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

Ludovica Gazze
Department of Economics, University of Warwick
Ludovica.Gazze@warwick.ac.uk

Claudia Persico,
Department of Public Administration and Policy, School of Public Affairs, American University, NBER, and IZA
cpersico@american.edu

Sandra Spirovska
Department of Economics, University of Wisconsin-Madison
spirovska@wisc.edu

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Abstract

Children exposed to pollutants like lead are more disruptive and have lower achievement. However, little is known about whether lead-exposed children affect the long-run outcomes of their peers. We estimate these spillover effects using new 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. Peer effects are larger for same-gendered students.

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

JEL Codes: Q52, I14, I24

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I. Introduction

A growing literature shows that early life exposure to pollution hinders health and human capital accumulation (Persico, Figlio, and Roth 2020; Alexander and Currie 2017). For example, lead poisoned children are more likely to be suspended and commit crimes (Aizer and Currie 2019; Reyes 2015) and have worse academic achievement (Ferrie, Rolf, and Troesken 2012; Grönqvist, Nilsson, and Robling 2020; Hollingsworth et al. 2020), consistent with lead's impacts on children's neurological development. These associations manifest at blood lead levels (BLLs) as low as 1–2 micrograms per deciliter (μg/dL) of blood (Aizer et al. 2018; Feigenbaum and Mueller 2016). Lead may also affect children's disability status (Gazze 2016). These negative effects of lead exposure are costly to children, families, and society in terms of reduced tax revenues and increased expenditure on special education, crime, and health care (Reyes 2014). Recent estimates suggest that at least 500,000 young children are still poisoned by lead each year in the US (Aizer et al. 2018). [1] Low-income children are up to 12 times more likely to have elevated BLLs (CDC 2005), and Black children are more than twice as likely to be lead poisoned than their White peers (CDC 2005).

So far, the literature has focused on estimating the effects of pollution and lead poisoning on directly exposed children. However, these children interact daily with peers. Because children exposed to lead are more disruptive, have lower achievement, and engage in risky behavior, the effects of lead exposure might spill over to affect everyone in the classroom. While these spillovers may have long term consequences for students, few papers credibly document the long-run impacts of childhood peers. In this paper, we document these spillover effects of lead

[1] In this paper, we use the words lead poisoning and lead exposure interchangeably. We follow the CDC guidelines and define lead poisoning/elevated BLLs as BLLs above 5ug/dL.

poisoning on children who are not directly exposed to lead, but are exposed to lead poisoned school peers.

In addition to considering the impacts of lead, a growing literature shows that various types of common pollution exposure (from highways, toxic sites, etc.) cause worse performance on exams and behavioral issues associated with suspensions from school (Heissel, Simon, and Persico 2021; Persico and Venator 2021). 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. When considering how common it is for low-income children to be exposed to pollution sources, the finding that there are spillovers from pollution exposure means that most children and public schools in the U.S. likely bear some of the cost of pollution. 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.[2] 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.

Rigorously estimating peer effects is challenging because peers influence each other simultaneously, so it is unclear whether a disruptive child causes their classmates to misbehave, or whether the classmates cause them to be disruptive (i.e., the reflection problem). In addition, peer groups are not randomly assigned;

[2] 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. Indeed, while the share of children tested for lead poisoning in North Carolina between 2012 and 2017 was 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.

they are selected based in part on unobserved characteristics (i.e., the selection problem). We overcome these identification challenges by using a novel data set and rigorous identification strategy. We use rich education data from public schools in North Carolina linked to data on children's BLLs measured by age six and compare siblings whose cohorts happen to randomly differ in the proportion of children with high preschool BLLs in their grade-cohort in the same school. Our preferred specification includes family, school, grade, birth month, birth order, and year fixed effects, and controls for a broad set of time varying child and cohort demographic characteristics, as well as school quality. Since lead poisoning has been linked to behavioral incidents, criminality, and lower test scores, we use it as a proxy for peers with potentially disruptive behavior and lower academic achievement. This methodology avoids the reflection problem because a child cannot affect the BLLs of their peers, but lead poisoning might affect children negatively, which in turn might affect peers. Including family fixed effects mitigates the selection problem by controlling for unobserved family characteristics that could be correlated with both peers' quality and a child's outcomes, such as selection into schools with fewer lead poisoned children. 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 peers in a child's cohort that are exposed to lead is associated with a 0.2 percentage point increase in the likelihood of suspension from school, a 1.6 percent increase. A ten percent increase in the share of peers in a cohort exposed to lead is also 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. We also find that having more lead-exposed children in a child's cohort is associated with a higher likelihood of chronic absenteeism and dropping out of school, and a decrease in the likelihood of taking the SAT. Disruptive 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 generally hold in samples limited to locations where we measure lead exposure and sibling matches less noisily, and where students are less likely to switch schools in response to disruptive peers. Our results are also largely robust to further testing for school-switching directly and by including sibling-by-school fixed effects.

We also find that disruptive peers disproportionally affect same-gendered and, in the short run, same-race students. Furthermore, we find that students going to school with a higher share of lead-exposed peers are more likely to be involved in behavioral incidents with these disruptive students. Finally, exposure to disruptive peers in middle school, rather than elementary school, appears to drive long-run outcomes. We interpret our results as suggestive that homophily in network formation might drive the spillover effects of lead poisoning through peers influencing each other 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. By exploiting rich individual-level data, we assess the costs of the spillover effects of lead exposure. 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 are known to affect behavior and suspensions from school.

Second, this is among the first studies to examine the long-run impacts of disruptive peers, 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. We show that exposure to lead poisoned peers can have long term consequences, including dropping out of high

school, even for those children who were not themselves exposed to lead. We also find suggestive evidence on the mechanisms – that homophily within groups and exposure to disruptive peers in middle school might drive some of these effects through the development of noncognitive skills. In particular, our robust results on suspensions and chronic absenteeism strengthen the suggestive evidence provided by Carrell, Hoekstra, and Kuka (2018) that noncognitive skills are a mechanism through which disruptive peers affect long-term outcomes.

Third, we contribute to a growing literature documenting the importance of neighborhood effects for health, education, and behavior outcomes. Our findings on the long-term effects of exposure to lead poisoned children might help explain why high-poverty and high-pollution neighborhoods have persistent effects (Chetty, Hendren, and Katz 2016). Low-income children are more likely to live near sources of toxic waste (Persico, Figlio, and Roth 2020; Banzhaf, Ma, and Timmins 2019), and neighborhood characteristics contribute significantly to health disparities, for example in asthma rates (Alexander and Currie 2017). Our paper presents another channel through which inequalities in prevalence of pollutants at the neighborhood level contribute to the persistence of inequality in the US.

II. Background

Lead Exposure

Ingestion or inhalation of lead causes lead poisoning, which, if severe, can induce widespread brain damage (Meyer, McGeehin, and Falk 2003). 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 (Meyer, McGeehin, and Falk 2003). Specifically, lead causes the axons of nerve cells to degenerate and lose their myelin coats (Meyer, McGreehin and Falk, 2003). Early life exposure to lead has

been shown to cause cognitive disabilities, lower test scores, increase suspensions from school, and even affect crime and wages in adulthood (Persico, Figlio, and Roth 2020; Gazze 2016; Grönqvist, Nilsson, and Robling 2020). Lead has also been associated with problems in cognition, executive functioning, abnormal social behavior (including aggression), and fine motor control (Cecil et al. 2008). Reyes (2014) estimates that lead poisoning costs $200 billion for a single birth-year cohort.

Peer Effects in the Classroom

Peer effects can work through different channels, both positively and negatively. Children teaching each other is an example of a positive peer effect, while disruptive behavior can negatively affect the learning of all children in a classroom (Carrell and Hoekstra 2010; Figlio 2007; Hoxby 2000; Lazear 2001). Using the random assignment of roommates in college, Sacerdote (2001) finds that roommates can influence college grade point averages positively or negatively. A variety of mechanisms link peer composition and academic outcomes, including differential curricular offerings and instructional practices in classes with higher 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.

One strand of the literature examines how low-performing and disruptive children affect peers. Having more low achieving peers or peers with learning disabilities is associated with lower achievement (Hoxby 2000; Fletcher 2010). Moreover, boys with feminine sounding names and children exposed to domestic

violence are both more likely to be disruptive and negatively affect peers' achievement and behavior (Figlio 2007; Carrell and Hoekstra 2010).[3]

Less is known about the long-term impacts of disruptive peers or the mechanisms through which disruptive peers affect long-run outcomes. Carrell, Hoekstra, and Kuka (2018) find that having more disruptive peers in elementary school leads to lower earnings in adulthood and lower college attendance. Bifulco, Fletcher, and Ross (2011) find that a higher percentage of high school classmates with college-educated mothers increases school completion and college attendance. Bobonis and Finan (2009) find that the PROGRESA program in Mexico increased college attendance of non-eligible peers. Black, Devereux, and Salvanes (2013) find that a higher share of girls in ninth grade reduces educational attainment and the likelihood of selecting an academic track for college but lowers teen birth rates.[4] By contrast, Anelli and Peri (2019) find that peers' gender in high school does not affect college major choice, college performance, or income in Italy.

