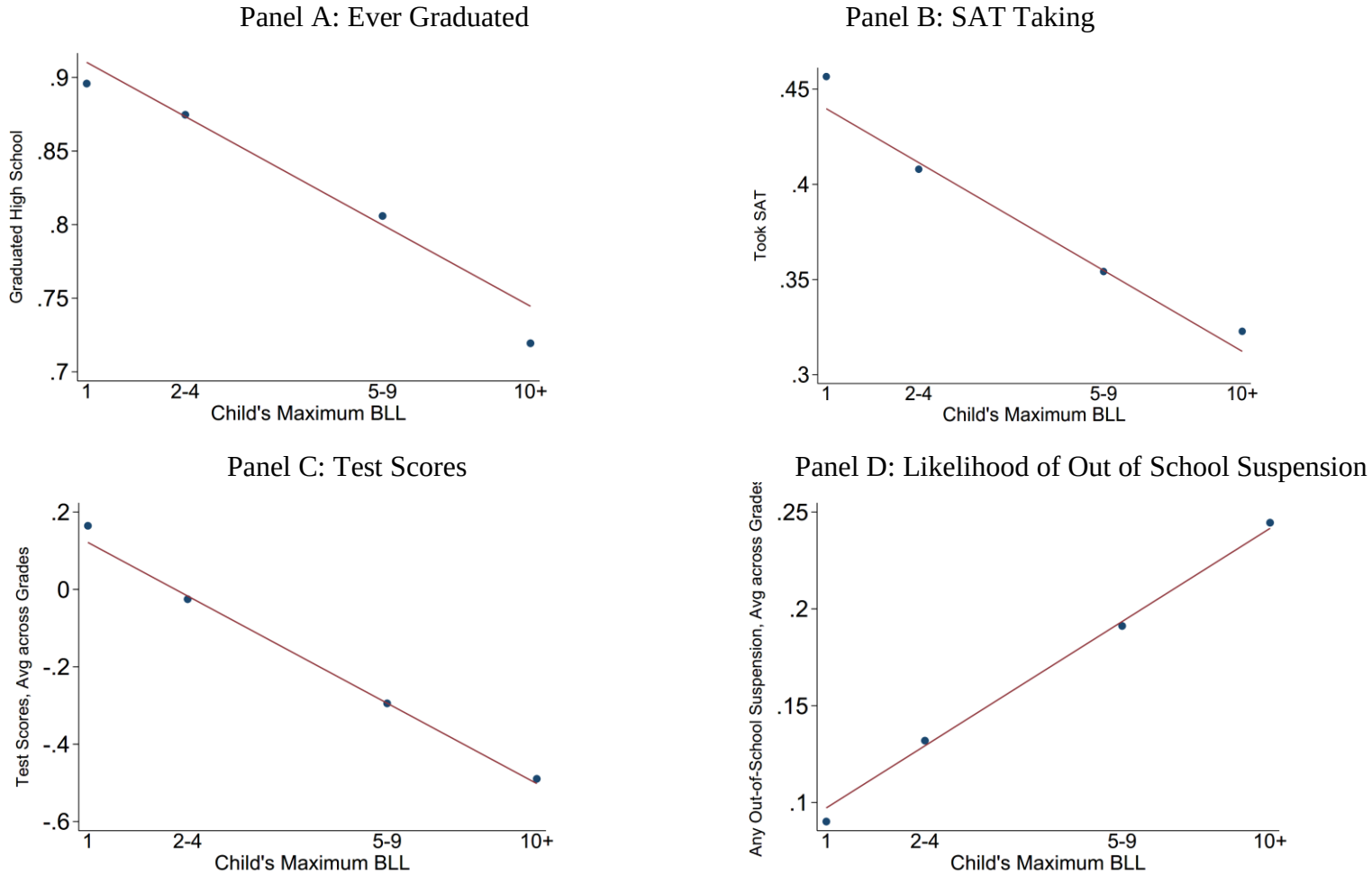
Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes. Each column reports results from a separate regression. Column 1 restricts the sample to Census tracts where more than half of homes are single family homes. Column 2 restricts the sample to students who live in zip codes that are subject to universal lead screening. Columns 3-6 and 8 add controls and alternative sets of fixed effects as specified at the top and bottom of each column. Column 7 restricts the sample to zip codes without charter schools or voucher programs. Block group characteristics of cohort peers include share of peers that live in block groups with above median income, above median percent Black and Hispanic population, above median percent of the population living in poverty, and with above median percent population with a high school degree. All regressions include cohort and individual controls, as well as birth month and birth order fixed effects. Individual controls include indicators for gender, race, economically disadvantaged status, and whether the student has a blood lead level test. Cohort controls include the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged. We also control for school size, the stability rate, and the percent of teachers with an MA degree. Cohort and school controls are averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Figure 1: Share of Children with Blood Lead Levels at or above 5µg/dL by Birth Cohort and Share of Children with Blood Lead Tests by Cohort



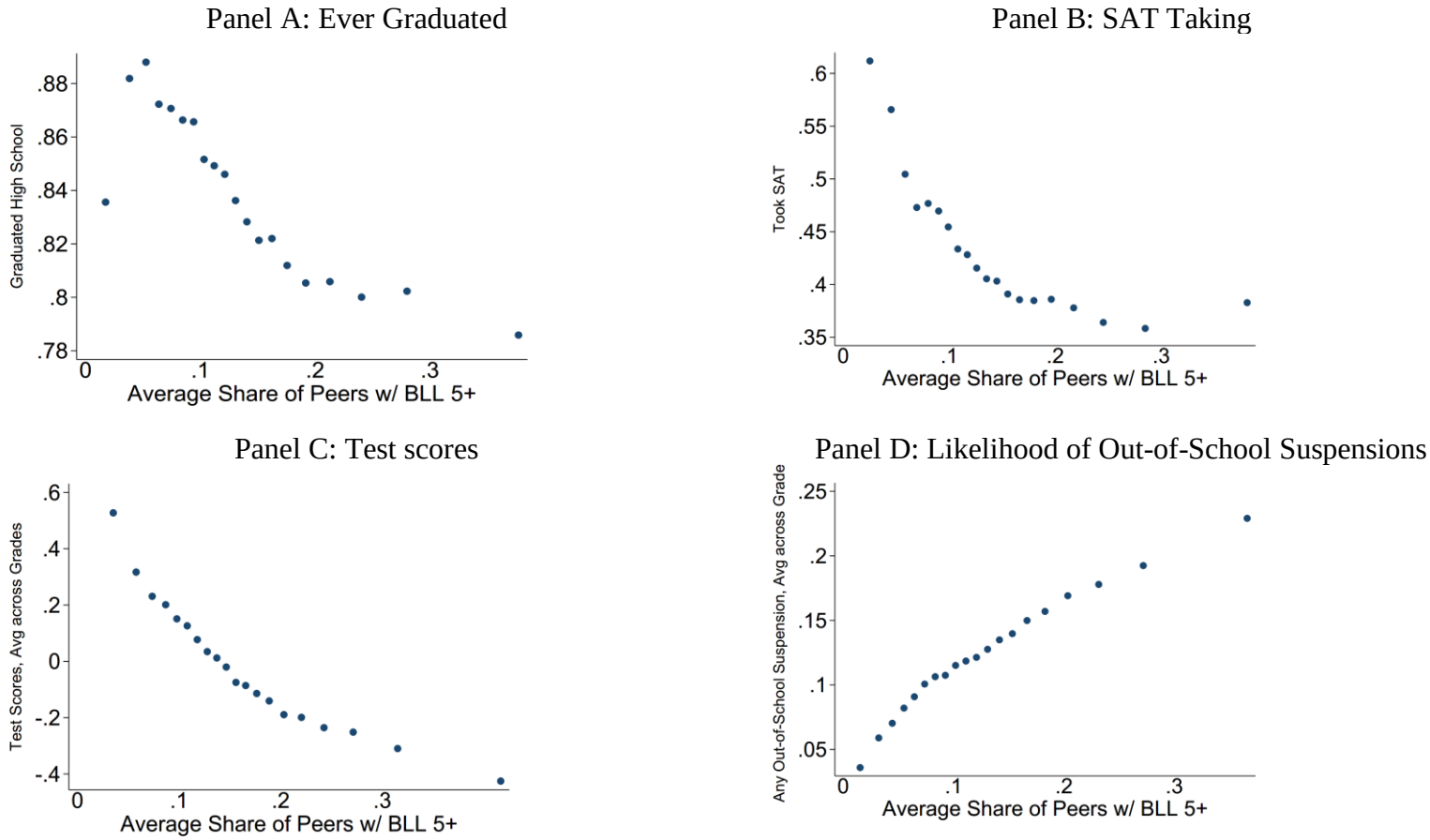
Notes: The figure plots the share of children in a school-grade-year cohort with at least one blood lead test (blue dashed line) and with a blood lead level of at least 5µg/dL (red solid line)

Figure 2: The Relationship Between a Child's Own Blood Lead Levels and Test Scores, Out-of-School Suspensions, High School Graduation, and SAT Taking



Notes: The figure plots graduation rates (Panel A), SAT taking rates (Panel B), average test scores (Panel C), and out-of-school suspension rates (Panel D) by students' blood lead levels and adds the line of best fit.

