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

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

I. Introduction

Commonly encountered pollution sources, such as lead paint, highways, and toxic sites, have been shown to affect children's academic achievement

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and behavior (Persico, Figlio, and Roth 2020; Persico and Venator 2021; Heissel, Persico, and Simon 2022). So far, researchers have focused on estimating the effects of pollution on directly exposed children. However, pollution-exposed children interact daily with peers. Because children exposed to pollution have lower achievement and engage in risky behavior, the effects of pollution might spill over to affect everyone in the classroom. Yet few papers credibly document the long-run impacts of childhood peers generally, and no existing studies explore the spillover effects of pollution on peers in school. Showing that pollution exposure has spillover effects is important because it reveals the scope of the problems pollution causes. If one child's exposure to pollution causes negative long-run spillover effects on the child's peers, this increases the true costs of pollution and changes our understanding of how pollution might affect long-run human capital attainment.

In this paper, we focus on one type of pollution: lead poisoning. A growing literature shows that children who are lead poisoned in early life are also more likely to be suspended and commit crimes and have worse academic achievement and long-run outcomes.[1] A recent UNICEF report reveals that lead poisoning is a global issue: as many as 800 million children, or around one in three, have blood lead levels (BLLs) at or above 5 μg/dL, the reference

data linked to education records data used in this analysis. The opinions expressed herein do not represent the views of the North Carolina Department of Public Instruction or the North Carolina Department of Health and Human Services. We are thankful to Manuel Bagues, Marco Bertoni, Eric Chyn, James Fenske, Fanny Landaud, Victor Lavy, Lauren Russell, Viviane Sanfelice, Anna Aizer, and seminar and conference participants at UChicago Urban Labs; University of Pennsylvania; York University; APPAM; Temple University; University of Warwick; Brown University; Arizona State University; Emory University; CREST; University of St. Andrews; University of Birmingham; University of Newcastle; University of Reading; University College Dublin; University of Central Oklahoma; University of Maryland; George Washington University; Online Summer Workshop in Environment, Energy, and Transportation; Women in Economics: Advancing Research in Economics; Allied Social Sciences Associations; Association For Education Finance and Policy; Midwest Economics Association; Royal Economic Society; Association of Environmental and Resource Economists; 2022 Junior Economist Meeting at the University of Milano; University of Barcelona Workshop on Public Policies; the 8th Institute of Labor Economics Workshop in Environment, Health and Labor Markets; and the 4th Annual London School of Economics–Imperial College Workshop in Environmental Economics for helpful comments and suggestions. We are also grateful to the Institute for Research on Poverty at the University of Wisconsin–Madison, the Becker Friedman Institute at the University of Chicago, and American University's School of Public Affairs and Provost for their initial financial support for data construction. All errors are our own. Contact the corresponding author, Claudia Persico, at cpersico@american.edu. Information concerning access to the data used in this paper is available as supplemental material online.

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

value most commonly used to identify children who have elevated BLLs (EBLLs) during our sample period (UNICEF 2020).[2] At this threshold, at least 500,000 young children are estimated to be poisoned by lead each year in the United States (Aizer et al. 2018), generating $200 billion per cohort in societal costs (Reyes 2014), including reduced tax revenues and increased expenditure on special education, crime prevention, and health care. Low-income and Black children are more likely to have EBLLs compared with higher-income and White children (CDC 2005).

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

Using unique data linking children's BLLs by age 6 to the universe of public school records in North Carolina, we are the first to investigate the negative long-run spillover effects of lead poisoning on children who are not directly exposed to lead but are exposed to lead-poisoned school peers. We identify the spillover effects of lead-exposed peers by comparing siblings whose cohorts happen to randomly differ in the proportion of children with high preschool BLLs in their grade cohort. Our preferred specification includes family, school, grade, birth month, birth order, and year fixed effects and controls for a broad set of time-varying child and cohort demographic characteristics, as well as school quality. Including family fixed effects controls for unobserved family characteristics, such as parental traits, that could be correlated with both peers' quality and a child's outcomes. 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 10% increase in the share of cohort peers exposed to lead is associated with a 1.7 percentage point decrease in the likelihood that a child graduates high school, a 2% decrease in the graduation rate. Having more

[2] In this paper we use the phrases "lead poisoning" and "lead exposure" interchangeably.

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

lead-exposed cohort peers is also associated with a higher likelihood of suspension from school, chronic absenteeism, and dropping out of school and a lower likelihood of taking the SAT. A back-of-the-envelope calculation suggests that the lost earnings of classmates of lead-poisoned children not graduating high school amount to $9.2 billion per cohort. Lead-exposed peers disproportionately affect the outcomes of Black students, suggesting that the spillover effects of pollution could be contributing to persistent inequality in human capital accumulation. These findings are generally robust to different specifications that account for potential selection, omitted-variable, and measurement error biases.

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

This paper makes three main contributions. First, this is the first study to investigate the spillover effects of lead exposure on peers' academic achievement, behavior, and long-run outcomes. Furthermore, our findings have implications for more than just lead: our estimates imply that the true costs of pollution are likely higher than the direct costs alone, especially for pollutants that affect behavior.

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

Several mechanisms could link peer composition and student outcomes, including differential curricular offerings and instructional practices depending on average ability (Jackson 2013), social dynamics in a student's reference group (Hoxby 2000; Brenøe and Zölitz 2020), and low-performing students not keeping up with higher-achieving peers (Imberman, Kugler,

[4] Other papers find that peers' parental education and peers' receipt of conditional cash transfers increases college attendance, while peers with special needs lower it (Bobonis and Finan 2009; Bifulco, Fletcher, and Ross 2011; Balestra, Eugster, and Liebert 2022). Evidence on the relationship between peers' gender and long-run outcomes is more mixed (Black, Devereux, and Salvanes 2013; Anelli and Peri 2017).

and Sacerdote 2012). Peers might also draw disproportionately on a teacher's time and influence class culture and standards. We find suggestive evidence that exposure to disruptive peers in middle school might drive some of these effects through the development of noncognitive skills. In particular, exposure to lead-poisoned peers increases suspensions and chronic absenteeism, which suggests that noncognitive skills are an important mechanism through which disruptive peers affect long-run outcomes.[5]

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

II. Background: Lead Exposure

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

Federal guidelines mandate that all children on Medicaid be screened for lead poisoning at ages 1 and 2. In addition, North Carolina mandates universal screening in some zip codes based on estimated lead exposure risk. We use these universal testing zip codes, where testing is much higher (about 60% of children living there are tested), in a robustness check later in the paper. Consistent with guidelines, most of the children in our sample who are ever tested are first tested by 13 months of age, and 75% are tested by 25 months of age. Testing is usually done preventatively and not in response to symptoms.

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

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

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

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

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

III. Data Description

A. Education Data

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

While we use the entire sample to calculate the number of children per school-grade-year cohort who have EBLLs (as well as all of our cohort controls), for our main analysis we drop children who do not have siblings and children who live in large buildings, since we cannot reliably identify families in those buildings. Our main analysis also drops students who themselves have an EBLL and estimates the spillover effects of lead exposure on children without known lead poisoning. In section V we perform extensive robustness tests using different samples and specifications. Appendix B (apps. A–C are available online) provides more information on the linkage performed by the North Carolina Education Research Data Center, our sibling identification algorithm, and variable construction.