In this paper, we show that children who were exposed to lead are associated with both short- and long-run negative outcomes for their peers. We provide fresh evidence on the spillover effects of lead, the long-run effects of having disruptive peers, and the mechanisms through which peers affect long-run outcomes.

III. Data Description

Education Data

We use 1997-2017 population-level data on every child attending public school in North Carolina, including charter schools, linked to blood lead test records when available. These unique data include home address identifiers that

[3] There are many excellent papers on short run peer effects. See Epple and Romano (2011) and Sacerdote (2011) for overviews of the literature on peer effects.

[4] Relatedly, Balestra, Eugster, and Liebert (2020) find that having peers with special needs lowers performance, the probability of entering post-compulsory education, and income.

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 students to classrooms. The data also include detailed information on students' race and economic disadvantage status in a given year, annual standardized test scores administered by the state, suspensions, absences, high school dropout and completion information, college intentions, as well as teacher characteristics.

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 of our cohort controls), for our main analysis we drop children who do not have siblings, as well as children who live in large buildings since we cannot reliably identify families in those buildings. In Section V, we show how omitting sibling fixed effects and using the whole sample affects our estimates and we test for whether error in sibling matches affects the results. 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. The Data Appendix provides more information on the linkage performed by the North Carolina Education Research Data Center (NCERDC) and our sibling identification algorithm.

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,[5] and having at least one out-of-school (OOS) suspension,[6] 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 EOG exams and exam scales changed

[5] The data break down absences into 0-7, 8-14, 15-21, and more than 21 days. We focus on the last bin as an indicator of chronic absenteeism.

[6] 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.

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.[7]

For our long-term outcomes, we use indicators for high school graduation, dropping out,[8] community and four-year college intentions in 12th grade, and whether the student took the SAT in high school. The Data Appendix provides more details on the construction of our outcome variables.

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, 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 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

[7] During our sample period, the scale for the math EOG exam changed in 2001-2002. The reading EOG exam scale changed in 2002-2003.

[8] The data include separate variables for dropping out and graduating. 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. If a student is ever observed as graduating, we count them as graduating and not dropping out.

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).[9]

Childhood lead screening is not mandatory in North Carolina. However, federal guidelines mandate that all children on Medicaid are screened for lead poisoning at ages one and two. Thus, we expect screening to be higher among low-income children, who have a higher likelihood of lead exposure. 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. Despite this secular trend, Appendix Figure A1 shows that first-born children have only 1.5 percentage points more lead-exposed peers than their younger siblings, suggesting that our identification strategy is not likely to be driven by differences in outcomes between older and younger siblings. Given the large literature on birth order showing that 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), it is unlikely that birth order effects would be driving our results since this would require that later-born siblings would have better outcomes than earlier-born siblings. Moreover, in our regressions on the siblings sample we control for birth order fixed effects.

Sample Description

[9] 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 define a child as having an elevated BLL if alternatively the mean of their BLLs is ≥5 μg/dL or their highest BLL is ≥10 μg/dL.

Since our blood lead level data begin in 1992 and include children tested up to age 6, 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). The Data Appendix details our sample selection criteria. 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. Children in our sibling sample are also marginally more likely to be economically disadvantaged, less likely to be Black, attend schools with slightly larger cohorts but have slightly more teachers with a Master's degree, and have slightly better outcomes. 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.

Children with EBLLs 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 (Columns 4 and 5). 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 lower test scores, higher suspension rates, lower graduation and SAT taking rates, and have a lower probability of intending to attend a four-year college (Columns 6 and 7). These children are also more likely to be Black, be economically disadvantaged, have teachers without Master's degrees, and have a blood lead test themselves. Our identification strategy controls for family background with family fixed effects, assuaging concerns of omitted variable bias due to these differences.

IV. Identification Strategy

Rigorously estimating peer effects has proven difficult methodologically and due to limitations of existing data. First, peers influence each other simultaneously, so it is unclear whether a disruptive child causes their classmates to misbehave, or whether the classmates cause them to be disruptive. This is called the reflection problem (Manski 1993). Second, peer groups are not randomly assigned; they are selected based in part on unobserved characteristics (Angrist 2014). Children in the same classroom often share similar backgrounds. Moreover, 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 (Carrell and Hoekstra 2010; Hoxby 2000). Third, unobserved factors might simultaneously cause students and their peers to perform poorly.

We solve the reflection problem by finding a predetermined proxy for peer ability: lead exposure. Consistent with the literature on lead exposure and academic outcomes, being exposed to lead is strongly associated with worse academic achievement, a higher likelihood of suspension, and a lower probability of graduating or intending to attend a four-year college in our sample (Figure 2).

Previous research has proxied for peer ability and behavior using preexisting measures such as peers' race and gender (Hoxby and Weingarth 2006; Hoxby 2000), feminine-sounding names of male peers (Figlio 2007), peers' retention status (Lavy, Paserman, and Schlosser 2012), peers' disability (Fletcher 2010), or peers' exposure to domestic violence (Carrell and Hoekstra 2010). Our approach is similar in that we use the presence of peers with elevated blood lead levels to estimate how early health shocks (i.e., lead exposure) spill over within school contexts to exacerbate inequality through peer effects. This is a valid approach as a student cannot affect their peers' elevated blood lead levels.

Yet, a child's lead exposure could be correlated with their socioeconomic status, which in turn has been associated with peers' learning disruptions (Hoxby and Weingarth 2006; Hoxby 2000). Thus, to causally identify the spillover effect of a child's lead exposure on their peers we further 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. Finally, we exclude children with known EBLLs so that we can isolate the spillover effects of lead poisoning on peers who are not lead poisoned.

We first examine how lead exposure affects contemporaneous outcomes, that is test scores, suspensions from school, and absences of peers without known EBLLs. To start, 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. 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:

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

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*. $\frac{\sum k \neq i \; PeersEBLLs_{ksgt}}{n_{sgt}-1}$ 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.

X_{it} is a vector of child-specific control variables, including gender, race, birth month fixed effects, economically disadvantaged (ED) status in each year, and an indicator for whether a child was tested for lead. The vector S_{sgt} controls for time-varying school-grade characteristics: the percent non-White students by school-grade-year, the percent economically disadvantaged by school-grade-year, and the share of students who have been tested for lead exposure by school-grade-year. 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. 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. γ_e is an exam type fixed effect that restricts our comparison to children who took the same exam. We cluster standard errors at the school level to account for arbitrary correlation in the error terms.

However, this specification does not account for two potential sources of bias. First, school composition may change over time, and perhaps in response to peers' quality. In addition, families 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, such as parents' propensity to move their children to schools with fewer lead poisoned children. Moreover, an advantage of the North Carolina setting is that over most of our study period there were relatively few options for choosing public schools – there was 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). Thus, as we will show, selection into schools was minimal. 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.[10] Our main estimation equation is thus given by:

$$(2)\ Y_{ijsgt} = \beta_1 \frac{\sum k \neq i\ PeersEBLLs_{ksgt}}{n_{sgt}-1} + \pi X_{it} + \omega S_{sgt} + \theta_j + \delta_s + \tau_g + \sigma_t + \gamma_e + \varepsilon_{ijsgt}$$

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

There are three main threats to the internal validity of our estimates. First, our estimates would be biased if a child's peers' BLLs were correlated to the child's own BLLs, or their ability, other than through classroom interactions. To address this issue, we measure lead exposure prior to school entrance. Moreover, family fixed effects account for omitted variables such as unobserved lead exposure or parental characteristics that could confound the effects of peer quality. School fixed effects help us account for selection into schools. Second, our estimates could be biased in the presence of common shocks that are systematically correlated with the proportion of peers with BLLs in a school-grade-year. Time-varying school and teacher controls help assuage concerns that these channels drive our results. Third, bias could arise if high-quality students systematically select out of schools when

[10] 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).

there are more students with EBLLs. We test for school switching across siblings in Section VD, where we also show that our results are largely robust to including school-by-family fixed effects.

Finally, it is important that we have enough variation in our regressor of interest, the share of children with EBLLs in a school-grade-year, after partialling out our preferred set of controls and fixed effects. Appendix Figure A3 shows the distribution of the residuals obtained from a regression of the share of a student's peers with EBLLs at the school-grade-year level on our preferred set of controls and fixed effects, including family, school, grade, and year fixed effects.

V. Results

A. The Contemporaneous Effects of Peers Exposed to Lead on Child Outcomes

We begin by showing the effects of peers with elevated BLLs on contemporaneous standardized test scores, out-of-school suspensions, and absences. Figure 3 shows that the share of a child's peers with EBLLs is negatively correlated with the child's test scores, and positively correlated with their likelihood of receiving a suspension in the raw data. Table 2 confirms these patterns are causal. Panel A presents the results for the effect of additional cohort peers who are lead poisoned on a child's outcomes using equation (1), while Panel B uses our preferred specification in equation (2) with family, school, grade, and year fixed effects.[11] The two panels show a very similar pattern of results: a higher share of peers with EBLLs is associated with a higher likelihood of, and longer, out-of-school suspensions, as well as a higher likelihood of absences. Generally, within-siblings comparisons appear to estimate slightly smaller effects than comparisons within a

[11] The sample size is smaller than in Column 1 of Table 1 due to singletons and missing outcomes.

school-grade, suggesting that family fixed effects better control for endogenous selection.[12]

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 out-of-school suspension, compared to siblings in the same school. In other words, attending school with 10 percent more lead poisoned peers increases the suspension rate by 1.6 percent above the mean of 12.4 percent, and increases the suspension duration by one hour based on a 8-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.[13] Finally, we note that the effect of lead poisoned peers on suspensions is similar to the effect of economically disadvantaged peers, while the coefficient on non-White peers is negative.