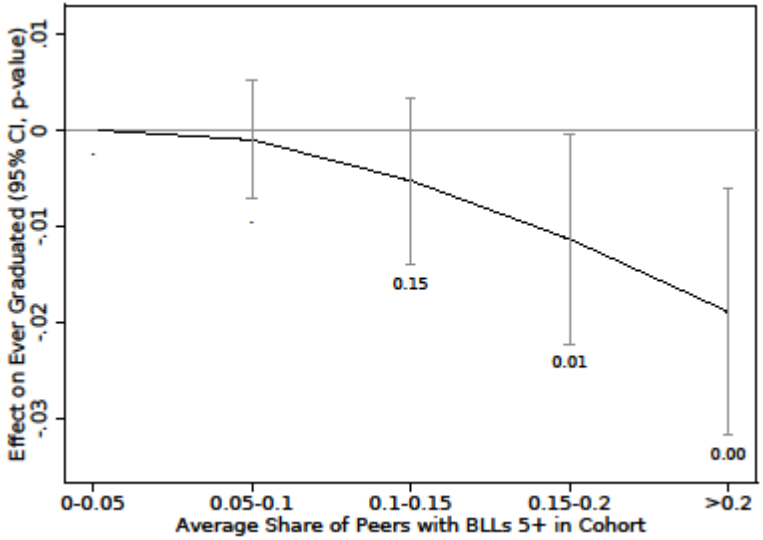
Figure 3: The Relationship Between Peers' Blood Lead Levels and Test Scores, Out-of-School Suspensions, High School Graduation, and SAT Taking



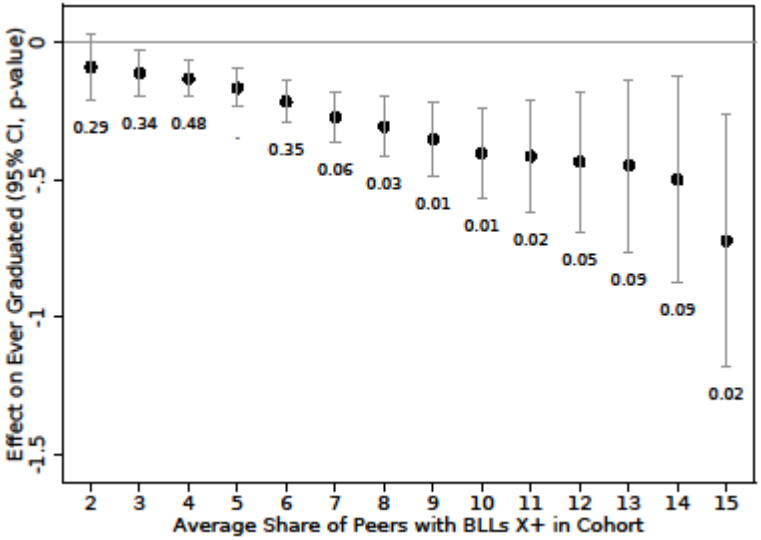
Notes: The figure plots graduation rates (Panel A), SAT taking rates (Panel B), average test scores (Panel B), and out-of-school suspension rates (Panel B) by vigintile of students' share of peers with blood lead levels at or above 5µg/dL.

Figure 4: Spillover Effects on Graduation by Binned Share of Peers with EBLL of 5+ µg/dL and by Different EBLL Thresholds

Panel A: Binned Effects of Share of Peers with Blood Lead Levels 5+ µg/dL



Panel B: The Effect of Peers with Different BLL Thresholds



Notes: Panel A plots non-parametric estimates of the effect of having different proportions (binned) of peers with BLLs 5+ in a child's cohort on the likelihood of high school graduation, where the omitted category is an indicator for share of peers with BLLs 5+ that is lower than 0.05. p-values for each bin relative to the 5-10 percent bin are shown under each confidence interval. Panel B plots the effect of the average share of peers with different BLLs of X+ in the cohort on graduation. As the BLLs increase, so does the negative effect on graduation. p-values relative to our baseline coefficient are shown under each confidence interval. In both figures, we control for all fixed effects and controls in our primary specification (which includes family, school, year, and grade fixed effects, and individual and demographic controls by cohort, averaged over elementary and middle school). Vertical bars represent 95% confidence intervals based on standard errors clustered at the school level.

ONLINE APPENDIX A: Additional Tables and Figures

Table A1: Results with School-grade and Grade-year Fixed Effects on the Sibling Sample and a Consistent Sample of Children for Whom We Observe Short and Long-run Outcomes

	Short-Run Outcomes		*Long-run Outcomes*	
	(1)	(2)	(3)	(4)
Dependent Variable:	Out of School Suspension	Absent 22+ Days	Ever Graduated	Took the SAT
Panel A: Results on a Consistent Sample of Children for Whom We Observe Short and Long-run Outcomes, Sibling Sample				
Share of peers with BLLs over 5 µg/dL	0.0549**	0.0218	-0.1119**	-0.2431**
	(0.0213)	(0.0133)	(0.0365)	(0.0780)
Observations	1,036,140	1,036,140	193,579	193,579
N Students	193,638	193,638	193,579	193,579
Mean of outcome	0.0691	0.0327	0.9515	0.5453
Panel B: Results with School-Grade and Grade-Year Fixed Effects on Sibling Sample				
Share of peers with BLLs over 5 µg/dL	0.0516*	0.0332*	-0.0927***	-0.2569***
	(0.0252)	(0.0131)	(0.0225)	(0.0526)
Observations	1,035,983	1,035,983	193,678	193,678
N Students	193,656	193,656	193,678	193,678
Mean of outcome	0.0690	0.0327	0.9517	0.5452

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes. The sample is limited to children with siblings who would be included in a regression with sibling fixed effects. In addition, Panel A further limits the sample to students for whom we observe all outcomes. Each column reports results from a separate regression. Regressions in Panels A and C include family, school, year, grade, birth order and birth month fixed effects Regressions in Panel B include school-by-grade, grade-by-year, and birth month fixed effects. All regressions include the cohort, school-level and individual controls listed in equation (1). Cohort and school controls are averaged over elementary and middle school in Columns 3 and 4. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A2: Contemporaneous Effects of Attending School with an Increased Share of Classroom Peers with Elevated BLLs

Dependent Variable:	(1) Out of School Suspension (OSS)	(2) Days Suspended	(3) OSS Same Day as Lead-Exposed Child	(4) Incident with Lead-Exposed Child	(5) Absent 22 or More Days
Share of peers with BLLs over 5 µg/dL	0.0755***	0.7258***	0.1234***	0.0895***	0.1381***
	(0.0087)	(0.1570)	(0.0080)	(0.0065)	(0.0079)
Observations	3,757,475	3,757,475	3,474,986	3,474,986	3,754,920
N Students	0.0944	0.6520	0.0318	0.0202	0.0509
Mean of outcome	879,806	879,806	827,909	827,909	886,511

Notes: The table reports the effect of a child's share of *classroom* peers with EBLLs on the child's school outcomes. We define peer exposure at the classroom level by averaging the number of peers with EBLLs across all classes a child takes in that year. We limit the sample to grades 6 and above in all columns. We do not report results on test scores due to data availability: we use test scores up to year 2006, while the classroom data is available only since 2007. All regressions include family, school, grade, and year fixed effects, and control for birth order. We control for individual and cohort characteristics, which include indicators for gender, race, economically disadvantaged status, whether the student has a blood lead level test, the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, the stability rate, and the percent of teachers with an MA degree. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A3: Heterogeneity by School-Level Demographics

Dependent Variable:	(1) Ever Graduated	(2) Took the SAT
Panel A: Schools in Lowest Tercile of Share Students who are Economically Disadvantaged		
Share of peers with BLLs over 5 µg/dL	-0.1265^{+}	-0.0924
	(0.0672)	(0.1657)
Panel B: Schools in Middle Tercile of Share Students who are Economically Disadvantaged		
Share of peers with BLLs over 5 µg/dL	-0.1441^{+}	-0.0642
	(0.0795)	(0.1462)
p-val = First Tercile	0.87	0.90
Panel C: Schools in Highest Tercile of Share Students who are Economically Disadvantaged		
Share of peers with BLLs over 5 µg/dL	-0.1074^{+}	-0.2025
	(0.0649)	(0.1357)
p-val = First Tercile	0.84	0.61
N Students	222,853	162,098
Mean of outcome	0.8964	0.5455

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes for children in schools with different shares of children who are economically disadvantaged in each panel. For each outcome, results are from a single regression. All regressions include cohort and individual controls, as well as family, birth month, birth order, school, grade, and year fixed effects. Individual controls include indicators for gender, race, economically disadvantaged status, and whether the student has a blood lead level test. Cohort controls include the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged. We also control for school size, the stability rate, and the percent of teachers with an MA degree. Cohort and school controls are averaged over elementary and middle school. Standard errors are clustered at the school level. + $p<0.10$, * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table A4: Potential Mechanisms: Homophily

Dependent Variable:	(1) Ever graduated	(2) Took the SAT
Panel A: By Same-Gender Lead-poisoned Peers		
Share of same-gender peers with BLLs ≥5 µg/dL	-0.1008^{+}	0.0326
	(0.0536)	(0.1294)
Share of peers with BLLs ≥5 µg/dL	-0.1166**	-0.2448*
	(0.0424)	(0.0975)
Panel B: By Same-Race Lead-poisoned Peers (White)		
Share of same-race peers with BLLs ≥5 µg/dL	0.0310	-0.1699
	(0.0581)	(0.1191)
Share of peers with BLLs ≥5 µg/dL	-0.1881***	-0.1124
	(0.0482)	(0.1110)
Panel C: By Same Gender-Race Lead-poisoned Peers (White)		
Share of same gender-race peers with BLLs ≥5 µg/dL	-0.0301	-0.0907
	(0.0610)	(0.1413)
Share of peers with BLLs ≥5 µg/dL	-0.1568***	-0.1971*
	(0.0373)	(0.0883)
N Students	282,964	201,713
Mean of outcome	0.8904	0.5320