For our long-run outcomes, we use indicators for high school graduation, dropping out, 2- and 4-year college intentions in twelfth grade, and whether the student took the SAT in high school from 2005 to 2017 (the period over

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

which we can match preschool BLLs of elementary and middle school peers to long-run outcomes).[9] 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 and having at least one out-of-school (OOS) suspension, and the number of days the child was in OOS suspension each year in grades 6–12.[10] We also construct indicators for being suspended on the same day as a lead-exposed cohort peer and for being involved in a behavioral incident with a lead-exposed cohort peer. Because exams changed multiple times over the sample period, we limit our analysis to exams taken between 1996–97 and 2004–5, which were administered to all children and had a similar structure.[11]

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 BLL test, birth month, and birth order. Our cohort-level covariates include the shares of cohort peers who 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 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).

B. Blood Lead Level Data

We obtained the universe of individual BLL test records for children up to age 6 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 micrograms per deciliter, and the child's identifier. We define a child as having an EBLL if their highest BLL is ≥ 5 μg/dL, the upper reference interval value per the 2012 guidelines by the CDC (2013).[12]

Because childhood lead screening is targeted at high-risk children and neighborhoods in North Carolina, we expect screening to be higher among

[9] Dropping out of school is distinct from school switching, death, moving, promotion, graduation, and other confounding factors, and specific reason codes are given for dropping out.

[10] Absences are grouped into 0–7, 8–14, 15–21, and more than 21 days, which is chronic absenteeism. 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.

[11] During our sample period, the scale for the math EOG exam changed in 2001–2. The scale for the reading EOG exam changed in 2002–3.

[12] This value is the 97.5th percentile of BLLs in US children aged 1–5 years from the combined 2007–8 and 2009–10 cycles of the National Health and Nutrition Examination Survey. Starting in 1991 and before 2012, the CDC defined 10 μg/dL as the "level of concern" for children aged 1–5 years. In robustness checks, we vary the definition of EBLL.

low-income children (who also must be screened under the Medicaid mandate).[13] We construct indicators for children missing BLL 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 BLL test. Figure 1 plots the share of children with blood tests and the share of children with EBLLs by birth cohort in our sample, showing that as lead screening increases over time, the incidence of lead poisoning decreases. Still, our identifying variation is not likely to be driven by differences in outcomes between older and younger siblings, as figure A1 (figs. A1–A5 are available online) shows that first-born children have only 1.5 percentage points more lead-exposed peers than their younger siblings. Since earlier-born siblings typically have better outcomes (see, e.g., Black, Devereux, and Salvanes 2005; Conley and Glauber 2006; Price 2008; Booth and Kee 2009), these birth order effects would go in opposite direction to the secular trends depicted in figure 1. Moreover, we generally control for birth order fixed effects.

C. Sample Description

Since our BLL 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; col. 1) and our analysis sample of siblings (1.3 million children; col. 2). In our analysis sample, 39.6% of children have a BLL test, and 10.9% have at least one test greater than or equal to 5 μg/dL, slightly higher shares than in the full sample in column 1. Overall, children with siblings are fairly similar to the full sample, and our results are very similar when we include all children in a model using school-grade and grade-year fixed effects, which lends support to the external validity of our results.

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

[13] According to the North Carolina Department of Health and Human Services' *NC Childhood Lead Testing and Follow-Up Manual*, BLL testing is required for children participating in Medicaid, Health Choice, and/or the Special Nutrition Program for Women, Infants, and Children.

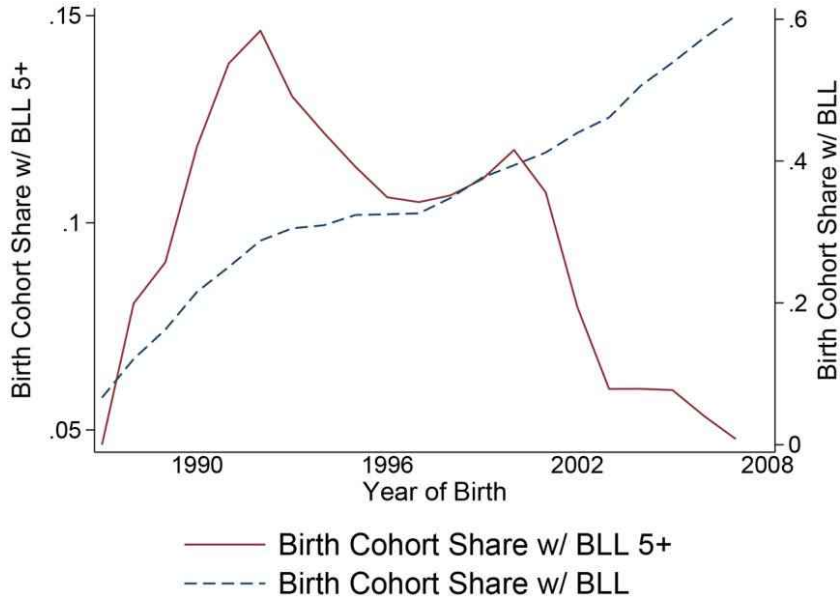

FIG. 1.—Share of children with BLLs at or above 5 μg/dL by birth cohort and share of children with BLL tests by cohort. This figure plots the share of children in a school-grade-year cohort with at least one BLL test (dashed line) and with a BLL of at least 5 μg/dL (solid line). A color version of this figure is available online.

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

IV. Identification Strategy

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

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

Table 1
Characteristics of Children and Schools

	All Children Attending Public School in North Carolina (1)	Children in Sibling Sample (2)	Children with BLL Test (3)	Children with EBLLs (4)	Children without Known EBLLs (5)	Children with Above-Median Share of EBLL Peers in at Least One Elementary Grade (6)	Children with Below-Median Share of EBLL Peers in All Elementary Grades (7)
Average test score	.001	.063	−.117	−.288	.128	−.093	.264
Any OOS suspension	.265	.258	.305	.404	.238	.314	.202
Ever graduated	.837	.872	.866	.816	.881	.848	.895
4-year college intentions	.418	.454	.398	.346	.471	.387	.516
Has taken the SAT	.434	.466	.411	.366	.482	.405	.521
Cohort size	220	225	199	203	229	193	262
Share of teachers with a master's degree	.338	.356	.346	.335	.359	.337	.377
Share economically disadvantaged	.438	.441	.512	.521	.429	.528	.343
Stability rate	.929	.957	.957	.953	.958	.955	.960
Share Black	.277	.266	.285	.331	.256	.309	.217
Share Hispanic	.102	.107	.120	.103	.108	.114	.099
Share with a BLL test	.338	.396	1	1	.322	.533	.256
Share with EBLL	.097	.109	.276	1	0	.165	.052
N students	3,334,365	1,326,622	525,535	144,957	1,181,665	670,559	656,063

NOTE.—This 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 means for all children in our original sample. Column 2 shows means for children with siblings, i.e., our main sample. Column 3 shows means for children that have a BLL test. Column 4 shows means for children with EBLLs, and col. 5 shows means for children without EBLLs. Column 6 shows means for children whose share of elementary school peers with EBLLs was above the median share at the grade-year level in at least one grade, while col. 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).

