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Toxic Neighborhoods: The Effects of Concentrated Poverty and Environmental Lead Contamination on Early Childhood Development

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ABSTRACT Although socioeconomic disparities in cognitive ability emerge early in the life course, most research on the consequences of living in a disadvantaged neighborhood has focused on school-age children or adolescents. In this study, we outline and test a theoretical model of neighborhood effects on cognitive development during early childhood that highlights the mediating role of exposure to neurotoxic lead. To evaluate this model, we follow 1,266 children in Chicago from birth through school entry and track both their areal risk of lead exposure and their neighborhoods' socioeconomic composition over time. With these data, we estimate the joint effects of neighborhood poverty and environmental lead contamination on receptive vocabulary ability. We find that sustained exposure to disadvantaged neighborhoods reduces vocabulary skills during early childhood and that this effect operates through a causal mechanism involving lead contamination.

KEYWORDS Poverty • Neighborhoods • Lead • Cognitive ability • Early childhood

Introduction

Socioeconomic disparities in cognitive development emerge among infants as young as 6 months (Hurt and Betancourt 2016). By the time children are 2 years old, these disparities become pronounced: at this age, children from advantaged families score significantly higher on many indicators of cognitive function (Noble et al. 2015). Later, by the start of kindergarten, disadvantaged children are even further behind their advantaged peers, and these disparities persist largely unchanged as children progress through school (von Hippel et al. 2018).

Exposure to disadvantaged neighborhoods is widely believed to shape socioeconomic disparities in cognitive skills (Jencks and Mayer 1990; Sharkey and Faber 2014). Few studies of neighborhood effects, however, focus on early childhood, even though this is the developmental period when cognitive disparities first emerge (Minh et al. 2017). Instead, prior studies have focused mainly on school-age children and adolescents because most theoretical models of neighborhood effects implicate causal

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mechanisms—such as differences in school quality (Jencks and Mayer 1990), socialization by adult role models (Wilson 1987), and collective supervision (Sampson 2012)—that are primarily relevant for older children with a more expansive sphere of social interaction. How, then, might spatially concentrated poverty affect cognitive ability during early childhood, when socioeconomic disparities first develop?

The importance of contextual influences on individual outcomes likely varies across developmental periods. In this study, we contend that differences in exposure to environmental health hazards are a central pathway through which neighborhood poverty may harm cognitive development during the first years of life. The most basic features of the neighborhoods surrounding children include the air they breathe, the water they drink, and the buildings in which they play (Sharkey and Faber 2014). Because disadvantaged neighborhoods often contain older and dilapidated housing, major roadways, and polluting industries, their residents are disproportionately exposed to harmful chemicals (Massey 2004; Muller et al. 2018). Some of these chemicals are highly neurotoxic, especially when exposure occurs during early childhood. One such chemical is lead, a neurotoxic heavy metal linked with lasting cognitive impairments among young children (Muller et al. 2018). If neighborhood poverty harms cognitive development through differences in environmental health hazards, lead may play a key explanatory role, given that children living in disadvantaged neighborhoods are at a substantially higher risk of exposure (Lanphear et al. 1998; Sampson and Winter 2016).

We evaluate this hypothesis by following a cohort of children in the Project on Human Development in Chicago Neighborhoods (PHDCN; Earls et al. 2007) from birth through school entry. At each survey wave, we match children with data on their neighborhoods' socioeconomic composition from the U.S. Census and with data on the areal risk of lead exposure from the Chicago Department of Public Health (CDPH). We then estimate the joint effects of sustained exposure to disadvantaged neighborhoods and environmental lead contamination throughout early childhood on receptive vocabulary abilities measured at the end of follow-up.

Estimating the effects of contextual exposures that vary endogenously over time poses difficult methodological challenges, including unobserved confounding and dynamic selection (Elwert and Winship 2014; Wodtke et al. 2011). We address these challenges in two ways. First, we resolve the problem of dynamic selection by using regression-with-residuals (RWR), which properly adjusts for observed time-varying confounders that may be affected by prior exposures (Wodtke 2020; Wodtke et al. 2020; Wodtke and Zhou 2020). Second, we mitigate concerns about unobserved confounding by combining RWR with a formal sensitivity analysis to construct a range of estimates adjusted for possible bias.

We find that sustained exposure to disadvantaged neighborhoods from birth through school entry reduces receptive vocabulary ability by one third of a standard deviation. Further, sustained exposure to neighborhoods with both a disadvantaged population and high levels of lead contamination is even more harmful, reducing receptive vocabulary ability by two fifths of a standard deviation. Finally, we find that the effect of neighborhood disadvantage operates through a causal mechanism involving lead.

This study makes several contributions to the literature on neighborhood poverty and child development. Theoretically, it outlines a model of neighborhood effects on

early skill formation that highlights the mediating role of environmental health hazards. Methodologically, it presents new and transferable methods for consistently estimating the effects of contextual exposures that vary endogenously over time. Empirically, it provides defensible estimates for the joint effects of exposure to disadvantaged and lead-contaminated neighborhoods on child cognitive ability. Our study therefore advances a long tradition of demographic research on how local contexts influence population health and human development (Entwisle 2007).