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels and the *additional* effect of those lead-poisoned peers who share a characteristic (gender, race, or gender and race) on the child's school outcomes. Thus, the *total* effect of same-characteristic peers is obtained by summing up both coefficients in each panel. Panel A reports the effect of a child's share of same-gendered peers with elevated blood lead levels on the child's school outcomes, Panel B reports the reports the effect of a child's share of same-race peers with elevated blood lead levels, and Panel C reports the effect of a child's same-race and same-gender share of peers with elevated blood lead levels. All regressions include the cohort, school-level and individual controls listed in equation (2), as well as family, birth month, birth order, school, grade, and year fixed effects. Cohort and school controls are averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A5: Alternative Measures of Peers' BLLs and Alternative Sample Definitions Based on EBLL and Testing Status

	(1) Ever Graduated	(2) Took the SAT
Panel A: Share of Male and Female Peers with Max BLL at or above 5 µg/dL		
Share of male peers with BLLs over 5 µg/dL	-0.1489**	-0.2523*
	(0.0454)	(0.1021)
Share of female peers with BLLs >= 5 µg/dL	-0.1861***	-0.1942+
	(0.0467)	(0.1066)
Panel B: Peers' Mean BLL is at or above 5 µg/dL		
Share of peers with BLLs over 5 µg/dL	-0.2080***	-0.2313**
	(0.0352)	(0.0740)
Panel C: Using the Earliest Observed Grade Share of Peers with Max BLL at or above 5 µg/dL		
Earliest observed share of peers with BLLs over 5 µg/dL	-0.0723***	-0.0838*
	(0.0196)	(0.0410)
Panel D: Highest Share of Peers with Max BLL at or above 5 µg/dL across Elementary and Middle School		
Max share of peers with BLLs at or above 5 µg/dL over grades 3-8	-0.1160***	-0.1571**
	(0.0232)	(0.0490)
N Students	282,964	201,713
Mean of outcome	0.8904	0.5320
Panel E: Including Only Tested Students with Blood Lead Levels <5 µg/dL		
Share of peers with BLLs over 5 µg/dL	-0.1941***	-0.1783+
	(0.0533)	(0.0975)
N Students	83,861	67,160
Mean of outcome	0.8669	0.4298
Panel F: Including Students with Elevated Blood Lead Levels		
Share of peers with BLLs over 5 µg/dL	-0.1768***	-0.2032***
	(0.0327)	(0.0595)
N Students	361715	257132
Mean of outcome	0.8771	0.5035

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes using different measures of peer exposure based on blood lead levels. Panel A uses the share of male and share of female peers with maximum BLL at or above 5 µg/dL *separately*. Panel B uses the share of peers with average BLL at or above 5 µg/dL. Panel C uses the earliest observed share of cohort of peers with maximum BLL at or above 5 µg/dL. Panel D uses the highest share of a student's peers across elementary and middle school with maximum BLL at or above 5 µg/dL. Panel E includes children who have maximum BLL at or above 5 µg/dL. Panel F only includes children who are tested and have maximum BLL below 5 µg/dL. All regressions include cohort and individual controls, as well as family, birth month, birth order, school, grade, and year fixed effects. Individual controls include indicators for gender, race, economically disadvantaged status, and whether the student has a blood lead level test. Cohort controls include the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged, school size, the stability rate, and the percent of teachers with an MA degree. Cohort and school controls are averaged over elementary and middle school. Standard errors are clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A6: Effects of Having an Elevated BLL on Peers' Average Graduation Outcomes Using Block group-by-Birth Year Fixed Effects and Family Fixed Effects

	(1)	(2)	(3)	(4)	(5)
	Block Group-By-Birth Year Fixed Effects				Family Fixed Effects
	No controls	Individual controls	Individual, parent, and family composition controls	Individual, parent, family composition, and school controls	Individual and school controls
Panel A: Tested by 72 months of age					
Child's maximum BLL is >=5	-0.0013***	-0.0010**	-0.0010**	-0.0010**	-0.0002
	(0.0003)	(0.0003)	(0.0003)	(0.0003)	(0.0007)
Observations	338,232	338,232	338,232	338,232	114,602
Children with Within-Group Variation in BLLs	304,647	304,647	304,647	304,647	45,963
Mean of outcome	0.6153	0.6153	0.6153	0.6153	0.6225
Panel B: Tested by 37 months of age					
Child's maximum BLL is >=5	-0.0016***	-0.0012**	-0.0012**	-0.0011**	-0.0017+
	(0.0004)	(0.0004)	(0.0004)	(0.0004)	(0.0009)
Observations	225,382	225,382	225,382	225,382	61,178
Children with Within-Group Variation in BLLs	193,448	193,448	193,448	193,448	24,330
Mean of outcome	0.6217	0.6217	0.6217	0.6217	0.6284
Panel C: Tested by 25 months of age					
Child's maximum BLL is >=5	-0.0014**	-0.0009+	-0.0010*	-0.0009+	-0.0021*
	(0.0005)	(0.0005)	(0.0005)	(0.0005)	(0.0010)
Observations	190,155	190,155	190,155	190,155	46,156
Children with Within-Group Variation in BLLs	159,908	159,908	159,908	159,908	18,262
Mean of outcome	0.6226	0.6226	0.6226	0.6226	0.6297

Notes: The table reports the effect of one additional child with elevated blood lead levels on the average graduation outcomes for their peers (equation 5). All specifications control for the share of peers that have EBLLs, the share of children with a lead test, as well as school, grade, birth month and birth order fixed effects. Column 1 reports the effects on the likelihood peers ever graduate from high school with no additional controls. Column 2 adds individual controls for gender, race, and economic disadvantage. Column 3 additionally controls for whether the mother has a high school degree, the number of children in the family and the gender mix of any siblings. Column 4 adds school controls (the share of peers that have EBLLs, the share of peers that are non-White or economically disadvantaged, school size, the stability rate, and the percent of teachers with an MA degree). Column 5 controls for individual and school characteristics and family fixed effects instead of block group-by-birth year effects. We report the number of children who contribute to variation in BLLs at the block group-by-birth year level (columns 1-4) and the number of siblings with different BLLs at the family level (column 5). Panel A uses all children with a blood lead test in our sample. Panels B and C limit the sample to children screened by 37 and 25 months of age, respectively. Standard errors are in parentheses and clustered at the school level. $^{+}$ $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A7: Long-run Outcomes of Exposure to Peers with Elevated BLLs Who Are Tested by 25 or 37 Months of Age

	(1) Ever graduated	(2) Took the SAT
	Panel A: Tested by 25 Months	
Share of peers with BLLs over 5 µg/dL	-0.1896***	-0.1056
	(0.0452)	(0.0912)
Observations	272,800	201,448
Mean of outcome	0.8925	0.5320
	Panel B: Tested by 37 Months	
Share of peers with BLLs over 5 µg/dL	-0.1823***	-0.1212
	(0.0391)	(0.0820)
Observations	278,120	201,668
Mean of outcome	0.8916	0.5320

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels, measured by 25 or 37 months of age, on the child's long-run outcomes. Control variables are measured in the highest grade a student is observed in. Column 1 reports the effects on the likelihood a student ever graduates from high school, and Column 2 shows the effects on the likelihood of ever taking the SAT. All regressions include individual controls for sibling, school, grade, year, birth month and birth order fixed effects, gender, race, economically disadvantaged status, and whether the student has a blood lead level test. We also control for the average share of elementary and middle school peers that are non-White or economically disadvantaged, average share of children with a lead test, school size, the stability rate, and the percent of teachers with an MA degree averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. $^{+}$ $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$

Table A8: Correlations between being tested for lead and child and school characteristics

	(1) Parental education	(2) Female	(3) Nonwhite	(4) Economically Disadvantaged	(5) Share of teachers with Masters or higher, by school-year	(6) School-Year Stability Rate
Was Tested for Lead	-0.0145	0.0168*	-0.0004	0.0017	0.0008	-0.0002
	(0.0166)	(0.0077)	(0.0015)	(0.0038)	(0.0006)	(0.0002)
Observations	159,974	193,778	193,778	193,778	182,586	182,586
Mean of outcome	4.8527	0.4911	0.3585	0.3093	0.3343	0.9580

Notes: This table reports the correlation between being tested for lead and various characteristics of the child, family and school they attend, conditional on our main controls and family, school, grade, year, birth month and birth order fixed effects. Each cell represents the output from a different regression. Standard errors are in parentheses and clustered at the school level. $^{+}$ $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$

Table A9: Results for Alternative Samples and Alternative Specifications, Additional Outcomes