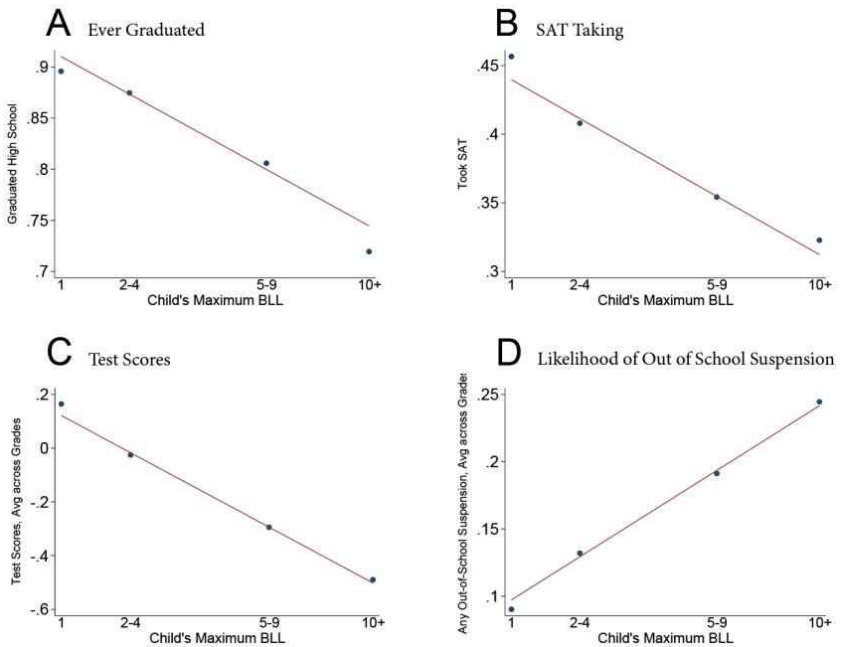

FIG. 2.—Relationship between a child's own BLLs and test scores, OOS suspensions, high school graduation, and SAT taking. This figure plots graduation rates (*A*), SAT-taking rates (*B*), average test scores (*C*), and OOS suspension rates (*D*) by students' BLLs and adds the line of best fit. A color version of this figure is available online.

period, there were relatively few options for choosing public schools.[14] Thus, as we will show, selection into schools was minimal.

Third, unobserved factors might simultaneously cause students and their peers to perform poorly. For example, a child's lead exposure could be correlated with socioeconomic status, which in turn has been associated with peers' learning disruptions (Hoxby 2000; Hoxby and Weingarth 2006). Thus, to causally identify the spillover effect of a child's lead exposure on their peers, we control for the share of cohort peers who are non-White or economically disadvantaged. We also control for the share of the student's peers who have been tested for lead exposure. Because screening rates are higher among students with low socioeconomic status, additionally controlling for screening rates mitigates concerns about selection into testing.

We first examine how lead exposure affects long-run outcomes—that is, graduating from high school, dropping out, intending to attend a 2- or 4-year college, and taking the SAT—of peers without known EBLLs. We compare students who attend the same school but whose grade cohorts randomly happen to have different proportions of children with EBLLs. This specification closely follows the one used by Carrell, Hoekstra, and Kuka (2018) and includes school-grade and grade-year fixed effects. The school-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:

$$Y_{isgt} = \beta_1 \frac{\overline{\sum_{k \neq i} \text{PeersEBLLs}_{ksgt}}}{n_{sgt} - 1} + \pi X_i + \eta_{sg} + \phi_{gt} + \varepsilon_{isgt}, \tag{1}$$

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 in year t. The expression $\overline{\sum_{k \neq i} \text{PeersEBLLs}_{ksgt}}/(n_{sgt} - 1)$ is the average share of the student's peers with known EBLLs across elementary and middle school cohorts, not including the student. The coefficient β_1 captures the effect of having 100% of peers with known EBLLs in elementary and middle school. The term X_i includes gender, race, and birth month fixed effects; economically disadvantaged status; an indicator for whether a child was tested for lead; average share of non-White peers across years; average share of economically disadvantaged peers; average share of peers tested for lead; average school size; school stability rate; and share of teachers with a master's degree over elementary and middle school. For each student, we use grade g and year

[14] North Carolina had no statewide voucher program (until 2015) and relatively few charter schools, which accept students independently of catchment areas (and whose students we observe). Thus, the only way to attend a school different from the one assigned by catchment zone in most places was by moving or attending, and fully paying for, private school. Only 5.3% of all North Carolina children attended private school over this time period (Public Schools First North Carolina 2023).

t from the most recent (i.e., the last) observation we have for that student to maximize sample size. The term η_{sg} is a school-grade fixed effect to account for school-grade-specific shocks. The term ϕ_{gt} is a grade-year fixed effect to account for secular cohort-level trends. We cluster standard errors at the school level to account for arbitrary correlation in the error terms.

However, this specification does not account for the fact that families might select into schools. Thus, in our preferred specification, we compare siblings whose grade cohorts randomly happen to have different proportions of children with EBLLs. Including family fixed effects mitigates the selection problem by controlling for unobserved family characteristics that could be correlated with both peer quality and child's outcomes. Including school fixed effects further controls for students' characteristics that are common to the school's catchment area. Remaining idiosyncratic variation in the BLLs of siblings' cohorts offers plausibly exogenous variation to identify the spillover effects of lead and the effects of peer quality more broadly.[15] Our main estimation equation is thus given by

$$Y_{ijsgt} = \beta_1 \frac{\overline{\sum_{k \neq i} \text{PeersEBLLs}_{ksgt}}}{n_{sgt} - 1} + \pi X_i + \theta_j + \delta_s + \tau_g + \sigma_t + \varepsilon_{ijsgt}, \tag{2}$$

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

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

[15] Of sibling groups in our sample, 97.9% 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 2023). Moreover, schools in our siblings sample have on average 53% of students who go to a school different from that of their siblings, meaning that a majority of students in the sample contribute to estimating the school fixed effects. Figure A2 shows the distribution of our regressor of interest, the average share of a student's peers with EBLLs over elementary and middle school, and the distributions of residuals obtained from regressions of this variable on our preferred set of controls and the fixed effects in eqs. (1) and (2).

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

V. Results

A. Long-Run Effects of Peers Exposed to Lead

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

Panel A of table 2 shows estimates of β_1 from equation (1), which includes school-grade and grade-year fixed effects. We find that a 10% increase in the average share of elementary and middle school peers with EBLLs decreases the likelihood of a student graduating and taking the SAT by 1.6 and 2.8 percentage points, respectively, and increases the likelihood that a student drops out of school by 1.5 percentage points. Panel B of table 2 presents estimates of β_1 from equation (2), which exploits within-sibling variation and is our preferred specification. Within-sibling comparisons generally estimate slightly smaller effects than comparisons within a school-grade, suggesting that family fixed effects better control for endogenous selection.[16]

Our preferred specification in panel B shows that a child whose average cohort in elementary and middle school has 10% more lead-poisoned peers has a 1.7 percentage point lower likelihood of graduating high school—a 2% decrease on the mean graduation rate of 89%. We also find that having 10% more lead-poisoned peers increases the likelihood of dropping out by 0.5 percentage points and decreases the likelihood of taking the SAT while in high school by 2.3 percentage points, or a 4.3% decrease on the mean rate of 53.2%.[17] While a higher share of lead-poisoned peers decreases the likelihood that a student intends to attend a 4-year college in panel A, this result is not statistically significant at conventional levels in our preferred specification. Finally, after controlling for the share of lead-poisoned peers, we find little

[16] Because we identify siblings based on home addresses, our sibling-matching algorithm could lead to error. Panel B of table A1 (tables A1–A14, B1–B4, C1 are available online) estimates the same specification as panel A of table 2 on the sibling sample and finds similar results using the sibling vs. all-children samples. Furthermore, col. 1 of table 7 shows results on the sample of census tracts where the majority of homes are single-family homes, where sibling attribution is more precise.