Increased suspensions for peers of lead poisoned children could be due to more punitive policies at the cohort-level. For example, teachers might be more prone to suspending students for minor misbehavior in cohorts with more disruptive students. 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 10 percent on a base of 4.2 percent, suggesting that our results are driven by students misbehaving more when they have more lead poisoned peers and not blanket-style school policies. Finally, some specifications show a decrease in test

[12] Appendix Table A1 estimates the same specification as in Panel A of Table 1 on the sibling sample to see if results differ when restricting to siblings. We find similar results using the sibling sample compared with the sample using all children.

[13] Placebo estimates in Appendix Figure A.2 show that we estimate effects of higher shares of peers with EBLLs on the likelihood of being suspended or involved in an incident with a student with EBLL that are 154-165% of what the mere size of the proportion of children with EBLLs in a given cohort implies.

scores among students with more lead poisoned peers, although the point estimate is positive and not statistically significant at conventional levels in our preferred specification. This suggests that the effects of having lead poisoned peers on the long run outcomes described in Section VB may operate through noncognitive skills and behavior, rather than a learning channel.[14] However, it is difficult to fully disambiguate between these explanations.

While we use cohort-level variation in our primary specification to avoid the issue of selection into classrooms by students, Panel C of Table 2 presents the estimates of the effect of having more lead poisoned peers in the same *classroom* using family, school, grade, and year fixed effects and all controls specified in equation (2). 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. Thus, if students switch classrooms, they will have more peers overall.[15] We find that classroom peers have a larger effect on suspensions and absences than cohort peers. These results could be due to both stronger connections with classroom peers and selection into classrooms.

B. Long Term Effects of Peers Exposed to Lead

We next examine whether a child's lead poisoned peers in elementary and middle school affect that child's long-run outcomes. Table 3 presents estimates of these long-run effects by estimating the following regressions at the student level:

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

[14] 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).

[15] 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 are counted as many times as the number of classes they take with each student.

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

where $\overline{X_\iota}$ and $\overline{S_{st}}$ include all of the individual-level controls from our primary specification, as well as the average share of non-White peers, the average share of economically disadvantaged peers, the average share of peers tested for lead, and the average school size, school stability rate and share of teachers with masters degrees over elementary and middle school. Equation (3) mirrors equation (1) by including school-by-grade (η_{sg}) and grade-by-year (ϕ_{gt}) fixed effects. In equation (4), θ_j, δ_s, τ_g and σ_t are family, school, grade, and year fixed effects as in equation (2). Grade and year are measured during the child's last observation. The coefficient β_1 captures the effect of having 100 percent of peers with *known* EBLLs in elementary and middle school.

Panels A and B of Table 3 present estimates of β_1 from equations (3) and (4) respectively. We examine the effect of elementary and middle school peers with EBLLs on graduation and dropout rates, 4- and 2-year college intentions, and SAT taking. As with short-run outcomes, within-siblings comparisons appear to estimate slightly smaller effects than comparisons within a school-grade, suggesting that family fixed effects better control for endogenous selection.

Our preferred specification in Panel B of Table 3 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, representing 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 0.48 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. Finally, a higher share of lead poisoned peers decreases the likelihood that a student intends to attend a four-year college in Panel

A, although this result is not statistically significant at conventional levels in our preferred specification.

Panel C of Table 3 estimates the long-run effect of lead poisoned peers in elementary and middle school cohorts *separately*. We find that long-run outcomes are largely driven by middle school peers. This result is in line with our findings in Table 2—that exposure to disruptive peers affects behavior in middle school, which in turn could set students on a path to lower graduation and college attendance rates. Peers in middle school also could be especially impactful for long-run outcomes if middle school is a time when some students are deciding whether to remain in school. Finally, student learning and behavior in middle school might be especially important for college readiness (Naven 2019).

While we find mixed evidence of peer effects on test scores, we estimate effects on college going that are similar in magnitude to those obtained by Carrell, Hoekstra, and Kuka (2018). Those authors find that one male peer exposed to domestic violence increases the number of suspensions by 0.01 (although this result is not statistically significant) and 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 adding an additional lead poisoned peer to each class, a 0.04% increase in the share of lead poisoned peers, would lead to a 0.08 percentage points increase in the probability of being suspended, a 12-minutes increase in the suspension duration, 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.

C. Mechanisms and Heterogeneity of Estimated Effects

We hypothesize that children friends' groups might drive peer effects. As we lack data on friendship networks, we exploit the fact that children likely sort into groups with similar characteristics (Jackson 2010). Table 4 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 with EBLLs have larger effects on both short-run outcomes (suspensions) and long-run outcomes (high school graduation and SAT taking), while same-race peers have larger effects on suspensions only. The limited effects of same-race peers on long-run outcomes suggest the effects of homophily in networks might diminish over time, potentially due to selection.

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 school, that could ameliorate the effects of peers with EBLLs. Table 5 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. Boys also seem more affected than girls by lead poisoned peers, although the difference is not statistically significant. Black and male students have lower graduation rates to start with, so our results suggest that these students' learning and behavior might be disproportionally affected when there are disruptive peers. Panel A of Appendix Table A2 shows that lead poisoned boys have larger negative effects on their peers than lead poisoned girls for suspensions from school and SAT taking. Together, these results support

the hypothesis that peer effects are mediated by assortative matching of peer groups, as shown in Table 4.

We find little systematic evidence of heterogeneity by socioeconomic status. Students who are economically disadvantaged only in some grades appear to have larger increases in suspensions and decreases in graduation rates than students who are either never or always economically disadvantaged, but this difference is not statistically significant. Appendix Table A3 presents estimates of the effects of lead poisoned peers for children in schools with different levels of poverty. We find stronger negative peer effects on suspensions in schools with the highest share of economically disadvantaged students, but mixed evidence of heterogeneity in long-run outcomes. This finding suggests that poverty might exacerbate the effects of having lead poisoned peers.

D. Additional Threats to Internal Validity

This section discusses and tests for threats to internal validity, including spurious correlation, selection into lead testing, measurement error, and endogenous sorting.

If our results are driven by increases in peers' blood lead levels, we would expect students exposed to a higher percentage of cohort peers with elevated BLLs to do worse. Figure 4 plots estimates from equations (2) and (4) using bins for different percentages of cohort peers with elevated BLLs (0-5%, 5%-10%, 10%-15%, 15%-20%, 20%-100%).[16] We find a stronger effect of lead poisoned peers on suspensions and graduation rates as the percentage of peers with elevated BLLs increases. Moreover, Figure 5 shows that our estimates are unlikely to be due to random chance. This figure plots the results from estimating 500 placebo specifications in which we assign a random share of lead poisoned peers to each

[16] We omit the indicator for having 0-5% of cohort peers with elevated BLLs from the regression.

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 suspensions and graduation rates fall well outside the distribution of estimates from the placebo specifications.

However, we do not observe lead exposure for all children and there may be selection in who is tested for lead, implying that we measure the share of lead poisoned children in each cohort with error. 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 would attenuate our results. North Carolina requires screening for all children living in zip codes where at least one block group within the zip code has 27 percent or more homes built prior to 1950.[17] Column 1 of Table 6 shows the effects of lead poisoned peers on children in these high-risk zip codes, where screening rates are 16 percent higher than average. We find only a slightly larger effect on graduation than in the full sample suggesting attenuation bias due to measurement error is a minor concern.

Furthermore, we identify siblings based on home addresses, which could lead to error, particularly in multi-family homes. Thus, Column 2 of Table 6 shows results on the sample of Census tracts where the majority of homes are single family homes. Again, we find only a slightly larger effect on graduation than in the full sample.

In addition, if parents of high-achieving students pull their children out of a cohort with particularly disadvantaged or lead poisoned students, such nonrandom selection could lead us to misattribute poor peers' performance to the larger presence of lead poisoned students. Importantly, most of North Carolina did not offer school choice options for public schools over our sample period: with one exception, up until the 2014-2015 school year, students could only switch schools

[17] The designation also adjusts for prevalence of elevated BLLs (Hanchette 1999).

if they switched into a charter or magnet school, which we observe in our data.[18] Column 3 of Table 6 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, suggesting our results in Table 3 are a lower bound on the true spillovers of lead poisoning.

Table 7 formally investigates the association between a student's share of lead poisoned peers and school switching. We find limited evidence of increased switching to public or charter schools of students with higher shares of lead poisoned peers or of their siblings, and if anything the coefficients are negative. Thus, differential sorting does not appear to drive our results. To further test whether differential school switching biases our results, Column 4 of Table 6 controls for siblings-by-school fixed effects, effectively comparing siblings only in grades during which they attend the same school.[19] We find spillover effects of lead poisoned peers that are two-thirds the size of our main result.