	(1) >50% of Homes in Census Tract are Single Family	(2) Zip Codes with Universal Screening	(3) Adding Cohort Block Group Characteristics	(4) Adding Block Group Fixed Effects	(5) Adding School-Grade Fixed Effects	(6) Adding School-Year Fixed Effects	(7) Zip Codes with No School Choice Options	(8) Adding Sibling-School Fixed Effects
				Panel A: Took the SAT				
Share of peers with	-0.1293	-0.1675^{+}	-0.2165**	-0.2179^{+}	-0.2198**	-0.1505^{+}	-0.2234*	-0.2139***
BLLs over 5 µg/dL	(0.1352)	(0.0985)	(0.0743)	(0.1232)	(0.0744)	(0.0884)	(0.0891)	(0.0632)
N Students	60,267	100,744	201,711	68,577	201,590	201,071	123,761	164,721
Mean of outcome	0.5106	0.5145	0.5320	0.5922	0.5321	0.5330	0.5134	0.5461
				Panel B: Out-of-School Suspensions				
Share of peers with	0.0347^{+}	0.0277^{+}	0.0290*	0.0414*	0.0138	0.0218*	0.0056	0.0274*
BLLs over 5 µg/dL	(0.0210)	(0.0162)	(0.0118)	(0.0168)	(0.0118)	(0.0103)	(0.0461)	(0.0128)
N Students	259,394	426,200	944,147	607,022	944,330	944,327	267,262	911,921
Mean of outcome	0.1053	0.1081	0.0942	0.0985	0.0942	0.0942	0.1311	0.0887
				Panel C: Absent 22+ Days				
Share of peers with	0.0688***	0.0472***	0.0442***	0.0568***	0.0400***	0.0549***	0.0305**	0.0302***
BLLs over 5 µg/dL	(0.0130)	(0.0093)	(0.0070)	(0.0093)	(0.0073)	(0.0070)	(0.0104)	(0.0079)
N Students	254,224	425,393	939,199	597,413	939,425	939,422	510,563	907,237
Mean of outcome	0.0543	0.0601	0.0520	0.0494	0.0520	0.0520	0.0503	0.0485
School FEs	X	X	X	X			X	
Sibling FEs	X	X	X	X	X	X	X	
Year FEs	X	X	X	X	X		X	X
Grade FEs	X	X	X	X		X	X	X

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes. Each column reports results from a separate regression. Column 1 restricts the sample to Census tracts where more than half of homes are single family homes. Column 2 restricts the sample to students who live in zip codes that are subject to universal lead screening. Columns 3-6 and 8 add controls and alternative sets of fixed effects as specified at the top and bottom of each column. Column 7 restricts the sample to zip codes without charter schools or voucher programs. Block group characteristics of cohort peers include share of peers that live in block groups with above median income, above median percent Black and Hispanic population, above median percent of the population living in poverty, and with above median percent population with a high school degree. All regressions include cohort and individual controls, as well as birth month and birth order fixed effects. Individual controls include indicators for gender, race, economically disadvantaged status, and whether the student has a blood lead level test. Cohort controls include the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, the stability rate, and the percent of teachers with an MA degree. In Panel A, cohort and school controls are averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A10: Long-run Outcomes of Exposure to Peers with Elevated BLLs, Accounting for Race and Socioeconomic Status

	(1) Ever graduated	(2) Took the SAT
	Panel A: White Children with EBLLs Only	
Share of White peers with BLLs over 5 µg/dL	-0.1376*	-0.0925
	(0.0546)	(0.0913)
Observations	283,032	201,784
Mean of outcome	0.8904	0.5319
	Panel B: Wealthier Children with EBLLs Only	
Share of less-poor peers with BLLs over 5 µg/dL	-0.1467***	-0.2694**
	(0.0386)	(0.0889)
Observations	283,032	201,784
Mean of outcome	0.8904	0.5319
	Panel C: Additional Control for Peers' Parental Education	
Share of peers with BLLs over 5 µg/dL	-0.1538***	-0.2236**
	(0.0382)	(0.0854)
Share of peers with parents with high school diploma or less	-0.0834***	0.0233
	(0.0113)	(0.0300)
Observations	250,392	167,310
Mean of outcome	0.8814	0.5416

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels and higher socioeconomic status on the child's long-run outcomes (Panels A and B) and controlling for peers' parental education (Panel C). Control variables are measured in the highest grade a student is observed in. Column 1 reports the effects on the likelihood a student ever graduates from high school, and Column 2 shows the effects on the likelihood of ever taking the SAT. Panel A estimates the effects using only white children with EBLLs. Panel B instead includes only children with EBLLs not consistently listed as economically disadvantaged. All regressions include individual controls for sibling, school, grade, year, birth month and birth order fixed effects, gender, race, economically disadvantaged status, and whether the student has a blood lead level test. We also control for the average share of elementary and middle school peers that are non-White or economically disadvantaged, average share of children with a lead test, school size, the stability rate, and the percent of teachers with an MA degree averaged over elementary and middle school. Panel C further controls for the share of peers with parents with high school diploma or less. Standard errors are in parentheses and clustered at the school level. $^{+}$ $p < .1$, * $p < .05$, ** $p < .01$, *** $p < .001$

Table A11: Results with Different Sets of Controls

Dependent Variable:	(1) Ever Graduated	(2) Took the SAT
Panel A: Controlling for Share of Cohort Peers Tested for Lead		
Share of peers with BLLs over 5 µg/dL	-0.1749***	-0.2315**
	(0.0344)	(0.0727)
N Students	282,964	201,713
Panel B: All Controls Except for Share Non-White and Share Economically Disadvantaged		
Share of peers with BLLs over 5 µg/dL	-0.1614***	-0.2147**
	(0.0340)	(0.0721)
N Students	282,964	201,713
Panel C: Family Fixed Effects		
Share of peers with BLLs over 5 µg/dL	-0.1658***	-0.2334**
	(0.0375)	(0.0739)
N Students	283,168	201,747
Panel D: School-Specific Time Trend		
Share of peers with BLLs over 5 µg/dL	-0.1454***	-0.1995**
	(0.0375)	(0.0768)
N Students	282,964	201,713
Panel E: Adding Controls for Family Composition		
Share of peers with BLLs over 5 µg/dL	-0.1638***	-0.2234**
	(0.0349)	(0.0740)
N Students	282,964	201,713

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes. Each cell reports results from a separate regression. All regressions include sibling, birth month, grade, year and birth order fixed effects. Panel A shows our results with no control variables except for the share tested for lead, school fixed effects, and the above-mentioned fixed effects. Panel B includes school fixed effects and controls for gender, race, economically disadvantaged status, whether the student has a blood lead level test, the average share of peers with a lead test, as well as average school size, stability rate, and percent of teachers with an MA degree. We omit average cohort-level controls for share of non-White peers and share of peers who are economically disadvantaged. Panel C includes our fixed effects together with all controls in our main specification but omits school fixed effects. Panel D includes our fixed effects, school fixed effects, and all controls, and adds controls for year-specific sibling gender composition and family size. Panel E includes our fixed effects, school fixed effects, and all controls, and adds school-specific linear time trends. Cohort and school controls are averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. + $p<0.10$, * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table A12: Correlation of Cohort Composition and Measures of School Quality

Dependent Variable:	(1) Share of teachers with Masters or higher in school-year	(2) School-year stability rate	(3) Missing teachers' education in school-year	(4) Missing school-year stability rate	(5) Number of students in school-year
Share of peers with	-0.0105	0.0047*	0.0040	-0.0002	126.6287***
BLLs over 5 µg/dL	(0.0077)	(0.0020)	(0.0035)	(0.0003)	(22.1455)
Observations	7,604,803	7,752,784	7,752,854	7,752,854	7,752,854
Mean ofoutcome	0.3656	0.9576	0.0190	0.0000	782.6351
N Students	1,171,271	1,177,602	1,177,603	1,177,603	1,177,603

Notes: The table reports the correlation of a child's share of peers with EBLLs in a cohort with school-year characteristics. Regressions include family, school, grade, and year fixed effects, controlling for birth order. All regressions control for individual and cohort controls, which include indicators for gender, race, economically disadvantaged status, whether the student has a blood lead level test, the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, the stability rate, and the percent of teachers with an MA degree when those are not the dependent variable. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A13: The Effects of Peers on Switching Schools

	(1) Changed Schools	(2) Changed to a Charter School	(3) Child's Sibling Changed Schools	(4) Both Siblings Changed Schools	(5) Consecutive Younger Sibling is in a Different School for the Same Grade
Share of peers with BLLs over 5 µg/dL	0.0001	-0.0038*	-0.0063	-0.0085	-0.0323*
	(0.0131)	(0.0015)	(0.0227)	(0.0138)	(0.0149)
Observations	6,548,425	6,446,798	1,574,485	838,558	4,188,008
N Students	1,094,634	1,089,308	491,532	309,454	586,937
Mean of outcome	0.3063	0.0050	0.3465	0.1308	0.1855