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

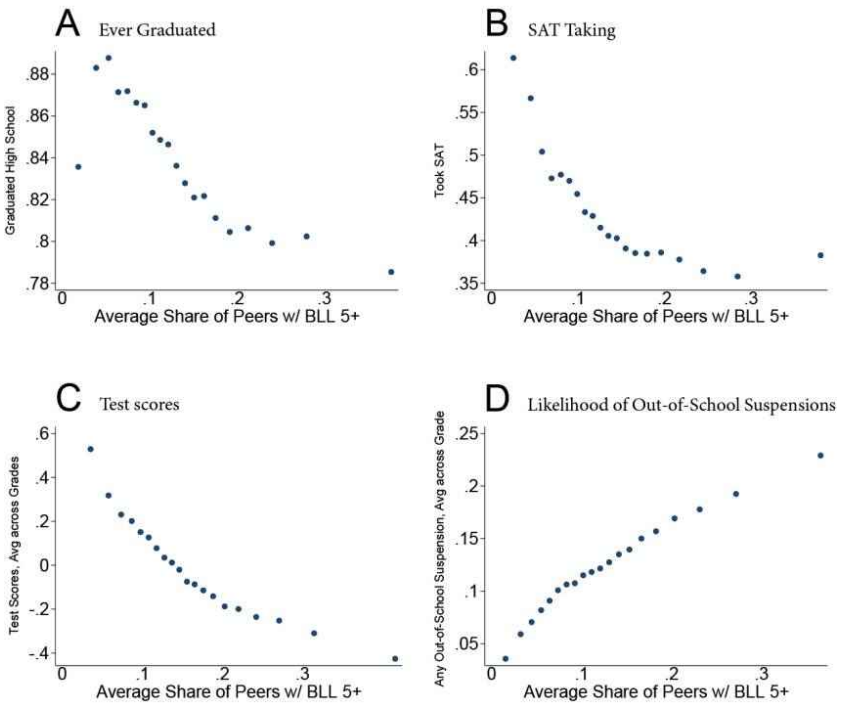

FIG. 3.—Relationship between peers' BLLs and test scores, OOS suspensions, high school graduation, and SAT taking. This figure plots graduation rates (*A*), SAT-taking rates (*B*), average test scores (*C*), and OOS suspension rates (*D*) by vigintile of students' share of peers with BLLs at or above 5 μg/dL. A color version of this figure is available online.

Table 2
Long-Run Outcomes of Exposure to Peers with EBLLs by Timing of Exposure

	Ever Graduated (1)	Ever Dropped Out (2)	Intention to Attend a 4-Year College (3)	Intention to Attend a Community College (4)	Took the SAT (5)
	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 ≥5 μg/dL	−.1558***	.1453***	−.1257***	−.0112	−.2769***
	(.0173)	(.0150)	(.0360)	(.0343)	(.0349)
Romano-Wolf adjusted *p*-value	.002**	.002**	.002**	.988	.002**
Mean of outcome	.8491	.0597	.4407	.3544	.4589
N students	831,147	1,155,293	666,613	665,951	657,670
	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 ≥5 μg/dL	−.1671***	.0470+	−.1171	.0457	−.2283**
	(.0350)	(.0246)	(.0720)	(.0785)	(.0740)
Romano-Wolf adjusted *p*-value	.002**	.040*	.118	.926	.002**
Mean of outcome	.8904	.0529	.5069	.3288	.5320
N students	282,964	414,562	205,760	205,688	201,713
	C. Share of Elementary versus Middle School Peers with EBLLs with Sibling, School, Grade, and Year Fixed Effects				
Share of peers with BLLs ≥5 μg/dL in elementary school	−.0611*	−.0064	−.0104	.0240	−.0024
	(.0295)	(.0218)	(.0671)	(.0685)	(.0669)
Romano-Wolf adjusted *p*-value	.016*	.988	.990	.986	.990
Share of peers with BLLs ≥5 μg/dL in middle school	−.1184**	.0710*	−.0545	−.0106	−.2214*
	(.0414)	(.0299)	(.0906)	(.0987)	(.0872)
Romano-Wolf adjusted *p*-value	.002**	.006**	.926	.990	.006**
p-value elementary =middle	.35	.08	.75	.81	.10

Table 2 (*Continued*)

	Ever Graduated (1)	Ever Dropped Out (2)	Intention to Attend a 4-Year College (3)	Intention to Attend a Community College (4)	Took the SAT (5)
Mean of outcome	.8945	.0519	.5108	.3299	.5380
N students	248,391	354,195	182,305	182,248	178,831

NOTE.—This table reports the effect of a child's share of peers with EBLLs on the child's long-run outcomes. Column 1 reports the effects on the likelihood a student ever graduates from high school, and col. 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 4-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-grade and grade-year fixed effects. Panels B and C instead include family, school, grade, and year fixed effects, controlling for birth order and birth month. All regressions include individual controls for gender, race, whether the student has a BLL test, and economically disadvantaged status measured in the highest grade a student is observed in. We also control for the average share of elementary and middle school peers that are non-White or economically disadvantaged, average share of children with a BLL test, school size, stability rate, and percentage of teachers with a master's degree averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level. We also report *p*-values corrected for multiple hypothesis testing. We use the Stata command rwolf2 based on Clarke, Romano, and Wolf (2020), which allows for dependence among *p*-values by bootstrap resampling.

$^{+}$ $p < .10$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

evidence of any effect of economically disadvantaged or non-White peers on graduation and dropout rates and an effect of economically disadvantaged peers on SAT taking that is less than half the effect of lead-poisoned peers.

We estimate effects on college going that are similar in magnitude to those obtained by Carrell, Hoekstra, and Kuka (2018). Those authors find that adding one male peer exposed to domestic violence to a classroom decreases 4-year college going by 1.4 percentage points. Using our cohort results and assuming that there are 25 students in a class, we calculate that one additional lead-poisoned peer in each class, a 4% increase in the share of lead-poisoned peers, would lead to a 0.92 percentage point reduction in the likelihood of taking the SATs, a proxy for college intentions, and a 0.67 percentage point reduction in graduating high school.

Next, we show that spillovers of lead poisoning increase both with the share of lead-exposed peers and with the severity of peers' lead exposure. Figure 4*A* plots estimates from equation (2) using bins for different percentages of cohort peers with EBLLs (0%–5%, 5%–10%, 10%–15%, 15%–20%, 20%–100%), where the 0%–5% bin is the omitted category. We also test whether estimates are statistically different from the 5%–10% bin coefficient and find a statistically significant stronger effect of lead-poisoned peers on graduation rates as the percentage of peers with EBLLs increases above 15%. In figure 4*B* we show the effects of the average share of lead-poisoned peers on the likelihood of graduation using different thresholds to define peers' lead poisoning (e.g., BLL ≥ 2 μg/dL, BLL ≥ 3 μg/dL). As the EBLL threshold increases, so does