Place, Poverty, and Environmental Inequality

Environmental health hazards are often clustered within disadvantaged communities (Elliott and Frickel 2013; Mohai et al. 2009; Muller et al. 2018). This pattern of spatial inequality is a function of several interrelated factors, including the siting of toxic infrastructure, unequal housing investment, disparate regulatory enforcement, and residential sorting.

Governments and corporations have long confronted the dilemma of where to place necessary but noxious infrastructure (e.g., factories, highways, and landfills) by pursuing the path of least political resistance (Elliott and Frickel 2013). In many cases, these actors have selected sites in or near communities with many poor and minority residents because such communities are not well equipped to mount effective opposition. As a result, the path of least political resistance often leads to low-income, racially segregated neighborhoods.

Unequal patterns of housing investment also engender environmental inequalities. A consistent flow of capital into the local housing stock is important for reducing health hazards because older and dilapidated structures are more likely to have been constructed with harmful materials to which residents are then exposed (Mohai et al. 2009; Muller et al. 2018). Historically, redlining and covenant agreements restricted housing investments in low-income and minority neighborhoods. Today, market forces interact with more subtle prejudices to produce similar outcomes (Massey and Denton 1998; Trounstine 2018).

Abating the hazards arising from toxic infrastructure and housing divestment is crucial but costly. In an era of chronically strained government budgets and powerful business interests, disadvantaged neighborhoods suffer from weaker enforcement of environmental health protections. For example, local officials and landlords often face few consequences for failing to properly implement lead abatement policies in poor communities (Markowitz and Rosner 2013).

As some neighborhoods become sites of noxious infrastructure, dilapidated housing, and unabated toxins, families with financial means will pay to avoid them, thereby tightening the link between neighborhood composition and environmental hazards (Crowder and Downey 2010). This self-reinforcing process creates durable environmental inequalities, exposing residents of disadvantaged neighborhoods to harmful toxins throughout the life course. Next, we implicate, in particular, lead exposure during early childhood as a key mechanism accounting for neighborhood effects on cognitive development.

Disadvantaged Neighborhoods and Environmental Lead Contamination

Children are exposed to lead primarily from ingesting dust or chips from deteriorating lead paint, drinking water from outdated lead plumbing, and ingesting soil contaminated with emissions from industrial facilities or leaded gasoline (Agency for Toxic Substances and Disease Registry [ATSDR] 2019)—all of which are more common in disadvantaged communities. Consequently, blood-lead levels (BLLs) are higher for children living in poor, racially segregated neighborhoods than for children living in more advantaged areas (Lanphear et al. 1998; Muller et al. 2018; Sampson and Winter 2016).

Household contamination by lead-based paint is the primary source of lead exposure among contemporary cohorts of American children (ATSDR 2019). Lead paint was a widely used wall covering from the first half of the twentieth century until the 1970s. Over time, lead paint peels, chips, and disintegrates into dust that then settles on floors and sills, where it is ingested by children. In 1978, lead paint was banned for use in residential construction, but it is still routinely found in older homes throughout the country (Cox et al. 2011).

An important secondary source of lead exposure arises from lead plumbing (Gleason et al. 2019; Troesken 2006), which came into common use in water distribution systems during the nineteenth century. When lead plumbing corrodes, it can contaminate residents' drinking water. The use of lead in potable water systems was prohibited in 1986. Nevertheless, more than six million lead service lines remain in use across the country, and older buildings still frequently contain lead pipes or solder (Cornwell et al. 2016; Troesken 2006).

The presence of lead paint and plumbing in homes is closely linked with their age and upkeep (Cox et al. 2011; Jacobs et al. 2002). The concentration of older dwellings in low-income communities therefore engenders an association between neighborhood poverty and lead contamination. But this association persists even after housing age is accounted for because older homes in poor communities are less likely to have their lead safely contained and because disadvantaged neighborhoods are contaminated by other sources of lead (Lanphear et al. 1998; Sampson and Winter 2016).

In particular, airborne emissions are a third important source of lead contamination in poor communities (Mielke et al. 2011; Muller et al. 2018). Combustion of leaded gasoline generated a large volume of airborne lead emissions until the 1970s, when restrictions were first imposed on its use. Today, industrial facilities that process materials containing lead produce the majority of airborne emissions, although pollution from these sources has also declined over time as a result of deindustrialization (Ard 2015). Despite these reductions, airborne lead emissions remain problematic because they do not biodegrade after falling to the ground. Consequently, soil lead levels are higher in low-income neighborhoods because of their proximity to polluting industries and to roads that were highly trafficked during the era of leaded gasoline (Aelion et al. 2013; Campanella and Mielke 2008).

The spatial distribution of lead is stratified not only by income but also by race and ethnicity. Both historical and contemporary patterns of discrimination have concentrated Blacks and Latinos in older, poorer, and more isolated sections of many American cities (Massey and Denton 1998). As a result, neighborhoods with greater proportions of Black and Latino residents are more likely to contain environmental health hazards, including lead, even after economic differences between neighborhoods are accounted for (Lanphear et al. 1998; Sampson and Winter 2016).