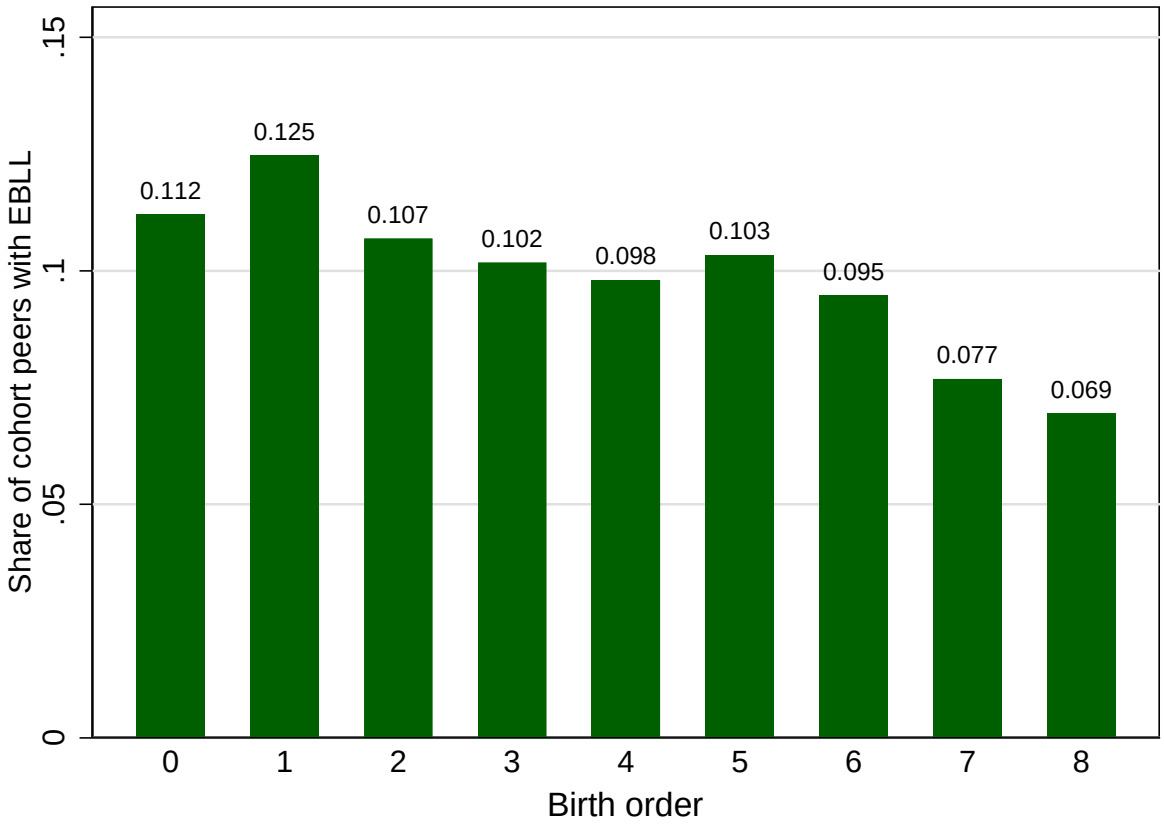
Notes: The table reports the association of a child's share of peers with elevated blood lead levels with the child's own likelihood of switching schools (Columns 1 and 2), the child's sibling's likelihood of switching schools conditional on attending the same school (Column 3), both children switching schools conditional on attending the same school (Column 4), and the likelihood that a consecutive younger sibling attends a different school than the child's school for the same grade (Column 5). All regressions include cohort and individual controls, as well as family, birth month, birth order, school, grade, and year fixed effects. Individual controls include indicators for gender, race, economically disadvantaged status, and whether the student has a blood lead level test. Cohort controls include the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, the stability rate, and the percent of teachers with an MA degree. Standard errors are clustered at the school level. + $p<0.10$, * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table A14: Controlling for Sibling's Peers' Lead Exposure

Dependent Variable:	(1) Ever graduated	(2) Took the SAT
Share of peers with BLLs over 5 µg/dL	-0.1286*	-0.1771
	(0.0516)	(0.1162)
Share of peers with BLLs over 5 µg/dL, Sibling 1	0.0283	0.0117
	(0.0504)	(0.1113)
Share of peers with BLLs over 5 µg/dL, Sibling 2	0.0528	0.1706+
	(0.0409)	(0.1000)
Share of peers with BLLs over 5 µg/dL, Siblings 3-6	0.0578	0.0336
	(0.0881)	(0.1952)
Mean of outcome	0.8904	0.5320
N Students	282,964	201,713

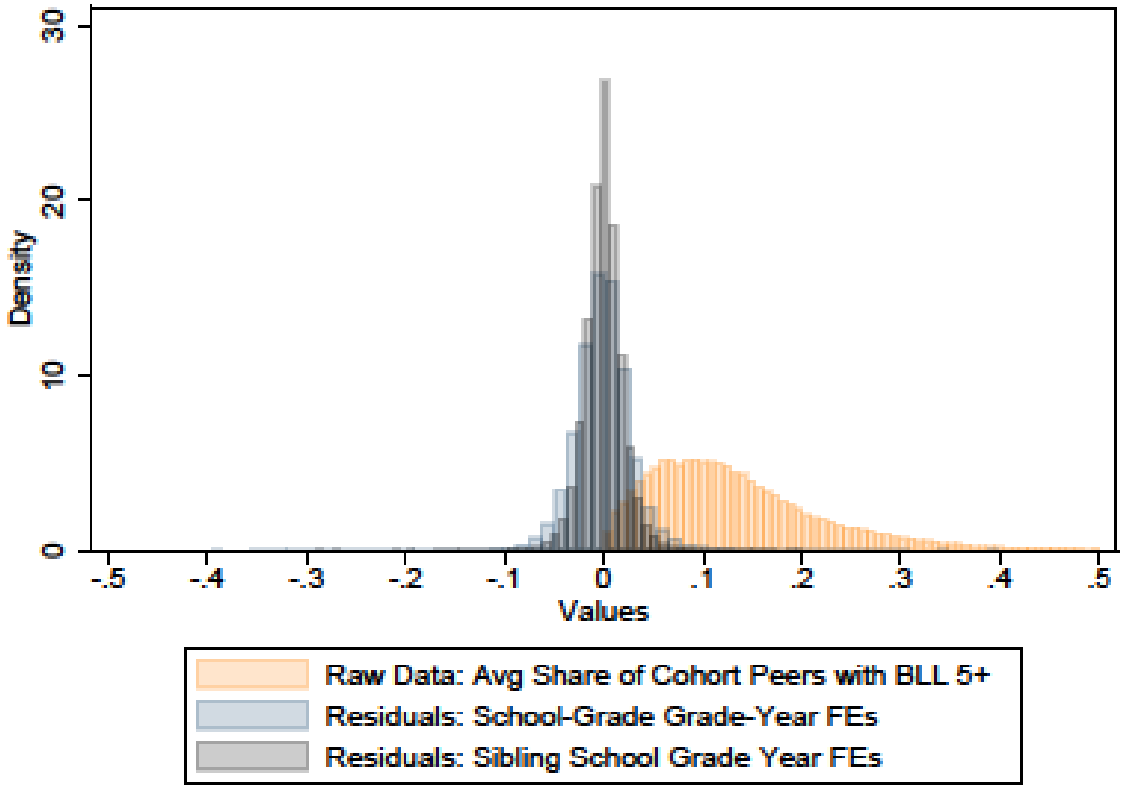
Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's long-run outcomes controlling for the share of peers with elevated blood lead levels of the child's siblings. For children with only one sibling, the share of peers with elevated blood lead levels of the child's second sibling and the average for siblings 3-6 are set to 0 and family fixed effects absorb the effect of family size. Regressions include family, school, grade, and year fixed effects, controlling for birth order. All regressions control for individual and cohort controls, which include indicators for gender, race, economically disadvantaged status, whether the student has a blood lead level test, the share of non-White peers, share of children with a lead test, and the share of peers who are economically disadvantaged. We also control for school size, the stability rate, and the percent of teachers with an MA degree when those are not the dependent variable. Cohort and school controls are averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Figure A1: Share of Lead-Exposed Peers by Birth Order



Notes: This figure plots the average share of cohort peers with blood lead levels at or above 5µg/dL by a student's birth order. Birth order is set to 0 for only children and children for which we are not able to match siblings.

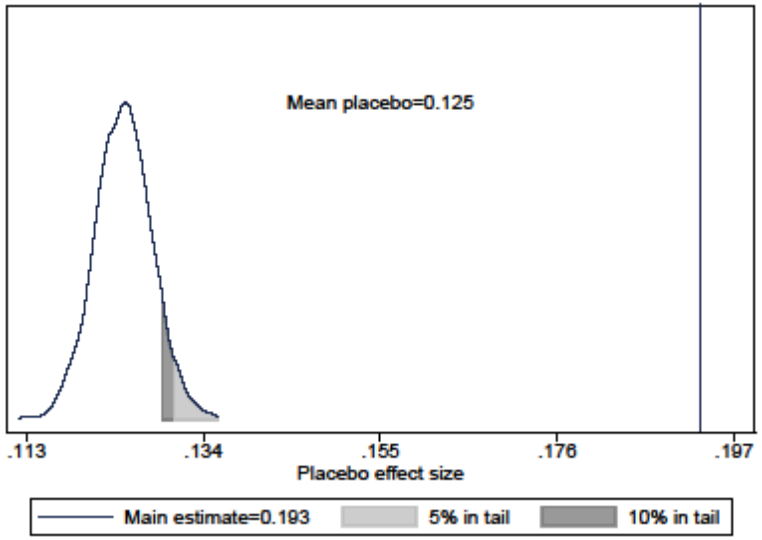
Figure A2: Identifying Variation: Residual Variation in Share of Peers with Elevated Blood Lead Levels