Our estimates could also be biased if the share of peers with EBLLs in a school-grade-year is systematically correlated with students' or peers' characteristics other than those included in equation (4). Column 5 of Table 6 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 3, suggesting that we are capturing the true effect of lead exposure, and not other correlates. Column 6 of Table 6 adds fixed effects for the Census block group where students reside when

[18] 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. In addition, the Charlotte Mecklenburg Public School district has had a school choice program from 2002 so we exclude that district.

[19] Bertoni, Brunello, and Cappellari (2020) use this design to study the effects of privileged peers.

they first appear in the school data. The results are similar to those in our main specification despite the sample size being smaller due to missing block group information, suggesting that neighborhood characteristics, including contemporaneous pollution exposure not captured by BLLs by age 6, do not drive the results. Column 7 of Table 6 further shows that estimates using more stringent school-grade fixed effects are similar to our main results that include school and grade fixed effects.

Finally, 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 8 of Table 6, we control for school-year fixed effects and find peer effects that are larger than our main results, suggesting that differential trends in neighborhood-level removal of lead hazards might lead us to underestimate the spillover effects of lead poisoning, if anything.

To address the concern that blood lead levels are measured with some error, in Panels B and C of Appendix Table A2, we show that our results are largely robust to using different measures of lead-exposed peers, although when we define EBLLs as BLLs≥10 μg/dL we find larger effects on long-run outcomes, suggesting that the severity of lead poisoning might affect the magnitude of these peer effects.[20] In Panel D we include all students, even those who are exposed to lead, and control for one's own lead exposure. The estimates are largely similar to our main results.

Finally, Appendix Table A4 shows the robustness of our specification to different sets of controls. Panel A shows that when omitting all controls other than family, school, grade, and year fixed effects, we would find larger

[20] We estimate a small and insignificant effect of peers with BLLs≥10 μg/dL on suspensions. One potential explanation is that interventions targeted at children with BLLs above this intervention threshold successfully improve these children's short-run outcomes, but do not completely mitigate damages in the long-run.

contemporaneous but smaller long-run peer effects, suggesting that spurious correlations might arise even with our conservative specification. Reassuringly, Panel B shows that once we add individual and school-level time-varying controls, omitting the share of students in a school-grade-year who are non-White and the share of students who are economically disadvantaged does not affect our estimates compared to our main results. In other words, peers' characteristics other than lead poisoning do not appear to explain much of the variation in students' outcomes after controlling for the set of fixed effects that provides our identification.[21] This finding suggests that the share of lead poisoned peers does not just capture the effect of non-White or poor peers. Panel C 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.

VI. Conclusion

This is the first study documenting the spillover effects of lead onto school peers. By comparing siblings who attend the same school, we find that a child's own lead exposure spills over to affect other children's behavior and long-run outcomes. A ten percent increase in middle school peers with elevated BLLs in a given year leads to a 1.6 percent increase above the mean in the likelihood of being suspended out of school and a ten percent increase in chronic absenteeism. A ten percent increase in peers with elevated BLLs over a student's elementary and middle school career causes a 2 percent decrease in the likelihood of graduating high school, and a 4.3 percent decrease in the likelihood of taking the SAT. These large effects suggest that the social cost of lead exposure has been underestimated

[21] Related, Appendix Table A5 shows limited evidence that peers' composition at the cohort level is related to school quality 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.

so far. Our results suggest that environmental hazards are an important factor contributing to human capital accumulation even for children who are not themselves exposed to these hazards. In addition, we show that peers can have long term consequences on human capital formation and reveal some mechanisms through which peer effects manifest, namely homophily in network formation and behavior shaping while in middle school likely through noncognitive skills. Furthermore, our findings have implications for other types of common pollution that are known to cause suspensions from school, such as traffic and industrial pollution (Persico and Venator 2020; Heissel, Persico, and Simon 2020), suggesting that the true cost of pollution has been underestimated.

We likely estimate a lower bound of the effect of lead poisoned peers. We find strong evidence of worse outcomes for children exposed to more lead poisoned peers despite their siblings are likely exposed to disruptive peers as well and despite potential spillovers within siblings, too. Moreover, missing BLLs for some lead poisoned children would attenuate our findings.

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 effect of one lead poisoned peer in a cohort of 220. We find that being exposed to one additional lead poisoned peer is associated with $71 in lost earnings per student from lower graduation rates alone.[22] This estimate does not include the additional costs of behavioral issues, yet, it implies a spillover effect of

[22] 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 of 1.9 percent and a discount rate of 3.38 (i.e., the 30-year Treasury bond rate). 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 (-16.63 percentage points) to obtain the impact of one child with EBLLs through elementary and middle school on graduation rates: -0.076 percentage points, or a decrease in the probability of 0.00076. Thus, one child with EBLLs in a cohort would decrease the net present value of lifetime earnings by 0.00076*$93,188=$71.

a lead poisoned child of $15,549 on their 219 school peers. As half a million young children appear to still be poisoned by lead each year (Aizer et al. 2018) and lead poisoned students appear to be quite dispersed across schools, these spillovers total almost $8 billion per birth-year cohort. Reyes (2014) estimates the direct social cost of lead poisoning at $200 billion per birth-year cohort. Thus, our, likely lower-bound, estimates suggest that the social cost of lead has been underestimated by at least 4 percent by not including these spillover effects. Importantly, our analysis suggests that most public school children in the United States 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 outcomes. Thus, early interventions might help both lead poisoned children and their peers.

Finally, school segregation by race and socioeconomic status likely exacerbates these peer effects, suggesting that additional efforts to desegregate students might be beneficial. Low-income schools have some of the largest achievement gaps (e.g., see Reardon 2015). Our results suggest that peer effects and lead exposure contribute to low performance in high-poverty schools, as well as to the negative long-run outcomes associated with poverty. Lead exposure and exposure to lead poisoned peers are both mechanisms through which poverty produces worse human capital outcomes. Understanding how the organization of schools mitigates these negative effects is crucial to design policies that curb the negative consequences of lead poisoning and pollution exposure.

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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	-0.094	0.265
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	0.522
Cohort size	220	225	199	203	229	193	261
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	0.272	0.249	0.308	0.432	0.226	0.308	0.188
Share Hispanic	0.122	0.124	0.144	0.107	0.126	0.136	0.112
Share with a BLL test	0.338	0.396	1	1	0.322	0.534	0.255
Share with EBLL	0.071	0.079	0.200	0.725	0	0.118	0.040
N Students	3,334,365	1,326,622	525,535	144,957	1,181,665	670,386	656,236

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: 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	-0.0354	0.0691***	1.1348***	0.2263***	0.1105***	0.0239**
	(0.0351)	(0.0187)	(0.2426)	(0.0143)	(0.0118)	(0.0091)
Observations	3,303,025	7,924,730	7,924,730	7,196,034	6,542,610	8,135,286
N Students	932,753	1,907,865	1,907,865	1,884,925	1,765,517	1,903,431
Mean of outcome	0.0572	0.1049	0.7744	0.0318	0.0202	0.0612
Panel B: Cohort Peers with Family, School, Grade and Year Fixed Effects						
Share of peers with BLLs over 5 µg/dL	0.0193	0.0202+	0.6305***	0.1933***	0.1040***	0.0440***
	(0.0360)	(0.0117)	(0.1804)	(0.0106)	(0.0082)	(0.0072)
Share of Non-White Children in School-Grade-Year	-0.0194	-0.0186+	-0.0071	0.0007	-0.0045	0.0136*
	(0.0212)	(0.0103)	(0.1660)	(0.0101)	(0.0075)	(0.0053)
Share of Economically Disadvantaged Children in School-Grade-Year	0.0243*	0.0204***	0.3224***	0.0166***	0.0055	0.0077**
	(0.0121)	(0.0061)	(0.0819)	(0.0050)	(0.0058)	(0.0030)
Observations	1,414,124	4,290,255	4,290,255	3,921,543	3,673,505	4,397,906
N Students	374,137	944,681	944,681	933,835	891,487	939,623
Mean of outcome	0.1169	0.1238	0.5041	0.0285	0.0187	0.0424
Panel C: Peers in the Same Classroom with Family and School Fixed Effects						
Share of peers with BLLs over 5 µg/dL	--------	0.0568***	0.3820+	0.2250***	0.1489***	0.0947***
		(0.0133)	(0.2146)	(0.0119)	(0.0091)	(0.0095)
Observations	--------	3,700,650	3,700,650	3,416,626	3,416,626	3,679,226
N Students	--------	877,934	877,934	824,400	824,400	884,139
Mean of outcome	--------	0.0931	0.6325	0.0138	0.0082	0.0507

Notes: The table reports the effect of a child's share of peers with EBLLs on the child's school outcomes. Panels A and B use the share of peers with maximum BLLs over 5 µg/dL *at the school-grade-year level* as the main explanatory variable, while panel C use the share of peers with maximum BLLs over 5 µg/dL *at the classroom level*. Panel A includes school-by-grade, grade-by-year and birth month fixed effects. Panels B and C instead include 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. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table 3: Long-Run Outcomes of Exposure to Peers with Elevated BLLs by Timing of Exposure