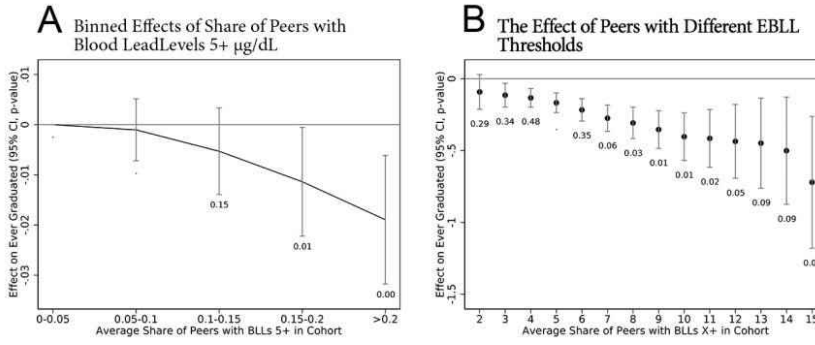

FIG. 4.—Spillover effects on graduation by binned share of peers with BLLs ≥5 µg/dL and by different EBLL thresholds. *A* plots nonparametric estimates of the effect of having different proportions (binned) of peers with BLLs ≥5 in a child's cohort on the likelihood of high school graduation, where the omitted category is an indicator for share of peers with BLLs ≥5 that is lower than 0.05. The *p*-values for each bin relative to the 5%–10% bin are shown under each confidence interval. *B* plots the effect of the average share of peers with different EBLLs >*x* in the cohort on graduation. As the BLLs increase, so does the negative effect on graduation. The *p*-values relative to our baseline coefficient are shown under each confidence interval. In both panels, we control for all fixed effects and controls in our primary specification (which includes family, school, year, and grade fixed effects and individual and demographic controls by cohort, averaged over elementary and middle school). Vertical bars represent 95% confidence intervals based on standard errors clustered at the school level.

the negative effect on graduation.[18] Estimates with lead poisoning defined as BLL ≥ 7 µg/dL are significantly larger than our main estimates (defined as BLL ≥ 5 µg/dL). These results suggest that BLLs drive these peer effects rather than other potentially correlated characteristics of children.

Because peers might impact each other differently at different ages, in panel C of table 2 we show the long-run effects of lead-poisoned peers from elementary and middle school cohorts separately. Peers in middle school could be especially impactful for long-run outcomes if middle school is when students decide whether to remain in school. Recent interventions in middle school have been very effective at reducing crime, suspensions, and dropping out of school, suggesting that students' outcomes can be strongly affected beyond early childhood.[19] Indeed, we find that our estimated long-run effects appear to be largely driven by middle school

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

[19] See, e.g., Guryan et al. (2021) and Heller et al. (2017).

peers.[20] Yet we note that elementary and middle school peers are highly correlated.

B. Mechanisms: Contemporaneous Effects of Peers Exposed to Lead

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

$$Y_{isgt} = \beta_1 \frac{\sum_{k \neq i} \text{PeersEBLLs}_{ksgt}}{n_{sgt} - 1} + \pi X_{it} + \omega S_{sgt} + \eta_{sg} + \phi_{gt} + \gamma_e + \varepsilon_{isgt}, \tag{3}$$

$$Y_{ijsgt} = \beta_1 \frac{\sum_{k \neq i} \text{PeersEBLLs}_{ksgt}}{n_{sgt} - 1} + \pi X_{it} + \omega S_{sgt} + \theta_j + \delta_s + \tau_g + \sigma_t + \gamma_e + \varepsilon_{ijsgt}, \tag{4}$$

where $\Sigma_{k \neq i}\text{PeersEBLLs}_{ksgt}/(n_{sgt} - 1)$ 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. The coefficient β_1 on $\Sigma_{k \neq i}\text{PeersEBLLs}_{ksgt}/(n_{sgt} - 1)$ captures the effect of having 100% of a child's peers in a given year with known EBLLs. Equation (3) mirrors equation (1) by including school-grade (η_{sg}) and grade-year (ϕ_{gt}) fixed effects. The term X_{it} is a vector of child-specific control variables, including gender, race, and birth month fixed effects; economically disadvantaged status in each year; and an indicator for whether a child was tested for lead. The vector S_{sgt} controls for time-varying characteristics at the school-grade-year level: percentage of non-White students, percentage of economically disadvantaged students, and share of students who have been tested for lead exposure. We also control for school time-varying characteristics: annual school size, share of teachers with a master's degree, and school-level stability rate. In equation (4), θ_j, δ_s, τ_g, and σ_t are family, school, grade, and year fixed effects, as in equation (2). In addition, when we look at test scores, we include γ_e, an exam fixed effect that restricts our comparison to children who took the same test.

Panel A of table 3 presents the results for the effect of additional cohort peers who are lead poisoned on a child's outcomes using equation (3), while panel B uses our preferred specification in equation (4).[21] The two panels show very similar results: a higher share of peers with EBLLs is associated

[20] While the coefficients on middle school peers are always larger in magnitudes than those on elementary school peers, we detect a statistically significant difference (at the 10% level) only for dropping out and taking the SAT.

[21] The sample size is smaller than in col. 1 of table 1 because of singletons and missing outcomes.

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

	Average Test Score (1)	OOS Suspension (2)	Days Suspended (3)	OSS Suspension Same Day as Lead-Exposed Child (4)	Incident with Lead-Exposed Child (5)	Absent 22 or More Days (6)
	A. Cohort Peers with School-Grade and Grade-Year Fixed Effects					
Share of peers with BLLs ≥5 μg/dL	−.0272	.0677***	1.0974***	.2250***	.1107***	.0234*
	(.0351)	(.0188)	(.2424)	(.0143)	(.0118)	(.0091)
Romano-Wolf adjusted *p*-value	.196	.020*	.020*	.020*	.020*	.020*
Observations	3,284,720	7,916,670	7,916,670	7,189,301	6,540,081	8,128,020
N students	930,228	1,906,345	1,906,345	1,883,489	1,764,684	1,902,185
Mean of outcome	.0572	.1048	.7732	.0318	.0202	.0611
	B. Cohort Peers with Family, School, Grade, and Year Fixed Effects					
Share of peers with BLLs ≥5 μg/dL	.0190	.0205^{+}	.6099***	.1929***	.1041***	.0434***
	(.0361)	(.0117)	(.1807)	(.0106)	(.0082)	(.0072)
Romano-Wolf adjusted *p*-value	.373	.020*	.020*	.020*	.020*	.020*
Observations	1,409,299	4,287,750	4,287,750	3,919,448	3,672,544	4,395,695
N students	373,801	944,335	944,335	933,508	891,259	939,430
Mean of outcome	.1298	.0942	.6762	.0285	.0187	.0520

NOTE.—This table reports the effect of a child's share of peers with EBLLs on the child's school outcomes. Both panels use the share of peers with maximum BLLs ≥5 μg/dL at the school-grade-year level as the main explanatory variable. Panel A includes school-grade, grade-year, and birth month fixed effects. Panel B instead includes family, school, grade, and year fixed effects, controlling for birth order. In col. 1 we take the average of math and reading test scores and additionally control for subject–test type fixed effects. In cols. 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 BLL test, share of non-White peers, share of children with a BLL test, and share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, stability rate, and percentage of teachers with a master's degree. Standard errors are in parentheses and clustered at the school level. We also report *p*-values corrected for multiple hypothesis testing. We use the Stata command rwolf2 based on Clarke, Romano, and Wolf (2020), which allows for dependence among *p*-values by bootstrap resampling.

$^{+}$ $p < .10$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

with a higher likelihood of, and longer, OOS suspensions and a higher likelihood of absences. Again, within-sibling comparisons generally estimate slightly smaller effects than comparisons within a school-grade.[22]

[22] Because we observe long-run outcomes for only early cohorts of students, students move out of state, and some data become available only in later years, the numbers of students differ for short- and long-run outcomes. However, in panel A of

In panel B we find that a 10% increase in the proportion of cohort-level peers with EBLLs in a given year leads to a 0.2 percentage point increase in the likelihood of OOS suspensions—a 2.1% increase over the mean of 9.4%—and increases the suspension duration by 40 minutes based on a 6-hour school day. Moreover, these increased suspensions appear to be driven at least in part by suspensions on the same day as suspensions for lead-poisoned children and behavioral incidents including lead-poisoned children.[23]

Increased suspensions for peers of lead-poisoned children could be due to more punitive policies at the cohort-level, such as teachers' responses. To disentangle peers' behavior from school policies, we look at the effects of lead-poisoned peers on absences, which should not be driven by school policies. We find that a 10% increase in the proportion of cohort-level peers with EBLLs increases the likelihood of chronic absenteeism by 0.4 percentage points, or 8% on a base of 5.2%, suggesting that our results are not driven by school policies. Finally, we find little evidence of lead-poisoned students affecting their peers' test scores, with estimates changing sign across specifications. This finding suggests that the effects of lead-poisoned peers on the long-run outcomes described in section V.A may operate through noncognitive skills and behavior rather than a learning channel.[24] Yet we cannot fully disambiguate between these channels.