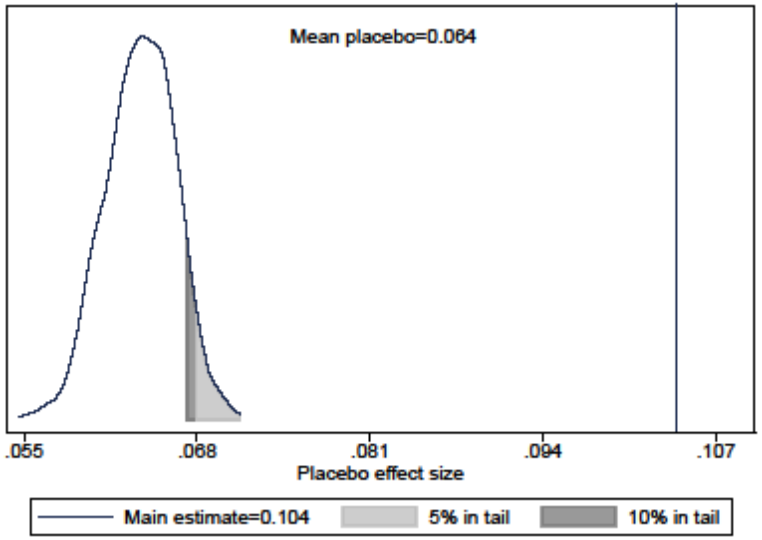
Notes: This figure plots the distribution of the residuals from a regression of our variable of interest, the average share of peers with blood lead levels at or above 5µg/dL over elementary and middle school on the fixed effects and controls included in our preferred specification. We include family, birth month, birth order, grade, school, and year fixed effects. Individual controls include indicators for gender, race, economically disadvantaged status, and whether the student has a blood lead level test. Cohort controls include the average share of non-White peers, average share of children with a lead test, and the average share of peers who are economically disadvantaged at the school-grade-year level. We also control for average school size, the average stability rate, and the average percent of teachers with an MA degree. The black solid line plots the kernel density of these residuals, while the blue dashed line plots a normal distribution.

Figure A3: Placebo Estimates: Out-of-School Suspensions and Incidents with Students with EBLLs

Panel A: OSS Same Day as Student with EBLL

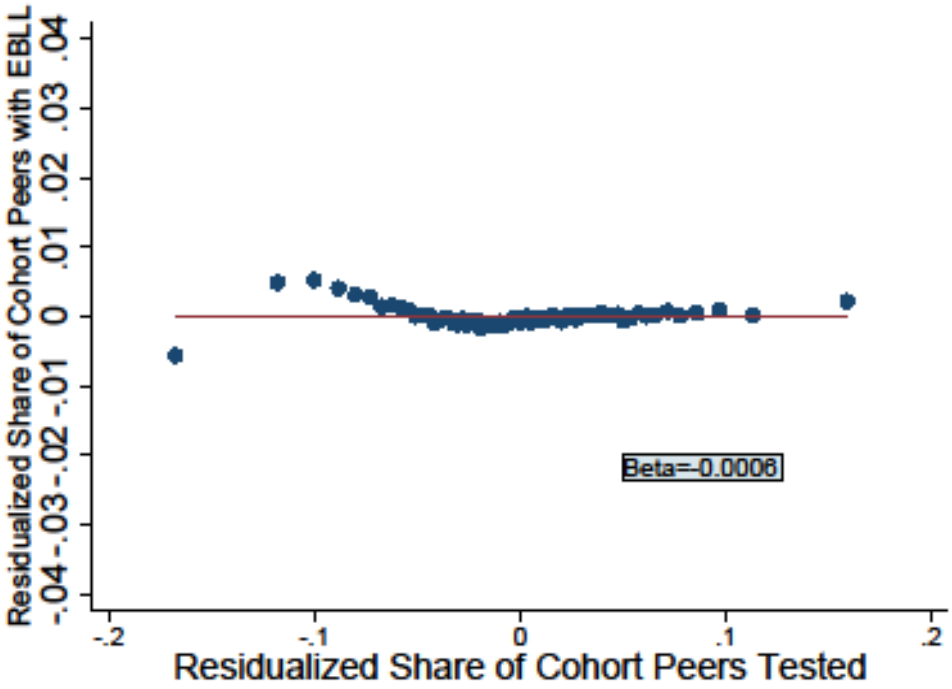


Panel B: Incident with Student with EBLL



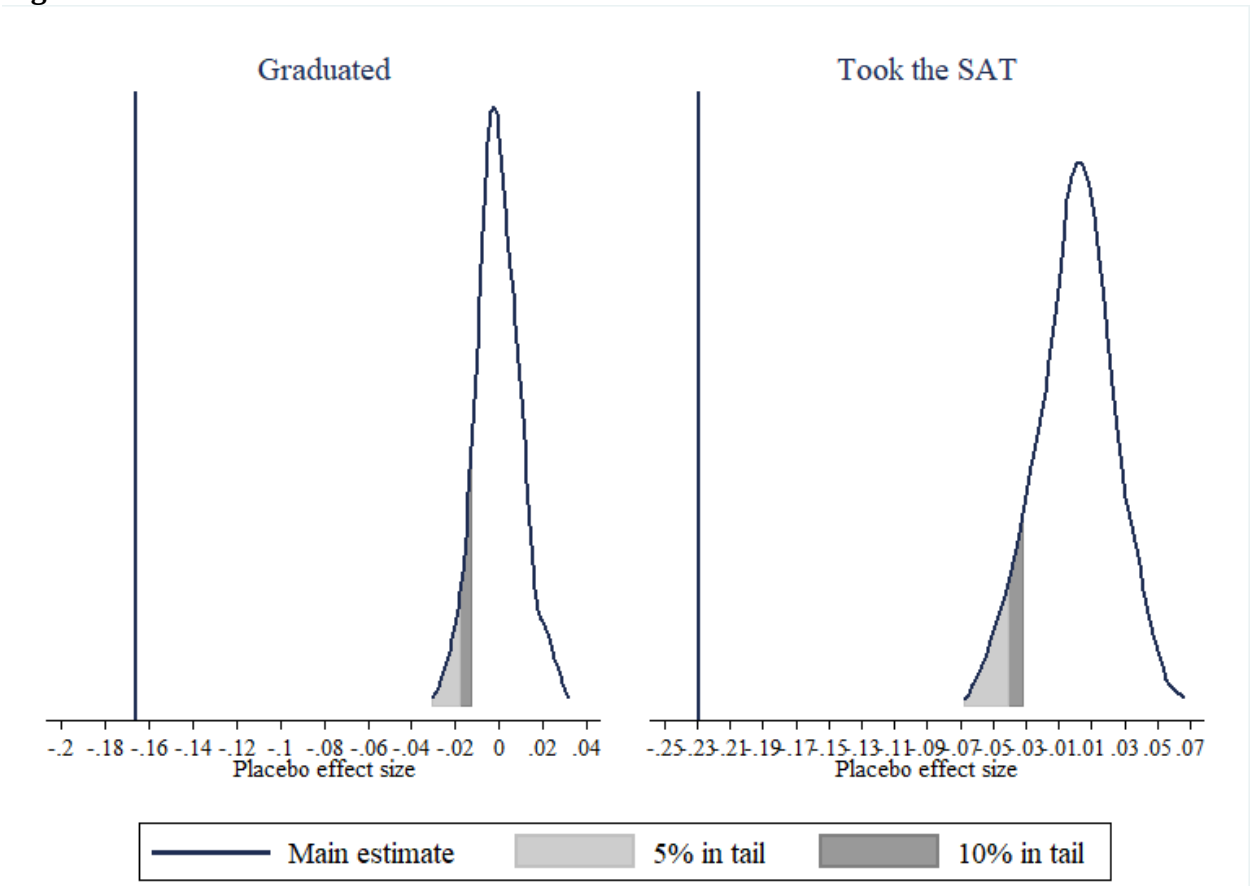
Notes: Distribution of results from 500 placebo tests per outcome. Our main estimates for our preferred specification are represented with a vertical line on the placebo effect size distribution. The lightly shaded gray region is the region of the graph where there is 5% in the tail of the distribution. The darker shaded gray region represents 10% in the tail of the distribution. For each placebo, we randomly selected a share of student equal to the observed share of students in EBLLs in that school-grade cohort and construct indicators for their peers being suspended out-of-school on the same day as one of these random students and having an incident together with one of these random students. We then estimated our main specification with these placebo outcomes

Figure A4: Share of Cohort Peers with EBLLs by Share of Cohort Peers Who Are Tested for Lead



Notes: This figure plots the residualized share of cohort peers with EBLLs over the share of cohort peers who were tested for lead (by quintiles) and adds the line of best fit. The residuals are obtained by regressing the share of cohort peers with EBLLs on individual controls for sibling, school, grade, year, block group, birth month and birth order fixed effects, gender, race, and economically disadvantaged status. We also control for the share of school-cohort peers that are non-White or economically disadvantaged, school size, the stability rate, and the percent of teachers with an MA degree.

Figure A5: Placebo Estimates



Notes: Figure A5 shows the distribution of results from 500 placebo tests per outcome. Our main estimates for our preferred specification are represented with a vertical line on the placebo effect size distribution. The lightly shaded gray region is the region of the graph where there is 5% in the tail of the distribution. The darker shaded gray region represents 10% in the tail of the distribution. For each placebo, school-grade cohorts were randomly assigned a percent of peers with EBLLs from the empirically observed distribution and we estimated our main specification.

Appendix B: Data Appendix
B1. Data linkage

The North Carolina Education Research Data Center (NCERDC) performed the linkage between the education and BLL data according to the following algorithm and anonymized the dataset for us. Table B1 reports the number of tests matched at each step.