Dependent Variable:	(1) Ever graduated	(2) Ever dropped out	(3) Intention to Attend a 4-Year College	(4) Intention to Attend a Community College	(5) Took 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***	0.1468***	-0.1257***	-0.0098	-0.2778***
	(0.0173)	(0.0150)	(0.0362)	(0.0344)	(0.0350)
Mean of outcome	0.8491	0.0597	0.4406	0.3544	0.4588
N Students	831,386	1,157,178	666,821	666,159	657,878
	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	-0.1663***	0.0476+	-0.1126	0.0464	-0.2289**
	(0.0351)	(0.0245)	(0.0720)	(0.0785)	(0.0740)
Mean of outcome	0.8904	0.0529	0.5068	0.3288	0.5319
N Students	283,032	415,049	205,833	205,761	201,784
	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	-0.0614*	-0.0025	-0.0100	0.0216	-0.0020
	(0.0295)	(0.0216)	(0.0671)	(0.0687)	(0.0669)
Share of peers with BLLs over 5 µg/dL in Middle School	-0.1169**	0.0680*	-0.0546	-0.0073	-0.2204*
	(0.0415)	(0.0298)	(0.0906)	(0.0990)	(0.0872)
Mean of outcome	0.8944	0.0519	0.5108	0.3299	0.5380
N Students	248,478	355,238	182,351	182,294	178,875

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. We restrict the sample to 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 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, 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<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table 4: Heterogeneity by Peer Gender and Race

	Short Run Outcomes	*Long Run Outcomes*	
Dependent Variable:	(1) Out of School suspension	(2) Ever graduated	(3) Took the SAT
Panel A: By Same-Gender Lead Poisoned Peers			
Share of same-gender peers with BLLs ≥5 µg/dL	0.0771***	-0.1012+	0.0409
	(0.0176)	(0.0536)	(0.1302)
Share of peers with BLLs ≥5 µg/dL	-0.0199	-0.1155**	-0.2496*
	(0.0146)	(0.0425)	(0.0978)
Panel B: By Same-Race Lead Poisoned Peers (White)			
Share of same-race peers with BLLs ≥5 µg/dL	0.0628***	0.0300	-0.1657
	(0.0158)	(0.0580)	(0.1196)
Share of peers with BLLs ≥5 µg/dL	-0.0196	-0.1867***	-0.1156
	(0.0145)	(0.0482)	(0.1112)
Panel C: By Same Gender-Race Lead Poisoned Peers (White)			
Share of same gender-race peers with BLLs ≥5 µg/dL	0.0994***	-0.0295	-0.0827
	(0.0173)	(0.0610)	(0.1416)
Share of peers with BLLs ≥5 µg/dL	-0.0123	-0.1562***	-0.2003*
	(0.0124)	(0.0373)	(0.0884)
N Students	944,678	283,032	201,784
Mean of outcome	0.1037	0.8904	0.5319

Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes. 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 in Columns 2-3. Standard errors are in parentheses and clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table 5: Heterogeneity by Demographic Subgroups

	Short Run Outcomes	*Long-run Outcomes*	
Dependent Variable:	(1) Out of School suspension	(2) Ever Graduated	(3) Took the SAT
	Panel A: White, non-Hispanic students		
Share of peers w/ BLLs ≥5µg/dL	0.0095	-0.1445***	-0.1196
	(0.0110)	(0.0404)	(0.0873)
	Panel B: Black non-Hispanic students		
Share of peers w/ BLLs ≥5µg/dL	-0.0045	-0.3133***	-0.3698*
	(0.0244)	(0.0772)	(0.1679)
p-val. =White	0.60	0.05	0.19
	Panel C: Hispanic students		
Share of peers w/ BLLs ≥5µg/dL	-0.0009	-0.1362	-0.4186+
	(0.0257)	(0.1251)	(0.2413)
p-val. =White	0.71	0.95	0.24
	Panel D: Never Economically Disadvantaged students		
Share of peers w/ BLLs ≥5µg/dL	0.0006	-0.0911*	-0.0170
	(0.0100)	(0.0402)	(0.1108)
	Panel E: Sometimes Economically Disadvantaged students		
Share of peers w/ BLLs ≥5µg/dL	0.0229	-0.1815**	-0.2745*
	(0.0177)	(0.0682)	(0.1358)
p-val. =Never	0.27	0.25	0.14
	Panel F: Always Economically Disadvantaged students		
Share of peers w/ BLLs ≥5µg/dL	0.0103	0.0249	-0.1424
	(0.0213)	(0.1033	(0.2021)
p-val. =Never	0.68	0.30	0.59
	Panel G: Girls		
Share of peers w/ BLLs ≥5µg/dL	0.0180	-0.1117+	-0.2350+
	(0.0127)	(0.0606)	(0.1309)
	Panel H: Boys		
Share of peers w/ BLLs ≥5µg/dL	0.0334*	-0.2469***	-0.2065
	(0.0165)	(0.0668)	(0.1361)
p-val. =Girls	0.46	0.13	0.88

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 in Columns 2-3. Standard errors are clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table 6: Results for Alternative Samples and Alternative Specifications

	Ever Graduated							
	(1) Zip Codes with Universal Screening	(2) >50% of Homes in Census Tract are Single Family	(3) Zip Codes with No School Choice Options	(4) Adding Sibling-School Fixed Effects	(5) Adding Cohort Block Group Characteristics	(6) Adding Block Group Fixed Effects	(7) Adding School-Grade Fixed Effects	(8) Adding School-Year Fixed Effects
Share of peers with BLLs over 5 µg/dL	-0.1854***	-0.1866**	-0.2681***	-0.1035***	-0.1704***	-0.2214***	-0.1462***	-0.2282***
	(0.0454)	(0.0721)	(0.0715)	(0.0267)	(0.0345)	(0.0594)	(0.0309)	(0.0449)
N Students	146,599	84,729	175,973	228,036	283,032	118,747	282,582	281,851
Mean of outcome	0.8800	0.8830	0.8934	0.8904	0.8904	0.8904	0.8904	0.8925
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

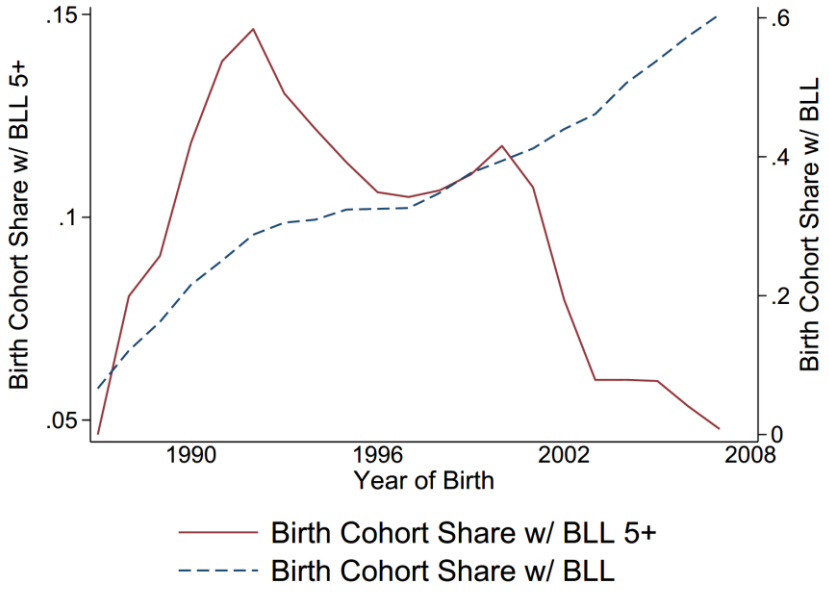
Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes. Each column reports results from a separate regression. Column 1 restricts the sample to students who live in zip codes that are subject to universal lead screening. Column 2 restricts the sample to Census tracts where more than half of homes are single family homes. Column 3 restricts the sample to zip codes without charter schools or voucher programs. Columns 4-8 add controls and alternative sets of fixed effects as specified at the top and bottom of each column. 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. 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 7: 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	0.0026	-0.0038*	-0.0093	-0.0095	-0.0335*
	(0.0131)	(0.0015)	(0.0226)	(0.0137)	(0.0149)
Observations	6,555,045	6,452,608	1,576,604	840,101	4,193,920
N Students	1,094,875	1,089,551	491,901	309,887	587,149
Mean of outcome	0.3063	0.0050	0.3464	0.1310	0.1858