While we use cohort-level variation in our primary specification to avoid the issue of selection into classrooms by students, table A2 presents the estimates of the effect of having more lead-poisoned peers in the same classroom.[25] Classroom peers have a larger effect on suspensions and absences than cohort peers, which could be due to stronger connections within or selection into classrooms.

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

Because exposure to lead-poisoned peers could interact with a child's background to shape their outcomes, we next study heterogeneity in peer

table A1 we estimate our primary specification only on students for whom we observe outcomes consistently. The results on this sample are very similar to our main results in tables 2 and 3.

[23] Placebo estimates in fig. A3 show that our estimated effects of higher shares of peers with EBLLs on the likelihood of being suspended or involved in an incident with a student with an EBLL are 154%–165% of what the mere proportion of children with EBLLs in a given cohort implies.

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

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

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

We find some evidence of heterogeneous effects of lead-poisoned peers on graduation by race and gender. Black students see the largest decrease in high school graduation from lead-poisoned peers. A 10% increase in the average share of lead-poisoned peers in elementary and middle school decreases the likelihood Black students graduate high school by 3 percentage points, compared with 1.5 percentage points for White students. Boys also seem more affected than girls by lead-poisoned peers, although the difference is not statistically significant. Importantly, Black and male students have lower graduation rates to start with, suggesting that peers might exacerbate existing educational disparities. However, we find little systematic evidence of heterogeneity by socioeconomic status.[26]

We also hypothesize that friend groups might drive peer effects.[27] As we lack data on friendship networks, we use the fact that children likely sort into groups with similar characteristics (Jackson 2010). Table A4 presents both the effect of exposure to a higher share of lead-poisoned peers and the additional effect of exposure to a higher share of lead-poisoned peers of the same gender (panel A), race (panel B), and same gender and same race (panel C). We find that peers of the same gender, but not peers of the same race, with EBLLs have an additional effect on high school graduation on top of the general effect generated by all lead-exposed peers, although the results are statistically significant only at the $p < .1$ level. Moreover, panel A of table A5 shows that lead-poisoned boys have larger negative effects on their peers than lead-poisoned girls for SAT taking. Together, these results suggest that peer effects are mediated by assortative matching of peer groups.

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

We do not observe lead exposure for all children, and there may be selection in who is tested for lead. Since we compute the share of lead-poisoned peers

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

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

Table 4
Heterogeneity by Demographic Subgroups, All Long-Run Outcomes

	Ever Graduated (1)	Ever Dropped Out (2)	Intention to Attend a 4-Year College (3)	Intention to Attend a Community College (4)	Took the SAT (5)
	A. White, Non-Hispanic Students				
Share of peers with BLLs ≥5 μg/dL	−.1467***	.0489	−.0952	.0013	−.1202
	(.0404)	(.0301)	(.0890)	(.0985)	(.0871)
	B. Black, Non-Hispanic Students				
Share of peers with BLLs ≥5 μg/dL	−.3140***	.0384	.0465	.0454	−.3714*
	(.0768)	(.0526)	(.1731)	(.1588)	(.1683)
p-value =White	.05	.86	.47	.81	.19
	C. Hispanic Students				
Share of peers with BLLs ≥5 μg/dL	−.1367	.0324	−.1250	−.1480	−.4038+
	(.1250)	(.0823)	(.2231)	(.2452)	(.2403)
p-value =White	.94	.85	.90	.57	.27
	D. Never Economically Disadvantaged Students				
Share of peers with BLLs ≥5 μg/dL	−.0911*	.0257	−.0419	−.0645	−.0157
	(.0401)	(.0222)	(.1041)	(.1138)	(.1106)
	E. Sometimes Economically Disadvantaged Students				
Share of peers with BLLs ≥5 μg/dL	−.1809**	.0476	−.1700	.1566	−.2764*
	(.0682)	(.0457)	(.1388)	(.1400)	(.1361)
p-value =never	.26	.67	.46	.22	.14
	F. Always Economically Disadvantaged Students				
Share of peers with BLLs ≥5 μg/dL	.0266	−.0445	−.1496	−.0163	−.1426
	(.1032)	(.0776)	(.1813)	(.2028)	(.2019)
p-value =never	.29	.38	.61	.84	.58
	G. Girls				
Share of peers with BLLs ≥5 μg/dL	−.1105+	.0328	−.0546	.0556	−.2376+
	(.0605)	(.0403)	(.1306)	(.1428)	(.1311)

Table 4 (*Continued*)

	Ever Graduated (1)	Ever Dropped Out (2)	Intention to Attend a 4-Year College (3)	Intention to Attend a Community College (4)	Took the SAT (5)
	H. Boys				
Share of peers with BLLs ≥5 μg/dL	−.2513***	.0724	−.0592	.0276	−.2072
	(.0668)	(.0486)	(.1295)	(.1461)	(.1358)
p-value =girls	.12	.53	.98	.89	.87

NOTE.—This table reports the effect of a child's share of peers with EBLLs 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 BLL test, gender, race, and economically disadvantaged status. Cohort controls include share of non-White peers, share of children with a BLL test, and share of peers who are economically disadvantaged at the school-grade-year level. We also control for school size, stability rate, and percentage of teachers with a master's degree. Cohort and school controls are averaged over elementary and middle school. Standard errors are clustered at the school level.

$^{+}$ $p < .10$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

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

$$\bar{Y}_{isg} = \beta_1 \text{LeadPoisoned}_{isg} + \pi X_i + \eta_{bt(i)} + \delta_s + \tau_g + \varepsilon_{isg}, \quad (5)$$

where $\bar{Y}_{isg}$ represents the average outcomes of student i's peers in a cohort in school s as of grade g, that is, the last grade we observe a student. The term $\text{LeadPoisoned}_{isg}$ is an indicator for student i attending school s in grade g having an EBLL by age 6. Thus, the coefficient of interest, β_1, estimates the effect of child i being lead poisoned on the average outcomes of their peers relative to other screened children who tested negative. We include block group–birth year fixed effects ($\eta_{bt(i)}$), as well as grade (τ_g) and school (δ_s) fixed effects. The term X_i includes gender, race, economically disadvantaged status, birth month, and birth order fixed effects; share of non-White peers; share of economically disadvantaged peers; share of peers tested for lead; share of peers with EBLLs; student's school size; school stability rate;

and share of teachers with a master's degree in the last grade we observe student *i*. The term ε_{isg} represents the error term.