1. Exact match on school district, that is local education agency (LEA), or county and first and last name, date of birth.
2. Exact match on first and last name, date of birth
3. Exact match on LEA or county and first and last name, but allow for mistakes in one of day, month, or year of birth
4. Exact match on LEA or county, last name, and date of birth, allow for close first name or nickname
5. Exact match on LEA or county, first name, and date of birth, allow for close last name
6. Exact match on last name, date of birth, allow for close first name or nickname
7. Exact match on first name, date of birth, allow for close last name
8. Exact match on first and last name, but allow for mistakes in one of day, month, or year of birth
9. Exact match on first and last name

Table B1: Match Results

(1) Match Step	(2) Number of Tests	(3) Share
1	1,352,623	0.606457
2	431,987	0.193684
3	24,098	0.010804
4	104,751	0.046966
5	190,154	0.085257
6	32,860	0.014733
7	44,963	0.020159
8	5,168	0.002317
9	43,765	0.019622

Notes: This table reports the additional number of tests matched at each step. Column 1 reports the match step, Column 2 reports the number of standardized tests, and Column 3 reports the share of children with each of these.

B2. Sibling Identification Algorithm

In this data appendix, we describe the algorithm used to identify siblings using students' geocoded home addresses.

There are 4.38 million unique students in the NCERDC data. Of these, about 740,000 do not have a home address and another 640,000 do not have birthday information. Since both home addresses and birthdays are crucial for identifying siblings, we drop these observations when running the linkage algorithm. We also ignore about 660,000 students who never share a home address with another student and therefore do not have siblings in our data.

We further restrict our sample to include home addresses with at most four students in any given year. We do this for several reasons. First, the geocoded address variable provided by NCERDC is based on street address and does not distinguish between different units that share the same street address. This means that students living in different apartment units within the same building appear to be living at the same home address. Because of this, we observe addresses with hundreds of students in a given year, and it is implausible that these students are siblings. Second, we observe that students who share a geocoded address with many other students often move across addresses. We suspect some of these students are in the foster care system and therefore it is difficult to identify their siblings with certainty. Three, according to the 2000 Census, the average number of children per family in North Carolina is 1.75, and thus we are conservative in limiting the number of children living together in any given year to at most four. Four, the algorithm speed is decreasing in the number of students living together in any given year. Thus, we apply our algorithm to addresses with no more than four students in a given year. This selection eliminates about 211,000 students, 80,000 of which always share an address with at least four other children.

We are left with about 2.12 million students on which we run the sibling identifying algorithm. The following steps summarize the process:

1. Identify all students who live together at any point or could be living together by transitivity and assign a tentative family identifier to these students. For example, Ana and Bob are observed living together in some years, Bob and Claire are observed living together in other years, but Ana and Claire are never observed living together. We temporarily assign Ana, Bob, and Claire to the same family.

2. For each potential sibling pair within the temporary families, check if the students are ever observed living at different addresses in the same year and if they are born between 2 and 240 days of each other. That is, we allow students to be born on the same or consecutive days to account for twins. If at least one of these holds, the students cannot be siblings. This step produces a dummy variable for each student within the temporary family that equals 1 whenever another student within the temporary family is a potential sibling, and zero otherwise. Table B2 shows a simple scenario for a tentative family with three students where all three can be siblings to one another. In such cases, we assign the temporary family a permanent household identifier.

Table B2

Student	Student1	Student2	Student3
1	1	1	1
2	1	1	1
3	1	1	1

Table B3

Student	Student1	Student2	Student3
1	1	1	0
2	1	1	1
3	0	1	1

3. Table B3 shows a tentative family where not all students can be siblings to one another: student 1 could be a sibling to student 2 but not to student 3, while student 2 could be a sibling to both students 1 and 3. Based on the indices, we conclude there are two potential true sibling groups: either students 1 and 2 are siblings, or students 2 and 3 are siblings. For each potential sibling group, we calculate a score based on the number of years students live together, the number of students in the subgroup, and the span of years for which the students are observed. Specifically:

$$score_g = \frac{\left(\sum_y \sum_{i,j \in g} \mathbb{I}_{j \neq i,y}\right)^2}{N_g} + \frac{\sum_y \sum_{i,j \in g} \mathbb{I}_{j \neq i,y}}{N_y}$$

where *i* and *j* denote students in subgroup *g*, and *y* denotes year. $\mathbb{I}_{j \neq i,y}$ equals 1 if student *i* and student *j* are observed living together in year *y*. $\sum_y \sum_{i,j \in g} \mathbb{I}_{j \neq i,y}$ equals the number of times students in the subgroup live with each other, allowing for double counting. N_g denotes the number of students in subgroup *g*. N_y the is the difference between the first and last year subgroup *g* is observed. For example, if a subgroup is first observed living together in 2000 and last observed in 2005, N_y equals 5. The first term of the index gives more weight to subgroups where students are observed living together more often per student. The second term gives more weight to subgroups observed living together in

consecutive years as opposed to many years apart. The subgroup with the highest score is assigned a permanent family identifier, and the step is repeated until all students in the temporary family are assigned a family identifier.

Table B4 shows the distribution of children across family size produced by our algorithm. Almost half of the children have only one sibling (columns 2 and 3), and about 84 percent of families have at most two children (column 5). Dividing the total number of children by the total number of families gives an average number of children per family of 1.80, which is similar to the figure provided by the Census.

Table B4: Distribution of children across family size

(1) Family size	(2) # of children	(3) % of children	(4) # of families	(5) % of families
1	457,796	21.56%	457,796	38.97%
2	1,054,842	49.68%	527,421	44.90%
3	458,760	21.61%	152,920	13.02%
4	127,036	5.98%	31,759	2.70%
5	19,960	0.94%	3,992	0.34%
6	3,798	0.18%	633	0.05%
7	791	0.04%	113	0.01%
8	144	0.01%	18	0.00%
9	45	0.00%	5	0.00%
Total	2,123,172	100%	1,174,657	100%

B3. Sample Selection and Variable Construction

In describing the sample, we refer to the year of the spring semester (e.g., 2000 refers to academic year 1999-2000).

Sample selection criteria*:* The raw data include all children who attended public school, including charter schools, between 1997 and 2017. Because our blood lead level data begin in 1992 and children are tested up to age 6, we only have blood lead level data for children born after 1986. Thus, we restrict the sample to children born after 1986. Since the raw birth year data have some error, we impute birth year based on year and grade, and assume students enter first grade by age 7.[37] Our full sample includes 3.3 million children with non-missing student identifier, and is described in Column 1 of Table 1 in the main text. All these children are used to

[37] Our results are robust to using birth year or other ages instead.

calculate standardized test scores, cohort and class size, as well as the percentages of EBLLs, ED students, and non-White students by cohort.

In our analyses, we restrict the sample to children for whom we have, at minimum, information on birthday, grade, and school attended in a given year, as well as at least one of the outcomes of interest (except for column 1 if Table 1 in which we include all children). We also impose several sample restrictions on our outcome variables (described below) which result in fewer unique student observations in our analyses samples. Using the family identifier constructed using our sibling matching algorithm described in Data Appendix B2, we further restrict our main analysis sample to siblings. Our main analysis sample has 1.3 million children and is described in Column 2 of Table 1.

The final sample for our short-run analysis is a panel with unique student observations at the grade and year level. For our long-run analysis, we further collapse the data to one observation per student by averaging all covariates over grades 3-8.

Lead exposure definition*:* Capillary tests are more prone to false positives than venous tests. Thus, to identify lead poisoned children we use the highest venous test result if available, and the highest capillary test result if no venous test was performed.

Our main explanatory variable is the share of peers that have elevated blood lead levels. We construct two measures of peer exposure, one at the cohort---that is school-grade-year level,---and one at the classroom level. The share of *cohort* peers that have elevated blood lead levels equals the number of cohort peers that have known EBLL, divided by the number of children in the cohort (excluding oneself). NCERDC provides data on classroom membership from 2006-2017 for all grades. We define a classroom at the school-year-term-teacher-course code level. To calculate classroom peer exposure, we first calculate the share of peers that are lead-exposed in each classroom, where a classroom is defined at the school-year-term-teacher-course code level. We then calculate the share of lead poisoned classroom peers by taking the average over all classrooms a student is in.

Test scores: NCERDC provides data on end-of-grade (EOG) mathematics and reading test scores for grades 3 through 8 between 1997 and 2017. When a student has more than one math or reading EOG score within a school-grade, we take the highest score. We standardize

math and reading test scores at the grade-year level, using the full sample of children with non-missing test scores. We then average the two to obtain our average test score outcome variable. When one either the mathematics or reading test score is missing, we retain the non-missing test score.

North Carolina changed the EOG exams and exam scales multiple times during the sample period. The scale for the math EOG exams changed in 2002, 2006, and 2013. The reading EOG exam scale changed 2003, 2007, and 2013. In addition, North Carolina started offering alternative assessment exams in 2006. Prior to 2006, all students took the same exam. Changes to the exams' scale, grading difficulty and accommodation may have shifted the distribution of test scores across schools in ways that were correlated with peer quality. For example, teachers in schools with more lead poisoned peers may have changed their teaching or grading of exams once the exam difficulty changed in order to meet proficiency thresholds. Schools not meeting proficiency standards were also marked for targeted interventions over this time period. It could also be that there was non-random selection of students allowed to take the regular exam after 2005. For these reasons, we focus on exams administered up to and including 2005. Since the mathematics and reading scale changed during this time period, we include indicators for the testing regime in our regressions.