Lead Exposure and Cognitive Development

Young children are the most susceptible to lead. They drink more water and breathe more air per unit of body weight, and they often play on floors and engage in hand-to-mouth behavior, elevating their risk of inhaling or ingesting leaded paint and soil. After lead enters the body, it is absorbed more efficiently by infants and toddlers than by older children and adults. Thus, lead contamination of disadvantaged neighborhoods places their youngest residents at the greatest risk (ATSDR 2019).

Within the central nervous system, lead inhibits the absorption and binding of calcium and zinc ions, disrupting an array of biological processes (Lidsky and Schneider 2003). High-dose exposure (e.g., BLLs > 70 μg/dL) causes acute symptomatic poisoning characterized by severe neurological injury, coma, or death. Although acute symptomatic poisoning is rare, even low-dose exposure (e.g., BLLs < 10 μg/dL) is associated with adverse neurological outcomes (ATSDR 2019).

Specifically, even at levels less than 10 μg/dL, BLLs are strongly and inversely related to measures of general intelligence among children (Canfield et al. 2003; Chiodo et al. 2004; Lanphear et al. 2000; Lanphear et al. 2005). In fact, the relationship between BLLs and measured intelligence appears to be nonlinear, with greater cognitive impairments arising with increases at lower concentrations (Canfield et al. 2003). Children with higher BLLs also perform significantly worse on school assessments of academic achievement (Aizer et al. 2018; Evens et al. 2015), and they are at greater risk of developing conduct disorders and exhibiting impulsive behavior (Goodlad et al. 2013; Sampson and Winter 2018; Winter and Sampson 2017).

In sum, disadvantaged neighborhoods are disproportionately contaminated by lead, and exposure to even low levels of the toxin harms cognitive development. Infants and toddlers are at the greatest risk. They are most likely to inhale or ingest lead in their environment, and they are the most susceptible to its neurological effects. Thus, living in a disadvantaged neighborhood is expected to impede early cognitive development by increasing exposure to lead.

Few studies have examined the effects of neighborhood poverty during early childhood, and none have evaluated whether lead contamination mediates these effects. Manduca and Sampson (2019) used ecological data to show that lead contamination is jointly correlated with neighborhood poverty and rates of intergenerational income mobility, but they did not examine outcomes among children or perform a formal mediation analysis. In the present study, we follow a birth cohort over time, tracking where they live and the amount of lead present in their residential environment to examine whether and how neighborhood poverty affects cognitive development during the early life course.

A Graphical Causal Model

Figure 1 presents a causal graph covering two generic time points. In this graph and henceforth, A_t denotes the socioeconomic composition of a child's neighborhood at time t, M_t denotes the degree to which a child's neighborhood is contaminated by lead at time t, and Y denotes cognitive ability at the end of follow-up. The graph also incorporates a set of observed baseline covariates, $\mathbf{C}$, as well as a set of observed time-varying characteristics, $\mathbf{L}_t$. Finally, it includes a set of unobserved variables, $\mathbf{U}$.

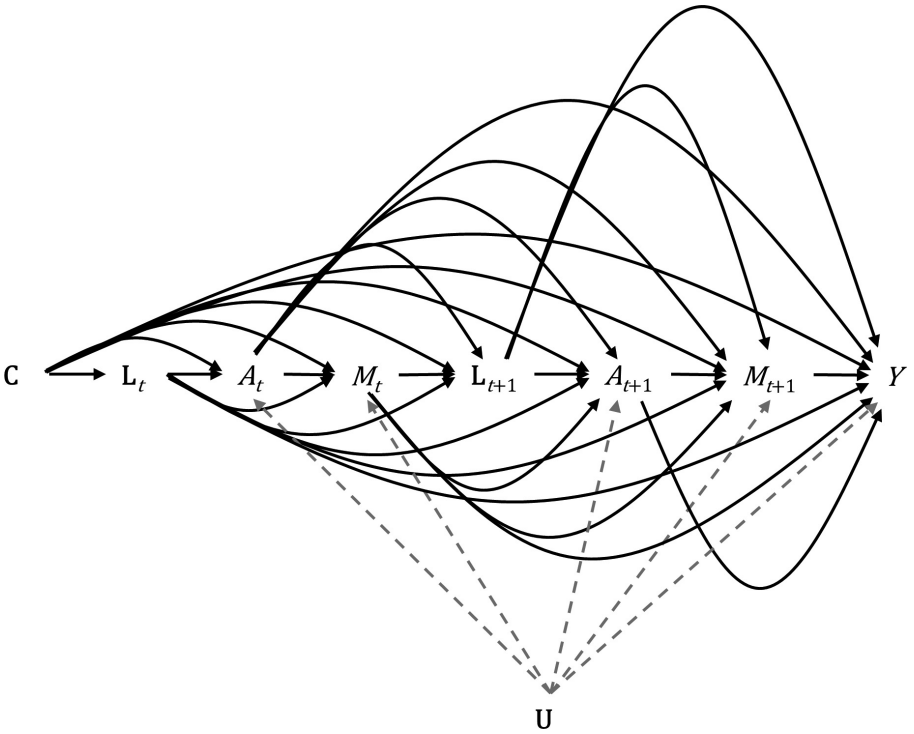

Fig. 1 Hypothesized causal relationships between baseline covariates (**C**), time-varying confounders ($\mathbf{L}_t$), neighborhood disadvantage (A_t), environmental lead contamination (M_t), cognitive ability (Y), and unobserved factors (**U**) among children