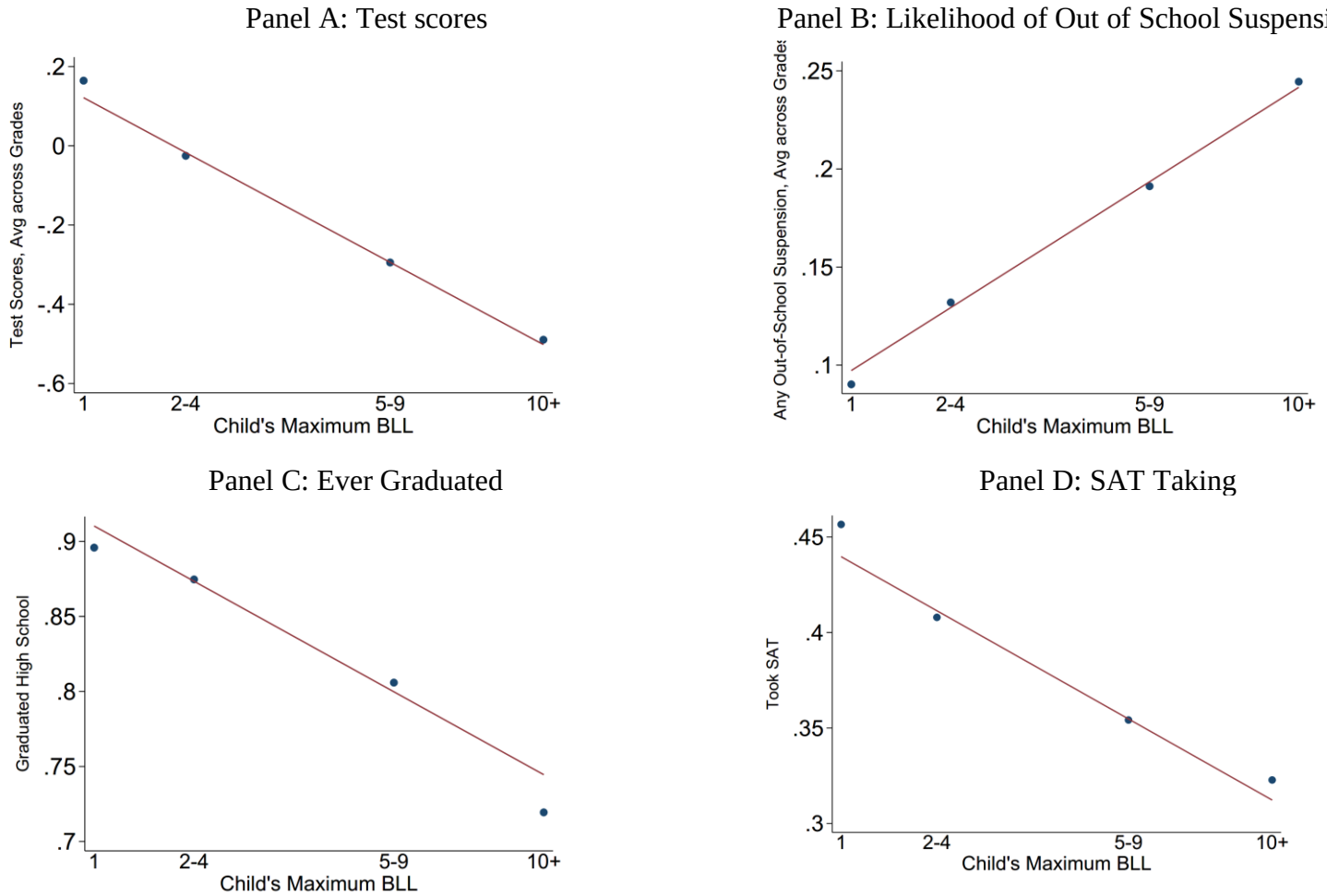
Notes: The table reports the association of a child's share of peers with elevated blood lead levels with the child's own likelihood of switching schools (Columns 1 and 2), the child's sibling's likelihood of switching schools conditional on attending the same school (Column 3), both children switching 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.

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



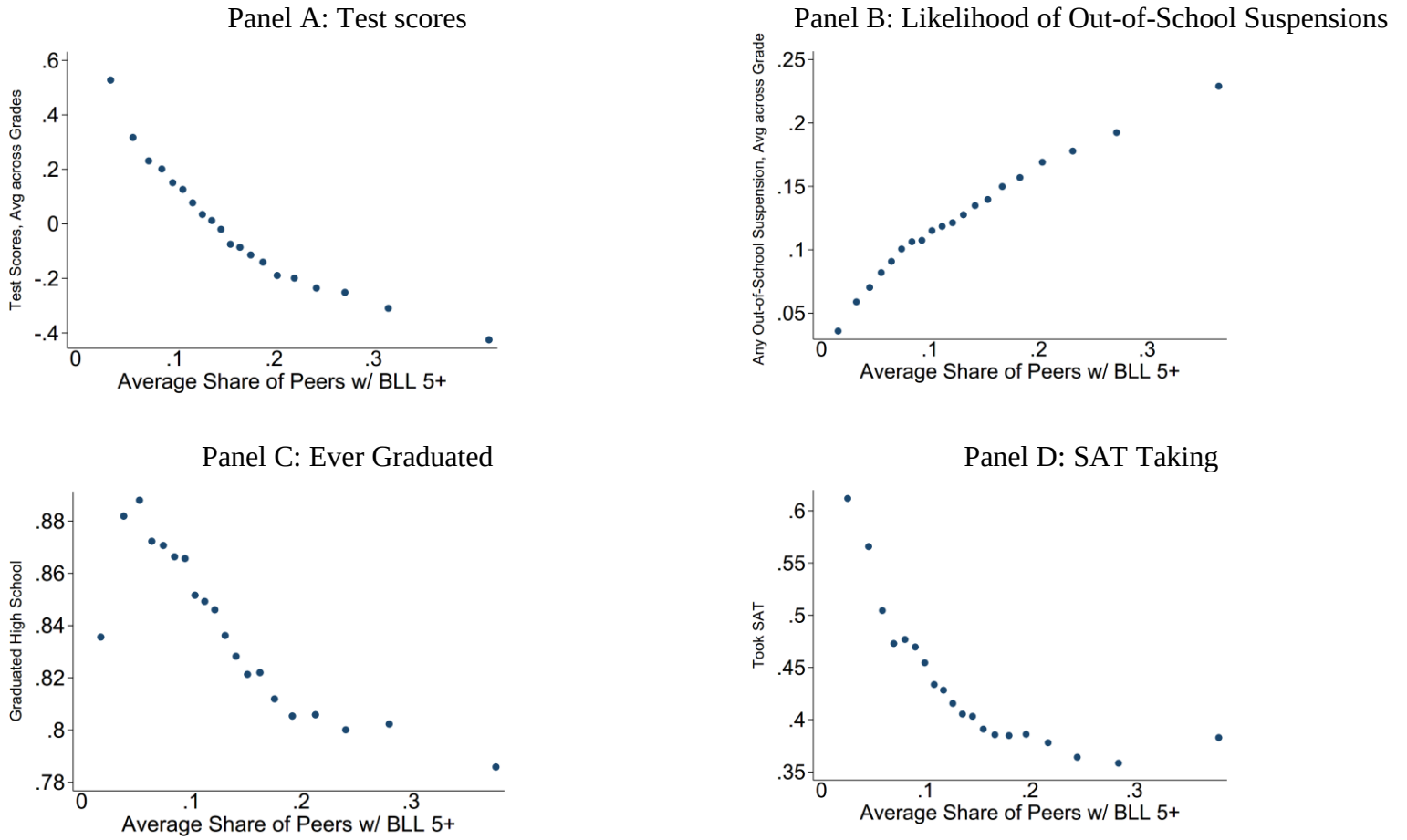
Notes: The figure plots the share of children in a school-grade-year cohort with at least one blood lead test (blue dashed line) and with a blood lead level of at least 5µg/dL (red solid line)

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

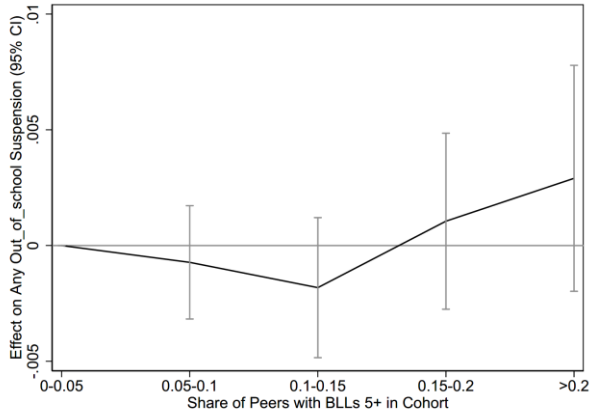
Figure 3: The Relationship Between Peers' Blood Lead Levels and Test Scores, Out-of-School Suspensions, High School Graduation, and SAT Taking



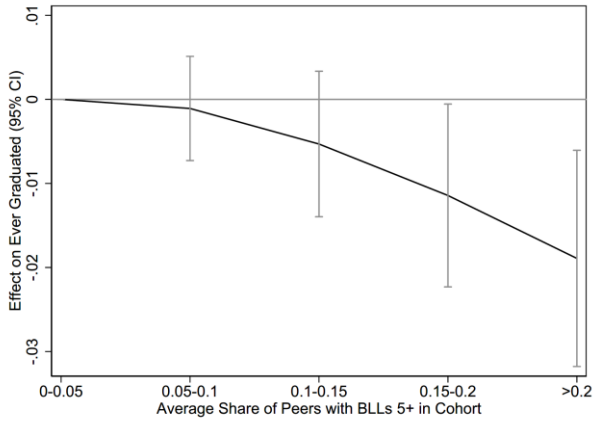
Notes: The figure plots average test scores (Panel A), out-of-school suspension rates (Panel B), graduation rates (Panel C), and SAT taking rates (Panel D) by vigintile of students' share of peers with blood lead levels at or above 5µg/dL.