We use block group–birth year fixed effects to absorb any time-varying neighborhood characteristics, including gentrifying patterns that could be correlated with home renovations and time-varying pollution that could correlate with lead poisoning. Thus, we identify the effect of lead poisoning by comparing peers' outcomes of children living in the same small neighborhood and time but who happen to have different EBLLs. Abbasi, Pals, and Gazze (2020) suggest that even within a census block, the age of the housing is highly predictive of BLLs, so we aim to capture this source of variation with this specification. In table 5, panel A, we find that one lead-poisoned student in a cohort of 220 is associated with a 0.09 percentage point decrease in the share of peers who graduate high school or take the SAT when using our full sample of BLL tests. This is very similar to what we obtain if we scale our main result in table 2, panel B.[28]

Table A6 further investigates whether our results might be driven by characteristics of the lead-poisoned student that are correlated with that student's lead poisoning, such as family background. We note that adding individual controls (gender, race, economic disadvantage) in column 2 decreases our estimates by about a quarter, in line with our suggestive findings of potential homophily by gender, for example. Yet adding additional controls for parental education, family composition (number of children in family and siblings' gender mix), and school controls (share non-White, share economically disadvantaged, school size, stability rate, and percentage of teachers with a master's degree) does not alter our estimates from equation (5) significantly (cols. 3, 4). Finally, column 5 shows that controlling for family fixed effects in this specification decreases our sample size by two-thirds and our effective sample size by 85%. Consistently, estimates in column 5 are noisier, with smaller point estimates in the whole sample (panel A) but much larger point estimates in the samples of children tested by 37 and 25 months (panels B and C, respectively).[29]

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

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

Table 5
Effects of Having an EBLL on Peers' Average Long-Run Outcomes Using Block Group–Birth Year Fixed Effects

	Ever Graduated (1)	Took the SAT (2)
	A. Tested by 72 Months of Age	
Child's maximum BLL is ≥5	−.0009**	−.0009***
	(.0003)	(.0002)
Observations	338,232	322,115
Mean of outcome	.6153	.2740
	B. Tested by 37 Months of Age	
Child's maximum BLL is ≥5	−.0011*	−.0011***
	(.0004)	(.0003)
Observations	225,382	232,965
Mean of outcome	.6217	.2673
	C. Tested by 25 Months of Age	
Child's maximum BLL is ≥5	−.0008^{+}	−.0009**
	(.0005)	(.0003)
Observations	190,155	198,367
Mean of outcome	.6226	.2636

NOTE.—This table reports the effect of one additional child with EBLLs on the average long-run outcomes for their peers (eq. [5]). Column 1 reports the effects on the likelihood peers ever graduate from high school, and col. 2 shows the effects on the likelihood of ever taking the SAT. Panel A uses all BLL tests in our sample. Panels B and C limit the sample to children screened by 37 and 25 months of age, respectively. We control for census block group–birth year fixed effects, as well as school, grade, birth month, and birth order fixed effects. We also control for share of peers with EBLLs, share of peers who are non-White or economically disadvantaged, share of children with a BLL test, school size, stability rate, and percentage of teachers with a master's degree. Standard errors are in parentheses and clustered at the school level.

$^{+}$ $p < .10$.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

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

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

effect. Thus, selection into testing is unlikely to bias our findings in an economically significant way.

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

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

$$\begin{aligned}\text{ImputedShareEBLL}^{p}_{sgt} = &\left(\frac{\sum_{k\neq i}\text{PeersEBLLs}_{ksgt}}{\text{NTested}_{sgt}-1}\right)\left(\frac{\text{NTested}_{sgt}-1}{N_{sgt}-1}\right)\\ &+p_s\left(\frac{\sum_{k\neq i}\text{PeersEBLLs}_{ksgt}}{\text{NTested}_{sgt}-1}\right)\left(1-\frac{\text{NTested}_{sgt}-1}{N_{sgt}-1}\right),\end{aligned} \tag{6}$$

where $p_s[\Sigma_{k\neq i}\text{PeersEBLLs}_{ksgt}/(\text{NTested}_{sgt}-1)]$ is the pth percentile of the distribution of observed shares of students with EBLLs within school s (or the district the school belongs to), NTested_{sgt} is the number of tested children in the school-grade-year, and N_{sgt} is the total number of students in the school-grade-year.

In panels A and B of table 6, we use the 90th and 10th percentiles of the empirical distribution of observed BLLs by school for missing BLL data, respectively. Imputing the 90th percentile of the distribution of children with EBLLs reduces the magnitude of our estimates on graduation and SAT taking by 25%–40%, but both coefficients remain negative and statistically significant at the $p < .01$ level.[30] Imputing the 10th percentile instead produces estimates that are very similar to our preferred ones in panel B of table 2. Panels C and D repeat this exercise using the 90th and 10th percentiles within school districts, respectively, and yield similar outcomes, albeit attenuated likely because of wider averaging. Finally, panel E presents results where we

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

Table 6
Bounding Our Estimates to Account for Missing BLL Data

	Ever Graduated (1)	Took the SAT (2)
	A. Imputing the 90th Percentile of the BLL Distribution by School for Missing BLL Data	
Share of peers with BLLs ≥5 μg/dL	−.0882***	−.1396**
	(.0231)	(.0514)
Mean of outcome	.8904	.5320
N students	282,964	201,711
	B. Imputing the 10th Percentile of the BLL Distribution by School for Missing BLL Data	
Share of peers with BLLs ≥5 μg/dL	−.1370***	−.1980**
	(.0318)	(.0642)
Mean of outcome	.8904	.5320
N students	282,964	201,711
	C. Imputing the 90th Percentile of the BLL Distribution by District Cohort for Missing BLL Data	
Share of peers with BLLs ≥5 μg/dL	−.1121***	−.1672***
	(.0261)	(.0420)
Mean of outcome	.8904	.5320
N students	282,964	201,711
	D. Imputing the 10th Percentile of the BLL Distribution by District Cohort for Missing BLL Data	
Share of peers with BLLs ≥5 μg/dL	−.0908***	−.1628***
	(.0204)	(.0422)
Mean of outcome	.8904	.5320
N students	282,964	201,711
	E. Using Predicted BLLs	
Share of peers with BLLs ≥5 μg/dL	−.1216***	−.2201***
	(.0304)	(.0664)
Mean of outcome	.8939	.5408
N students	264,999	189,611

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

** $p < .01$.

*** $p < .001$.

predict BLLs for the sample of unscreened children based on our rich school and census data and set of fixed effects and use the predicted shares of EBLLs for the share of missing BLLs. Again, we obtain estimates similar to those in table 2. We interpret these findings as suggestive that missing BLL data are not causing us to attribute spurious effects to lead-poisoned peers. As an additional check, column 2 of table 7 shows the effects of lead-poisoned peers on children in universal screening zip codes, where screening rates are 16% higher than average.[31] We find only a slightly larger effect on graduation than in the full sample.

To address further concerns on measurement of lead poisoning, in panels B–D of table A5, we show that our results are largely robust to using different measures of lead-exposed peers. Moreover, missing information on a student's own lead-poisoning status does not bias our estimates: when we estimate the effects of lead-poisoned peers only on students who have tested negative for lead poisoning (panel E), we obtain coefficients that are very similar to those in table 2. The same is true when we also include children who tested positive (panel F), suggesting that spillover effects are not mediated by one's own poisoning status.

E. Additional Threats to Internal Validity

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

We also test whether our estimated effect of lead poisoning is driven by the socioeconomic status of the lead-poisoned children. To do so, we estimate

[31] These zip codes cover at least one block group with 27% or more homes built before 1950 and areas with high prevalence of EBLLs (Hanchette 1999). Table A9 repeats the robustness exercise shown in table 7 for SAT taking, as well as OOS suspensions and chronic absenteeism. Our results on SAT taking and absences are very robust to different samples and specifications. Our suspensions results are similarly robust in terms of magnitude but noisy at times.