The graph shows that neighborhood poverty is hypothesized to affect end-of-study cognitive ability via multiple channels. This effect may operate directly, or it may operate indirectly via paths that emanate from A_t and traverse the level of environmental lead contamination, M_t, which is itself a direct cause of cognitive ability. Figure 1 also illustrates the challenges associated with analyzing contextual exposures that vary endogenously over time. First, unobserved confounders, **U**, affect both cognitive ability and selection into different neighborhoods. Second, observed time-varying factors, such as parental income or marital status, may confound the effects of future contextual exposures and, via the paths emanating from A_t and M_t into $\mathbf{L}_{t+1}$, are affected by prior contextual exposures. In other words, families select into different neighborhoods dynamically, with past residential choices affecting the time-varying determinants of both future residential choices and child outcomes (Wodtke et al. 2011). These challenges inform our analytic strategy.

Methods

Data

To investigate the effects of neighborhood disadvantage and environmental lead contamination, we combine data from the PHDCN, CDPH, and GeoLytics Neighborhood

Change Database. The PHDCN is a longitudinal study based on a representative sample of more than 6,000 children living in Chicago. Sampled children and their families were surveyed in 1994–1997 (baseline), 1997–1999, and 1999–2002. These surveys focused on children in seven age-groups, including a birth cohort under 1 year of age at baseline. Our analytic sample includes all 1,266 children from the birth cohort living at baseline within 80 neighborhood clusters, defined as groups of one to three census tracts.

At each wave, we match PHDCN sample members with information on their neighborhood's socioeconomic composition and degree of lead contamination. Data on the socioeconomic composition of neighborhoods come from the Neighborhood Change Database, which contains harmonized tract-level data from the 1970–2010 U.S. Censuses. Data on lead contamination come from the CDPH blood-lead surveillance database. Since 1993, the Illinois Lead Program has mandated blood-lead screening for children who live in areas deemed to be at high risk for lead poisoning, which includes the entire city of Chicago. Testing is indicated for all children at ages 12, 24, and 36 months, with additional screening until age 6 if other risk factors are present. Laboratories report all test results to the CDPH, where this information is compiled into the surveillance database.[1] The database covers more than two million tests and includes information about the date of sample collection, the result in μg/dL, and the tested child's home address. Access to these data was obtained under special contractual arrangements with the CDPH.[2]

We focus on Chicago because it is a large urban center for which high-quality data on both cognitive ability and lead exposure during early childhood are available. The city also suffers from concentrated poverty, racial segregation, and extensive lead hazards, making it an ideal case for evaluating our theoretical model.

Measures

The exposure of interest is the socioeconomic composition of a child's neighborhood. Specifically, we generate a composite measure of neighborhood disadvantage by applying principal components analysis to the following characteristics: the poverty rate, the proportion of adult residents with less than a high school education, the proportion of female-headed households, and the proportion of residents who identify as non-White. The resulting measure is continuous, with higher values representing more disadvantaged neighborhoods. To facilitate interpretation, we standardize the measure using the citywide mean and variance. Parallel analyses based on several alternative formulations of this multidimensional scale (e.g., with more detailed measures of racial composition) yield nearly identical results.

To measure environmental lead contamination, we use the CDPH blood-lead surveillance data to estimate—separately by year—the proportion of children under age 6 living in each neighborhood with a BLL ≥5 μg/dL.[3] We then smooth these

[1] Under the Illinois Lead Program, healthcare providers are required to order testing for children age 3 or younger who reside in Chicago, and parents must provide proof of lead testing for their child upon enrollment in a daycare facility or kindergarten. These requirements generate testing data with extensive and representative coverage (Evens et al. 2015).

[2] The CDPH disclaims responsibility for any analysis, interpretations, or conclusions drawn from these data.

[3] We compute these estimates from tests conducted with venous samples at labs with limits of detection <5 μg/dL. The median number of children per neighborhood per year who were tested according to these criteria is 157.

estimates over time using kernel regression and match them to PHDCN participants at each survey wave. The local prevalence of elevated BLLs provides an ecological proxy for the degree to which a neighborhood is contaminated by lead from all sources. The threshold we use to define elevated BLLs is consistent with recent monitoring guidelines from the U.S. Centers for Disease Control and Prevention (ATSDR 2019). Parallel analyses based on a threshold of ≥10 μg/dL yield similar results.