Figure 4: Binned Effects of Share of Peers with Blood Lead Levels above 5 µg/dL

Panel A: Likelihood of Out-of-School Suspension

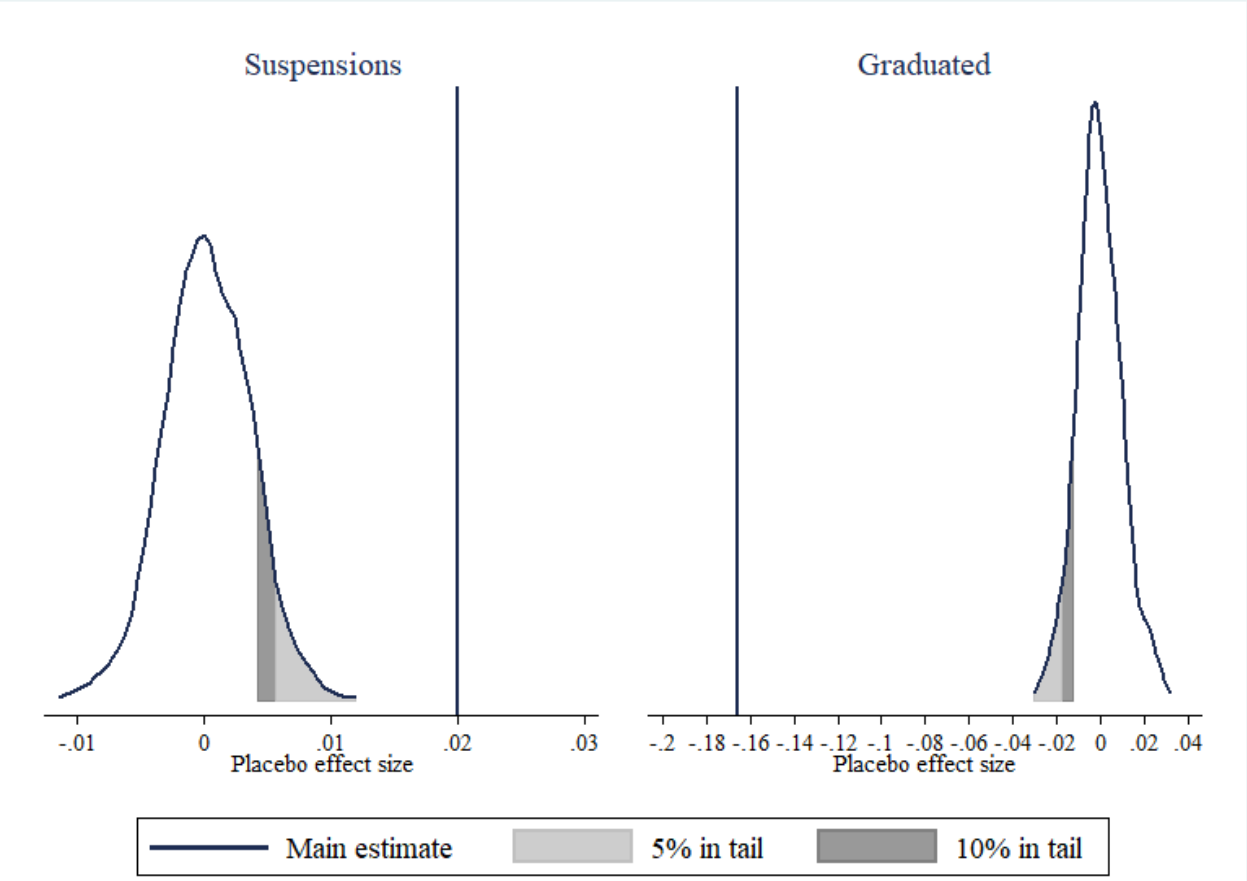


Panel B: Ever Graduated



Notes: Each figure plots non-parametric estimates of the effect of having different proportions (binned) of peers with BLLs 5+ in a child's cohort on out-of-school suspension rates (Panel A) and graduation rates (Panel B). The omitted category is an indicator for share of peers with BLLs 5+ that is lower than 0.05. 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 in Panel B). Vertical bars represent 95% confidence intervals based on standard errors clustered at the school level.

Figure 5: Placebo Estimates



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, school-grade cohorts were randomly assigned a percent of peers with EBLLs from the empirically observed distribution and we estimated our main specification.

APPENDIX A: Additional Tables and Figures

Table A1: Results with School-grade and grade-year fixed effects on sibling sample

	Short-run Outcomes	*Long-run Outcomes*	
	(1)	(2)	(3)
Dependent Variable:	Out of School Suspension	Ever Graduated	Took the SAT
Share of peers with BLLs over 5 µg/dL	0.0584***	-0.1060***	-0.2587***
	(0.0170)	(0.0203)	(0.0516)
Observations	4,290,161	282,782	201,719
N Students	944,679	282,782	201,719
Mean of outcome	0.0942	0.8911	0.5320

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. Each column reports results from a separate regression. All regressions 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 2-3. 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: Alternative measures of peers' BLLs and sample including lead poisoned students

	Short Run Outcomes	*Long-run Outcomes*	
	(1) Out of School Suspension	(2) Ever Graduated	(3) Took the SAT
Panel A: Share of Male and Female Peers with Max BLL over 5 µg/dL			
Share of male peers with BLLs over 5 µg/dL	0.0565***	-0.1489**	-0.2523*
	(0.0158)	(0.0454)	(0.1021)
Share of female peers with BLLs over 5 µg/dL	-0.0255	-0.1861***	-0.1942+
	(0.0171)	(0.0467)	(0.1066)
Panel B: Mean BLL is over 5 µg/dL			
Share of peers with BLLs over 5 µg/dL	0.0177	-0.2073***	-0.2320**
	(0.0133)	(0.0352)	(0.0740)
Panel C: Max BLL is over 10 µg/dL			
Share of peers with BLLs over 10 µg/dL	0.0006	-0.4047***	-0.4660**
	(0.0339)	(0.0849)	(0.1503)
N Students	944,681	281,098	200,186
Mean of outcome	0.1037	0.8902	0.5316
Panel D: Including Students with Elevated Blood Lead Levels			
Share of peers with BLLs over 5 µg/dL	0.0268*	-0.1764***	-0.2031***
	(0.0121)	(0.0327)	(0.0596)
N Students	1,081,179	361,787	257,202
Mean of outcome	0.1125	0.8768	0.5031

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 over 5 µg/dL. Panel B uses the share of peers with average BLL above 5 µg/dL. Panel C uses the share of peers with maximum BLL over 10 µg/dL. Panel D includes children who have maximum BLL over 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 at the school-grade-year level, 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 in Columns 2-3. Standard errors are clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A3: Heterogeneity by School-Level Demographics

	Short Run Outcomes	*Long-run Outcomes*	
Dependent Variable:	(1) Out of School Suspension	(2) Ever Graduated	(3) 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	0.0249	-0.1251^{+}	-0.0926
	(0.0238)	(0.0674)	(0.1656)
Panel B: Schools in Middle Tercile of Share Students who are Economically Disadvantaged			
Share of peers with BLLs over 5 µg/dL	0.0365^{+}	-0.1452^{+}	-0.0626
	(0.0201)	(0.0796)	(0.1464)
p-val = First Tercile	0.33	0.85	0.89
Panel C: Schools in Highest Tercile of Share Students who are Economically Disadvantaged			
Share of peers with BLLs over 5 µg/dL	0.0324^{+}	-0.1049	-0.2013
	(0.0193)	(0.0652)	(0.1346)
p-val = First Tercile	0.06	0.83	0.61
N Students	931,381	222,894	162,137
Mean of outcome	0.1034	0.8964	0.5454

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 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 in Columns 2-3. Standard errors are clustered at the school level. + p<0.10, * p<0.05, ** p<0.01, *** p<0.001.

Table A4: Results with Fewer Controls

	Short-run Outcomes	*Long-run Outcomes*	
	(1)	(2)	(3)
Dependent Variable:	Out of School Suspension	Ever Graduated	Took the SAT
	Panel A: No controls		
Share of peers with BLLs over 5 µg/dL	0.0433***	-0.0170	-0.0770
	(0.0102)	(0.0241)	(0.0560)
Observations	4,290,255	283,032	201,784
N Students	944,681	283,032	201,784
	Panel B: All Controls Except for Share Non-White and Share Educationally Disadvantaged		
Share of peers with BLLs over 5 µg/dL	0.0249*	-0.1609***	-0.1718*
	(0.0115)	(0.0349)	(0.0769)
Observations	4,290,255	248,478	178,875
N Students	944,681	248,478	178,875
	Panel C: Family Fixed Effects		
Share of peers with BLLs over 5 µg/dL	0.0368**	-0.1578***	-0.2331**
	(0.0117)	(0.0372)	(0.0739)
Observations	4,290,270	283,236	201,814
N Students	944,682	283,236	201,814