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

	>50% of Homes in Census Tract Are Single Family (1)	Zip Codes with Universal Screening (2)	Adding Cohort Block Group Characteristics (3)	Adding Block Group Fixed Effects (4)	Adding School-Grade Fixed Effects (5)	Adding School-Year Fixed Effects (6)	Zip Codes with No School Choice Options (7)	Adding Sibling-School Fixed Effects (8)
Share of peers with BLLs ≥5 μg/dL	−.1846*	−.1851***	−.1713***	−.2234***	−.1467***	−.2293***	−.2668***	−.1030***
	(.0719)	(.0453)	(.0345)	(.0591)	(.0308)	(.0449)	(.0714)	(.0266)
N students	84,711	146,559	282,962	118,713	282,514	281,789	175,941	228,002
Mean of outcome	.8830	.8800	.8904	.8835	.8913	.8925	.8934	.9094
School fixed effects	X	X	X	X			X	
Sibling fixed effects	X	X	X	X	X	X	X	
Year fixed effects	X	X	X	X	X		X	X
Grade fixed effects	X	X	X	X		X	X	X

NOTE.—This table reports the effect of a child's share of peers with EBLLs on the child's school outcomes. Each column reports results from a separate regression. Column 1 restricts the sample to census tracts where more than half of homes are single-family homes. Column 2 restricts the sample to students who live in zip codes that are subject to universal lead screening. Columns 3–6 and 8 add controls and alternative sets of fixed effects, as specified at the top and bottom of each column. Column 7 restricts the sample to zip codes without charter schools or voucher programs. Block group characteristics of cohort peers include share of peers that live in block groups with above-median income, above-median percentage Black and Hispanic population, above-median percentage of the population living in poverty, and above-median percentage of the 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 BLL test. Cohort controls include share of non-White peers, share of children with a BLL test, and share of peers who are economically disadvantaged. We also control for school size, stability rate, and percentage of teachers with a master's degree. Cohort and school controls are averaged over elementary and middle school. Standard errors are in parentheses and clustered at the school level.

* $p < .05$.

*** $p < .001$.

the effect of lead-poisoned children with higher income and who are White (table A10, panels A and B) and further control for peers' parental education (table A10, panel C). Our results are robust to these alternative specifications, which suggests that our results are not driven by socioeconomic status of children with EBLLs.

To further rule out spurious correlation, table A11 shows the robustness of our specification to different sets of controls. When we omit all controls other than family, school, grade, and year fixed effects and the share of peers tested for lead (panel A), estimates are similar to those in our main specification, albeit slightly larger. Panel B shows that once we add individual and school-level controls, omitting the average share of students who are non-White and the average share of students who are economically disadvantaged does not affect our estimates compared with our main results. So peers' characteristics other than lead poisoning do not appear to explain much of the variation in students' outcomes after including the set of fixed effects and controls that provides our identification. Relatedly, table A12 shows limited evidence that peers' composition at the cohort level is related to school quality or resources in a way that could confound our estimates. Cohorts with a higher share of students with EBLLs appear to be in school-years with a higher stability rate, if anything, and larger student bodies.[32] Panel C of table A11 shows that excluding school fixed effects yields slightly larger peer effects on suspensions compared with our main results. These results suggest that our more conservative primary specification controls for unobserved time-invariant school characteristics. Our results also hold when we add school-specific linear time trends to our preferred specification (panel D).

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

Because the incidence of lead poisoning has decreased over time (fig. 1), our primary estimates might capture similarly occurring trends in outcomes, despite controlling for grade and year fixed effects. To assuage this concern,

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

in column 6 of table 7, we control for school-year fixed effects and find peer effects that are larger than our main results.

Endogenous sorting into peer groups could also bias our results if high-achieving students sort out of cohorts with many lead-poisoned students, for example. Importantly, most of North Carolina did not offer school choice options for public schools up until the 2014–15 school year, unless students switched to a charter or magnet school, which we observe.[33] Column 7 of table 7 shows that our results are larger for children in zip codes with no charter schools or other school choice options (at the time), which are effectively no-choice zip codes. Column 8 of table 7 controls for siblings-school fixed effects, effectively comparing siblings only in grades during which they attend the same school, as in Bertoni, Brunello, and Cappellari (2020). We find spillover effects of lead-poisoned peers that are two-thirds the size of our main result. Finally, table A13 formally investigates the association between a student's share of lead-poisoned peers and students or their siblings switching to public or charter schools. The results indicate that endogenous sorting is not a concern in our setting.

Finally, including siblings fixed effects could lead us to underestimate the spillover effects of lead exposure in the presence of within-family spillovers. For example, if parents respond to their children's performance by shifting resources across offspring, a student's peers could also affect the outcomes of that student's siblings. Table A14 shows no evidence of this by controlling for the share of lead-exposed peers of a student's siblings. Our results are also similar when we add controls for sibling gender composition and family size to our preferred specification (table A11, panel E).

VI. Conclusion

This is the first study showing that pollution has long-run spillover effects on school peers. By comparing siblings, we show that a child's own lead exposure spills over to affect other children's long-run outcomes, including high school graduation and SAT taking. These effects suggest that the social cost of lead exposure has been underestimated so far. In addition, we reveal some mechanisms through which peer effects manifest—namely, behavior shaping while in middle school likely through noncognitive skills. Thus, our findings have implications for other types of common pollution that have been linked to suspensions from school, such as traffic and industrial pollution (Persico and Venator 2021; Heissel, Persico, and Simon 2022), suggesting

[33] In the 2014–15 school year North Carolina implemented the Opportunity Scholarships program, a voucher program for low-income children. Children whose families make less than 133% of the qualifying amount for the federal free or reduced-price lunch program qualify for the voucher, which can be used for any school. Because the Charlotte-Mecklenburg public school district has had a school choice program since 2002, we exclude it in this robustness check.

that the cost of pollution has been underestimated. These findings hold even though we likely underestimate the effect of lead-poisoned peers because of potential within-family spillovers and measurement error. Thus, environmental hazards appear to contribute to human capital accumulation, even for children who are not themselves exposed to these hazards.

While external validity issues make it difficult to extrapolate how lead exposure might affect labor market outcomes, we attempt a back-of-the-envelope calculation for the social cost of the spillovers of lead poisoning. We find that being exposed to one additional lead-poisoned peer in a cohort of 220 is associated with $84 in lost earnings per student from lower graduation rates alone, that is, excluding the additional costs of behavioral issues and absences.[34] This estimate implies a spillover effect of a lead-poisoned child of $18,368 on their 219 school peers. As half a million young children are poisoned by lead each year (Aizer et al. 2018), these spillovers total almost $9.2 billion per birth-year cohort. Reyes (2014) estimates that the direct social cost of lead poisoning is $200 billion per birth-year cohort. Thus, our lower-bound estimates suggest that the social cost of lead has been underestimated by at least 4.6% by not including these spillover effects. Because lead-poisoned students are quite dispersed across schools, 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 BLLs and test scores (Sorensen et al. 2019). In addition, Billings and Schnepel (2018) show that offering early interventions for lead-poisoned children improves their school performance and decreases antisocial behavior, for a total benefit of $9,666 per directly exposed child. Our estimates suggest an additional potential benefit of about twice the direct benefit when accounting for the spillover effects of lead poisoning on school peers.

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

[34] 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 nonparametrically 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% and a discount rate of 3.38% (i.e., the 30-year Treasury bond rate). Thus, one child with an EBLL in a cohort decreases the net present value of lifetime earnings by $0.0009 \times \$93{,}188 = \$84$.

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