The outcome of interest is a child's cognitive ability, which we measure using scores on the Peabody Picture Vocabulary Test (PPVT; Dunn 1997). The PPVT was administered at the third wave of the PHDCN, when most members of the birth cohort were aged 4–5. The PPVT is a standardized assessment of a particular dimension of cognitive ability—namely, receptive vocabulary skills in Standard American English. We focus on vocabulary skills because language is a powerful symbolic system through which children learn or acquire most other abilities. The PPVT has desirable psychometric properties, including high validity and reliability, even among linguistically diverse samples (Campbell 1998; Dunn 1997). In our multivariate analyses, we standardize scores to have a mean of 0 and variance of 1.

To adjust for confounding, we control for several covariates measured at baseline, including gender, race and ethnicity, family size, the age and education level of a child's primary caregiver, and homeownership status. Some of these characteristics, such as family size and homeownership, often change over time, but they were recorded only at the baseline wave of the PHDCN. We also adjust for a set of time-varying covariates measured at each survey wave: the natural log of household income; parental marital status; and whether a child's primary caregiver is employed, receives public assistance, or speaks mainly English at home. The coding of these variables is outlined in Tables 1 and 2, which also provide descriptive statistics.

Estimands

We rely on potential outcomes notation to define the effects of time-varying contextual exposures (Rubin 1974). Let $\underline{\mathbf{a}} = (a_1, a_2, a_3)$ denote a sequence of exposures to different levels of neighborhood disadvantage at waves $t = 1$, 2, and 3 of the PHDCN. Next, let $Y(\underline{\mathbf{a}})$ denote a child's receptive vocabulary ability measured at the end of follow-up had the child been exposed to neighborhoods with levels of disadvantage given by $\underline{\mathbf{a}}$. The observed outcome, Y, is assumed to equal the potential outcome, $Y(\underline{\mathbf{a}})$, for the single exposure sequence that the child did in fact experience; the other potential outcomes are counterfactuals.

We first focus on estimating the average total effects of exposure to different levels of neighborhood disadvantage, which can be formally defined as follows:

$$ATE(\underline{\mathbf{a}}, \underline{\mathbf{a}}') = E(Y(\underline{\mathbf{a}}) - Y(\underline{\mathbf{a}}')). \quad (1)$$

This expression represents the expected difference in vocabulary ability if children were exposed to the sequence of neighborhood conditions defined by $\underline{\mathbf{a}}$ rather than some other sequence $\underline{\mathbf{a}}'$. With a continuous measure of neighborhood disadvantage, there are infinitely many contrasts between exposure sequences. Thus, we model the total effects using the parametric function

Table 1 Time-invariant sample characteristics measured at baseline, Project on Human Development in Chicago Neighborhoods birth cohort (n=1,266)

Variable	Mean	SD
Child Characteristics		
Female (vs. not female)	.49	—
Race/ethnicity		
White	.16	—
Black	.30	—
Hispanic	.49	—
Other	.05	—
Family Characteristics		
Family size (number of coresidents)	5.26	2.08
Homeowner (vs. otherwise)	.20	—
Primary Caregiver Characteristics		
Age (years)	27.36	6.92
Education		
Less than high school	.44	—
High school graduate	.13	—
Some college	.31	—
College graduate	.12	—

Note: The table presents combined estimates from 50 imputations.

Table 2 Time-varying sample characteristics, Project on Human Development in Chicago Neighborhoods birth cohort (n=1,266)

	Wave 1 (1994–1997)		Wave 2 (1997–1999)		Wave 3 (1999–2002)	
Variable	Mean	SD	Mean	SD	Mean	SD
Contextual Measures						
Concentrated disadvantage	–.06	.74	–.08	.78	–.13	.77
Elevated blood-lead prevalence (≥5 μg/dL)	.54	.17	.48	.18	.39	.16
Child Outcome						
Peabody Picture Vocabulary Test scores	—	—	—	—	41.91	27.33
Primary Caregiver Characteristics						
Household income (log)	9.65	1.08	9.85	1.05	10.09	0.94
Employed (vs. not employed)	.42	—	.52	—	.58	—
Receives public assistance (vs. no receipt)	.40	—	.28	—	.18	—
Married (vs. unmarried)	.51	—	.55	—	.57	—
Speaks primarily English (vs. otherwise)	.67	—	.72	—	.73	—

Note: The table presents combined estimates from 50 imputations.

$$ATE(\underline{\mathbf{a}},\underline{\mathbf{a}}') = \beta\left[\text{avg}(\underline{\mathbf{a}}) - \text{avg}(\underline{\mathbf{a}}')\right], \quad (2)$$

where $\text{avg}(\underline{\mathbf{a}}) = \sum_t a_t / 3$ denotes an average computed over time and β captures the influence of neighborhood composition from birth through the end of follow-up.