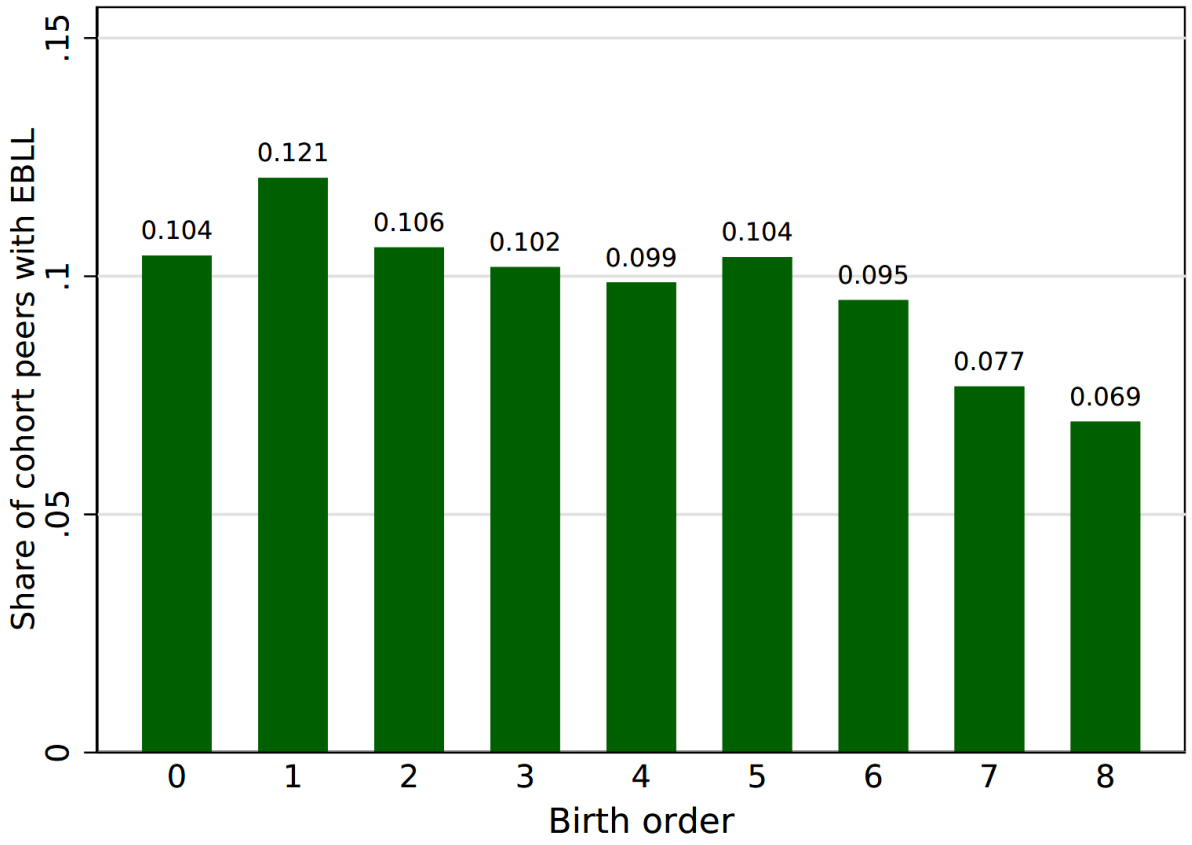
Notes: The table reports the effect of a child's share of peers with elevated blood lead levels on the child's school outcomes. Each cell reports results from a separate regression. All regressions include sibling, birth month, grade, year and birth order fixed effects. Panel A shows our results with no control variables except for our fixed effects and school 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 share of children with a lead test at the school-grade-year level, as well as school size, the stability rate, and the percent of teachers with an MA degree. We omit 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. Cohort and school controls are averaged over elementary and middle school in Columns 2-3. 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: 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 BLLs over 5 µg/dL	-0.0104	0.0049*	0.0041	-0.0003	127.6286***
	(0.0077)	(0.0020)	(0.0035)	(0.0003)	(22.1179)
Observations	7,611,499	7,760,468	7,760,559	7,760,559	7,760,559
Mean ofoutcome	0.3657	0.9575	0.0191	0.0000	782.1071
N Students	1,171,476	1,177,800	1,177,800	1,177,800	1,177,800

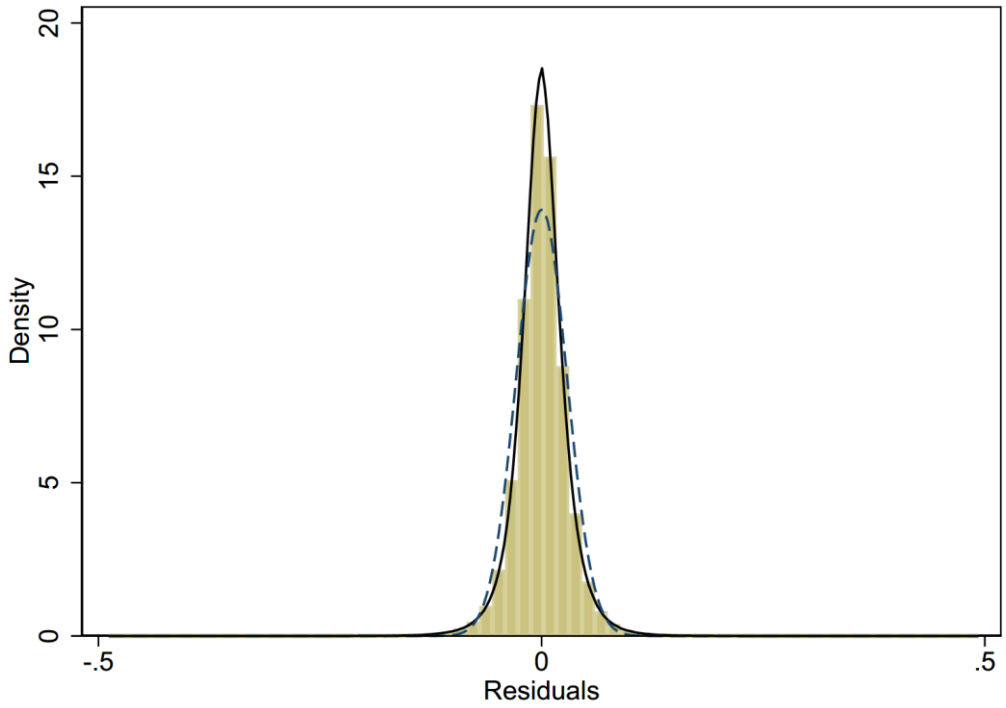
Notes: The table reports the correlation of a child's share of peers with EBLLs in a cohort with school-year characteristics. Regressions include family, school, grade, and year fixed effects, controlling for birth order. All regressions control for individual and cohort controls, which include indicators 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.

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.

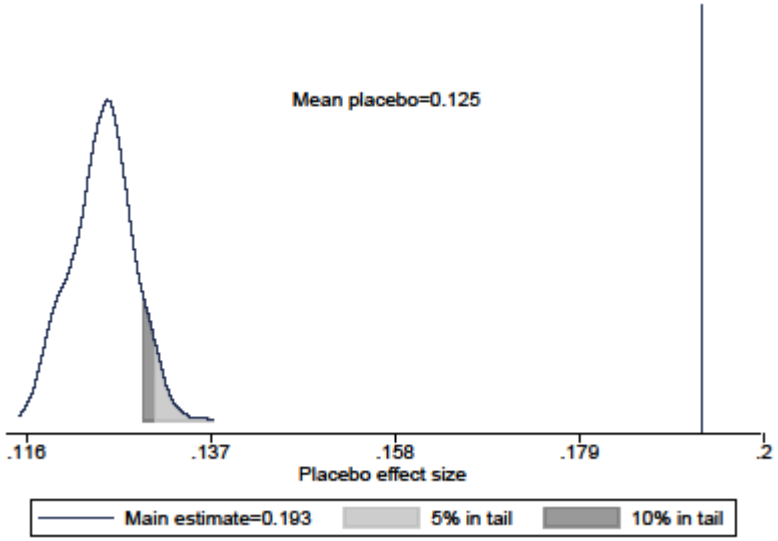
Figure A2: Identifying Variation: Residual Variation in Share of Peers with Elevated Blood Lead Levels



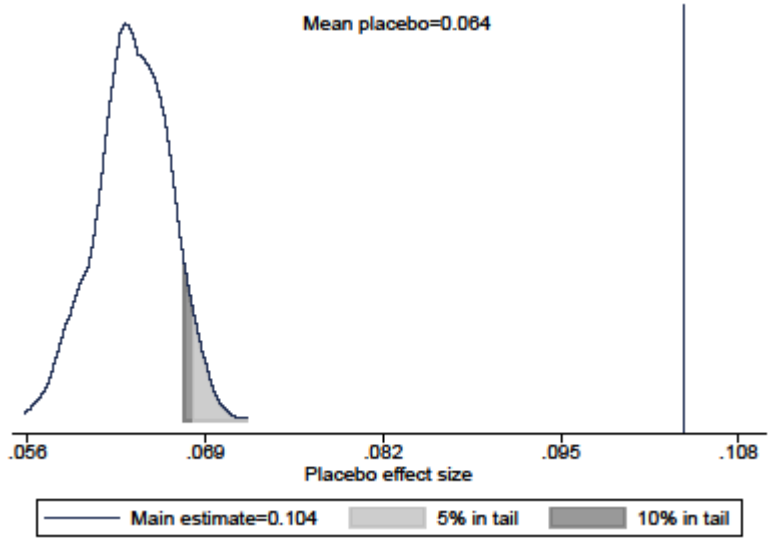
Notes: This figure plots the distribution of the residuals from a regression of our variable of interest, share of peers with blood lead levels at or above 5µg/dL on the fixed effects and controls included in our preferred specification. We include family, birth month, birth order, grade, school, and 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 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. 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