Suspensions: Data on suspensions are available beginning in 2001. We have information on in and out-of-school suspensions, length of out-of-school suspensions, number of suspensions, and expulsions. Reporting requirements changed in 2009, which resulted in more in-school suspensions and other previously non-reportable incidents to be reported. Reporting for out-of-school suspensions did not change, so in our analysis we focus on out-of-school suspensions (OOS). We create an OOS suspension indicator that equals one if a student had at least one OOS suspension during the academic year. We calculate days spent in OOS suspensions over the academic year by summing over all OOS suspensions in that year. If an OOS suspension is longer than 365 days, we set the length to 365 days.

In all analyses, we limit suspensions to grades 6-12 as suspensions in elementary grades are unlikely. We also drop 2005 from these analyses because the 2005 suspension file does not have a unique student identifier and we cannot link it with other data. We perform two additional sample restrictions to minimize measurement error from reporting. One, for each school we calculate cumulative OOS suspensions over time and drop school-year observations if the

cumulative sum is zero. In other words, we drop schools in years where no student was suspended, as long as that year precedes the first year with positive school-level OOS suspensions. Two, we drop school years with implausibly low suspension rates based on the size of the student body and patterns in the same grades at other schools.

School absences: NCERDC provides data on the number of days a student was absent from school in a given year. These data are available for years 1997-2000 and 2004-2017. At the beginning of our sample, absences are reported in four bins---0-7, 8-14, 15-21, and more than 21 days absent---and we follow this breakdown throughout the sample. We create an indicator for whether a student is absent from school more than 21 days. Following the sample restrictions we impose for OOS suspensions above, we limit absences to grades 6-12 and to school-years where absences are consistently reported.

Same-day OOS suspension and incidents with cohort peer with EBLL: The suspension data provide information on the exact date the suspension occurred, as well as an incident identifier (ID). OOS suspension dates are available from 2001, and incident ID is available from 2004. Both OOS suspension dates and incident ID are missing for 2005-2007. We create a yearly indicator that equals one if a non-poisoned student is suspended OOS on the same day as a lead poisoned cohort peer. Similarly, using the incident ID, we create an indicator for whether a non-poisoned student is involved in the same behavioral incident as a lead poisoned peer each year. We follow the same sample restrictions applied to OOS suspensions.

Same-day OOS suspension and incidents with classroom peer with EBLL: We combine the classroom membership data with the OOS suspension and incident ID data to create indicators for being suspended out of school on the same day as a lead poisoned classroom peer, and for being involved in the same behavioral incident as a lead poisoned classroom peer. We apply the sample restrictions described for OOS suspensions.

Our long-run outcomes are defined only for students who are old enough to reach grade 11 during our sample period, i.e., students born before 1999.

High school graduation: Graduation status is available for students in grade 12 for the entire sample period. Our indicator for having graduated high school equals one if a student has ever earned one of the following diplomas: career preparation, college technical preparation,

college/university preparation, occupational course of study, vocation, or general diploma. NCERDC provides data on graduations and school exit, which complement our data on high school diplomas earned. Students who have obtained a certificate of achievement, graduation certification, or no diploma are not considered high school graduates.

School drop-out: Dropout information is available for the entire sample period, and we use it to create and indicator for having ever dropped out of school. All school systems in North Carolina define a dropout as

> "an individual who was enrolled in school at some time during the reporting year, who was not enrolled on day 20 of the current year, and has not graduated from high school or completed a state or district approved educational program. Students who transferred to another public school district, private school, or home school are not considered dropouts. Students who are temporarily absent due to suspension or illness are not considered dropouts. School leavers whose status is unknown must be included in the total count of dropouts for that year. In 1998, the State Board of Education approved changes to the definition of dropouts. Students who leave high school for a community college GED, adult high school, or other program are considered dropouts."

College intentions: NCERDC provides information on post-high school graduation plans, and actual and intended course of study while in school. Post-graduation intentions are available from 2009, and intended course of study is available from 1998-2005. We define having 4-year college intentions if a student plans on attending a 4-year institution, or attends/ intends to take college prep courses. A student has 2-year college intentions if they plan on attending community or technical college, a trade, business or nursing school, or attend/intend to take college tech prep, occupational or vocational courses.

SAT taking: Data on SAT taking by high school students are available from 2009. Our indicator for having taken the SAT equals one if a student has either math or verbal SAT score. When we don't observe either a math or verbal SAT score, we assume the student did not take the SATs while in high school.

Appendix C: Details of the Measurement Error Simulation in Table C1

In Table C1 we present results from 100 simulations to estimate the direction and magnitude of the potential bias from not having lead testing data on all children. In each iteration, we consider 100,000 students grouped into 500 cohorts of 200 students each. We randomly assign elevated blood lead levels to 10 percent of students, roughly the share in our

sample, and calculate the leave-one-out mean share of peers with elevated blood lead levels in each cohort. Outcome Y (e.g., graduation rate) follows the true data generating process (DGP):

DGP 1: $Y_{ic} = -\ 0.16*\text{Mean_Lead}_c + \varepsilon_i$

where *i* denotes student, *c* denotes cohort, Mean_Lead_c is the share of lead poisoned children in a cohort, and ε_i is the individual ability draw, which is independently and identically distributed (iid) normal with a mean of 0 and standard deviation of 0.1. In other words, we assume the true effect of 100% lead peers is to lower outcomes by 0.16, similar to what we see in the main result in the paper. Column 1 of Table C1 (truth) shows the estimated coefficient from a regression of Y on the true share of EBLL peers in a cohort for the sample of children with no EBLLs (all regressions in the table exclude children with detected BLLs under that scenario, to reproduce the regressions we run in the paper).

Next, we test the extent of bias in our estimates under different testing regimes. In Column 2, we randomly assign 40% of students to be tested for lead (to reflect the share of screened children in our sample) and assume that we only observe an EBLL if a student has an EBLL *and* is tested for lead. We report the coefficient from regressing Y on the average *observed* EBLL share in a cohort controlling for the share of students who have a blood lead test in that cohort, as in our main specification. We show attenuation bias, as expected, but its extent is minimal. In Column 3, we further randomly assign higher testing rates in some cohorts and maintain the assumption that testing is random within cohorts. In Column 4, we introduce a positive correlation between true EBLL and testing. To do so, we randomly assign a risk factor to some students and allow both EBLL status and testing to be correlated with that underlying risk. In both Columns 3 and 4 we slightly underestimate the effect of lead poisoning on peers' outcomes by about 2.3%.

Finally, we investigate the potential role of externalizing behavior in driving both testing and peer effects (even though, as explained in the main text of the paper, we do not think testing is driven by externalizing behavior). We assume that only some students with EBLLs have externalizing behavior (50% in this case), and that it is this behavior that affects peers' outcomes per the following DGP:

DGP 4: $Y_{ic} = -\ 0.2*\text{Mean_Behavior}_c + \varepsilon_i$

where *i* denotes student, *c* denotes cohort, Mean_Behavior_c is the share of lead poisoned children in a cohort with externalizing behavior, and ε_i is iid normal with a mean of 0 and standard

deviation of 0.1. Because only half of the students with EBLLs actually affect their peers, the true estimate of the *average* effect of *any* lead-poisoned student on their peers, in Column 5 is roughly half of the effect of a lead-poisoned peer with externalizing behavior. However, screening might also be correlated with externalizing behavior. To test how this correlation affects our estimates, we further assign 45% of students with externalizing behavior to be tested for lead, and 34% of students without bad behavior to be tested for lead in Column 6. We draw these numbers from the data: 42.5% of children who are ever suspended out of school are tested for lead, and 34% of children without suspensions are tested for lead. We assign more students with externalizing behavior to be tested for lead than are reflected in the NC data to be conservative, since not all externalizing behavior might translate into suspensions from school. This scenario slightly overestimates the average effect of lead poisoned peers.

Summing up, we find that in nearly all our simulations, a lack of perfect information on lead poisoning from not testing every child leads to underestimate the effect of lead-poisoned peers. DGPs 1-3 in columns 2-4 show that this lack of information leads to between 1.6- 2.3% reduction in the magnitude of the estimated coefficients compared to Column 1 (the truth). In the scenario in which behavioral issues lead to greater lead testing, the coefficient in Column 6 would be biased upwards by about 8% compared to the true coefficient in Column 5. Thus, we conclude that selection into testing is unlikely to bias our findings in an economically significant way.

Table C1: Measurement Error Simulation Results

	(1) Truth, DGP 1	(2) DGP 1	(3) DGP 2	(4) DGP 3	(5) Truth, DGP 4	(6) DGP 4
Mean Percent with EBLLs	-0.1612*** (0.0155)				-0.1012*** (0.0165)	
Mean Percent EBLL detected		-0.1574*** (0.0268)	-0.1586*** (0.0246)	-0.1587*** (0.0177)		-0.1094*** (0.0251)
Observations	90003	96048	96010	91400	90003	95879

Notes: Each cell in Table R6 represents the results of a different regression using simulated data. Columns 1 and 5 represent the true coefficients from perfect information under different scenarios in which all or only some lead-poisoned students affect their peers, while columns 2, 3, 4, and 6 report on different DGPs in which the true percentage of lead poisoned children is approximated based on our empirically derived testing rates to see how having imperfect information on testing for lead affects the coefficients. Columns 5 and 6 further illustrate how selection into testing for lead based on behavioral issues affects the estimation of our coefficients. We report average coefficients and their standard deviations from 100 simulations.