Second, we focus on estimating the average joint effects of exposure to neighborhoods with different socioeconomic composition and different levels of lead

contamination. Let $\underline{\mathbf{m}} = (m_1, m_2, m_3)$ represent a sequence of exposures to neighborhoods with different levels of lead contamination from baseline through the end of follow-up. By extension, let $Y(\underline{\mathbf{a}}, \underline{\mathbf{m}})$ denote a child's vocabulary ability had the child previously lived in a sequence of neighborhoods, possibly contrary to fact, with levels of disadvantage given by $\underline{\mathbf{a}}$ and levels of lead contamination given by $\underline{\mathbf{m}}$. With this notation, the average joint effects can be formally defined as

$$AJE(\underline{\mathbf{a}}, \underline{\mathbf{a}}', \underline{\mathbf{m}}, \underline{\mathbf{m}}') = E\big(Y(\underline{\mathbf{a}}, \underline{\mathbf{m}}) - Y(\underline{\mathbf{a}}', \underline{\mathbf{m}}')\big), \tag{3}$$

which represents the expected difference in vocabulary ability if children were exposed to the sequence of neighborhood disadvantage and lead contamination defined by $\{\underline{\mathbf{a}}, \underline{\mathbf{m}}\}$ rather than some other sequence $\{\underline{\mathbf{a}}', \underline{\mathbf{m}}'\}$. We model the average joint effects using the parametric function

$$AJE(\underline{\mathbf{a}}, \underline{\mathbf{a}}', \underline{\mathbf{m}}, \underline{\mathbf{m}}') = \gamma\big[\text{avg}(\underline{\mathbf{a}}) - \text{avg}(\underline{\mathbf{a}}')\big] + \theta\big[\text{avg}(\underline{\mathbf{m}}) - \text{avg}(\underline{\mathbf{m}}')\big], \tag{4}$$

where $\{\gamma, \theta\}$ together capture the influence of concentrated disadvantage and lead contamination. Although this model is restrictive, experimentation with more flexible specifications, including several that permit complex forms of interaction and nonlinearity, yields similar results (see parts A and B of the online appendix).

Under this model, the average joint effects can be separated into the sum of a controlled direct effect of neighborhood composition and a controlled mediator effect of lead contamination. Specifically, the controlled direct effect is given by

$$CDE\big(\underline{\mathbf{a}}, \underline{\mathbf{a}}'\big) = E\Big(Y\big(\underline{\mathbf{a}}, \underline{\mathbf{m}}\big) - Y\big(\underline{\mathbf{a}}', \underline{\mathbf{m}}\big)\Big) = \gamma\Big[\text{avg}\big(\underline{\mathbf{a}}\big) - \text{avg}\big(\underline{\mathbf{a}}'\big)\Big]. \tag{5}$$

This expression represents the expected difference in vocabulary ability if children were exposed to neighborhoods with different levels of concentrated disadvantage but the same level of lead contamination. Similarly, the controlled mediator effect is given by

$$CME\big(\underline{\mathbf{m}}, \underline{\mathbf{m}}'\big) = E\Big(Y\big(\underline{\mathbf{a}}, \underline{\mathbf{m}}\big) - Y\big(\underline{\mathbf{a}}, \underline{\mathbf{m}}'\big)\Big) = \theta\Big[\text{avg}\big(\underline{\mathbf{m}}\big) - \text{avg}\big(\underline{\mathbf{m}}'\big)\Big]. \tag{6}$$

This expression represents the expected difference in vocabulary ability if children were exposed to neighborhoods with different levels of lead contamination but the same socioeconomic composition.

Finally, we examine the difference between the average total effect and the controlled direct effect, which can be interpreted as a measure of the degree to which lead contamination mediates the effect of neighborhood disadvantage. Under the models outlined previously, this difference can be expressed as follows:

$$ATE(\underline{\mathbf{a}}, \underline{\mathbf{a}}') - CDE(\underline{\mathbf{a}}, \underline{\mathbf{a}}') = (\beta - \gamma)\big[\text{avg}(\underline{\mathbf{a}}) - \text{avg}(\underline{\mathbf{a}}')\big], \tag{7}$$

which captures an effect of neighborhood poverty operating through a mechanism that involves lead contamination.

Identification

The average total effect can be identified under the assumption of sequential ignorability (Robins et al. 2000). This assumption can be formally expressed as

$$Y(\underline{\mathbf{a}}) \perp A_t \,|\, \mathbf{C}, \underline{\mathbf{L}}_t, \underline{\mathbf{A}}_{t-1}, \underline{\mathbf{M}}_{t-1} \forall\ t, \tag{8}$$

where $\perp$ denotes statistical independence; A_t denotes a child's observed exposure to neighborhood disadvantage at time t; $\underline{\mathbf{A}}_{t-1}$ and $\underline{\mathbf{M}}_{t-1}$, respectively, denote a child's history of exposure to neighborhood disadvantage and environmental lead through time $t-1$; $\underline{\mathbf{L}}_t$ denotes a child's history of time-varying covariates through time t; and $\mathbf{C}$ denotes the vector of baseline controls. In words, this assumption states that the potential outcomes of exposure to neighborhood disadvantage, $Y(\underline{\mathbf{a}})$, must be independent of a child's observed exposure at each time point, A_t, conditional on the observed past, $\{\mathbf{C}, \underline{\mathbf{L}}_t, \underline{\mathbf{A}}_{t-1}, \underline{\mathbf{M}}_{t-1}\}$. Substantively, the assumption implies that there must not be any unobserved confounders for the effects of neighborhood composition on vocabulary ability.

The joint, controlled direct, and controlled mediator effects can be identified under the following set of two sequential ignorability assumptions (VanderWeele 2009):

$$Y\left(\underline{\mathbf{a}}, \underline{\mathbf{m}}\right) \perp A_t \,|\, \mathbf{C}, \underline{\mathbf{L}}_t, \underline{\mathbf{A}}_{t-1}, \underline{\mathbf{M}}_{t-1} \forall\ t \tag{9}$$

$$Y\left(\underline{\mathbf{a}}, \underline{\mathbf{m}}\right) \perp M_t \,|\, \mathbf{C}, \underline{\mathbf{L}}_t, \underline{\mathbf{A}}_t, \underline{\mathbf{M}}_{t-1} \forall\ t. \tag{10}$$

As before, $\perp$ denotes statistical independence, and underbars denote variable histories. This set of assumptions requires that there not be any unobserved confounders for the effects of either neighborhood disadvantage or lead contamination on vocabulary ability.

We attempt to satisfy the assumptions outlined previously by adjusting for the most powerful predictors of both neighborhood selection and child outcomes. Then, to address the likely presence of unobserved confounding, we conduct a formal sensitivity analysis that reevaluates our findings across hypothetical patterns of nonrandom selection into neighborhoods.

Estimation

Estimating the effects of time-varying exposures is methodologically challenging. Even if the ignorability assumptions outlined previously are satisfied, conventional methods of covariate adjustment remain biased when any confounders are themselves time-varying and affected by prior exposures. This pattern of dynamic selection is depicted graphically in Figure 1 via the carryover and feedback effects between the contextual exposures, $\{A_t, M_t\}$, and time-varying characteristics of families, $\mathbf{L}_t$.

Adjusting naively for time-varying confounders may lead to two forms of bias: (1) bias from overcontrolling intermediate pathways if the focal exposures affect the outcome indirectly via the time-varying confounders (Wodtke et al. 2011); and (2) bias from endogenous selection if unobserved factors affect both the time-varying confounders and the outcome (Elwert and Winship 2014). In the latter situation, adjusting naively for the time-varying confounders would *induce* a spurious association between the exposures and outcome.

To address these challenges, we estimate contextual effects using RWR (Wodtke 2020; Wodtke et al. 2020; Wodtke and Zhou 2020), which is implemented in two steps. First, the confounders at each time point are regressed on all prior variables and

then residualized. Second, to estimate the contextual effects of interest, the outcome is regressed on prior exposures and the residualized confounders from the first stage. Because residualizing the time-varying confounders with respect to the observed past purges them of their association with prior exposures, these terms can be included in an outcome regression to adjust for confounding without engendering bias due to overcontrol or endogenous selection.

Specifically, RWR estimates of the $ATE(\underline{\mathbf{a}},\underline{\mathbf{a}}')$ come from a regression that can be expressed as

$$E(Y\,|\,\mathbf{C},\underline{\mathbf{L}},\underline{\mathbf{A}},\underline{\mathbf{M}}) = \alpha_0 + \boldsymbol{\alpha}_1^T\mathbf{C}^{\perp} + \boldsymbol{\alpha}_2^T\textstyle\sum_t\mathbf{L}_t^{\perp} + \beta\ \text{avg}(\underline{\mathbf{A}}) + \alpha_3\textstyle\sum_t M_t^{\perp}, \tag{11}$$

where $\mathbf{C}^{\perp} = \mathbf{C} - E(\mathbf{C})$ denotes a vector of baseline covariates centered on their marginal means, $\mathbf{L}_t^{\perp} = \mathbf{L}_t - E(\mathbf{L}_t|\mathbf{C},\underline{\mathbf{L}}_{t-1},\underline{\mathbf{A}}_{t-1},\underline{\mathbf{M}}_{t-1})$ denotes a residual transformation of the time-varying confounders, and $M_t^{\perp} = M_t - E(M_t|\mathbf{C},\underline{\mathbf{L}}_t,\underline{\mathbf{A}}_t,\underline{\mathbf{M}}_{t-1})$ denotes a similar residual transformation of the mediator. These residual terms are estimated from a set of first-stage regressions for $E(\mathbf{L}_t|\mathbf{C},\underline{\mathbf{L}}_{t-1},\underline{\mathbf{A}}_{t-1},\underline{\mathbf{M}}_{t-1})$ and $E(M_t|\mathbf{C},\underline{\mathbf{L}}_t,\underline{\mathbf{A}}_t,\underline{\mathbf{M}}_{t-1})$. They are then substituted into the outcome regression to adjust for observed confounding of the relationship between neighborhood disadvantage and vocabulary ability. If these regressions are correctly specified and exposure to neighborhood disadvantage is sequentially ignorable, RWR estimates of $\beta[\text{avg}(\underline{\mathbf{a}}) - \text{avg}(\underline{\mathbf{a}}')]$ from Eq. (11) are consistent for the $ATE(\underline{\mathbf{a}},\underline{\mathbf{a}}')$.

Similarly, the joint effects are estimated from another regression with the form

$$E\left(Y\,|\,\mathbf{C},\underline{\mathbf{L}},\underline{\mathbf{A}},\underline{\mathbf{M}}\right) = \eta_0 + \boldsymbol{\eta}_1^T\mathbf{C}^{\perp} + \boldsymbol{\eta}_2^T\textstyle\sum_t\mathbf{L}_t^{\perp} + \gamma\ \text{avg}\left(\underline{\mathbf{A}}\right) + \theta\ \text{avg}\left(\underline{\mathbf{M}}\right), \tag{12}$$

where $\mathbf{C}^{\perp}$ and $\mathbf{L}_t^{\perp}$ are residual terms defined as earlier. This regression differs from that outlined previously only in that it includes $\text{avg}(\underline{\mathbf{M}})$ as a predictor, which is computed from untransformed rather than residualized values of the mediator, to estimate the joint effects of interest. Under the assumptions of correct model specification and sequential ignorability for both neighborhood disadvantage and lead contamination, RWR estimates of $\gamma[\text{avg}(\underline{\mathbf{a}}) - \text{avg}(\underline{\mathbf{a}}')]$ and $\theta[\text{avg}(\underline{\mathbf{m}}) - \text{avg}(\underline{\mathbf{m}}')]$ from Eq. (12) are consistent for the $CDE(\underline{\mathbf{a}},\underline{\mathbf{a}}')$ and $CME(\underline{\mathbf{m}},\underline{\mathbf{m}}')$, respectively. By extension, their sum is consistent for the $AJE(\underline{\mathbf{a}},\underline{\mathbf{a}}',\underline{\mathbf{m}},\underline{\mathbf{m}}')$.

The simplicity of RWR is premised on several strong modeling constraints. For example, the regressions outlined previously constrain the contextual effects of interest to be invariant across the covariates. In a set of ancillary analyses, we relax these constraints by including two-way interactions between elements of $\mathbf{C}^{\perp}$ and $\mathbf{L}_t^{\perp}$, on the one hand, with measures of neighborhood disadvantage and environmental lead contamination, on the other. Including these interactions allows for effect moderation by characteristics of children and their families—for example, by gender, race, and household income. Because we construct interaction terms using the residualized covariates, the total and joint effects of interest can still be computed as outlined previously (Wodtke et al. 2020).

We also estimate these effects using the method of residual balancing (Zhou and Wodtke 2020). As with RWR, residual balancing is implemented by first regressing the confounders at each time point on all prior variables and then computing residuals. Next, a set of weights is constructed to satisfy the following two conditions: (1) the residualized confounders are orthogonal to future exposures, past

exposures, and past confounders in the weighted sample; and (2) the entropy of the weights is minimized. Weighted regressions similar to those outlined previously but omitting the residualized covariates can then be used to estimate the effects of neighborhood disadvantage and lead contamination. In this way, residual balancing adjusts for dynamic selection while obviating the need for restrictive modeling assumptions.

We compute standard errors for all effect estimates using the stratified cluster bootstrap to adjust for the PHDCN's complex sample design (Rao and Wu 1988).[4] We then repeat this analysis across 50 complete data sets with missing values for all variables simulated via multiple imputation, and we combine estimates following Rubin (1987). Overall, the proportion of missing information in this analysis is approximately 17% and primarily reflects panel attrition.[5]

Sensitivity Analysis

Unobserved confounding is a ubiquitous threat to causal inference in observational studies and may lead to bias in estimates of contextual effects. To account for this possibility, we implement a formal sensitivity analysis. With this approach, confounding bias is modeled using a selection function that captures hypothetical departures from the ignorability assumptions outlined previously (Brumback et al. 2004). Consider, for example, an analysis of a point-in-time neighborhood exposure, a. In this setting, bias in estimates of the total effect would occur if

$$E\left(Y(a)|A=a,\mathbf{C}\right) \neq E\left(Y(a)|A=a',\mathbf{C}\right), \tag{13}$$

that is, if the observed mean outcome is not exchangeable with the counterfactual mean outcome. Its form and magnitude can be modeled with the selection function

$$s(a,a') = E\left(Y(a)|A=a,\mathbf{C}\right) - E\left(Y(a)|A=a',\mathbf{C}\right) = (a-a')\tau, \tag{14}$$

where τ is an unknown sensitivity parameter that determines the sign and magnitude of bias in estimates of the total effect. If $\tau > 0$, these estimates are biased upward because children in more disadvantaged neighborhoods differ from those in less disadvantaged neighborhoods on unobserved factors that improve their vocabulary skills. If $\tau < 0$, estimates are biased downward because children in more disadvantaged neighborhoods differ from those in less disadvantaged neighborhoods on unobserved factors that suppress their vocabulary skills.

[4] This method is implemented by selecting with replacement $j_k - 1$ neighborhood clusters from within each sampling stratum, where j_k denotes the number of clusters in stratum k.

[5] Replication code is available at https://github.com/gtwodtke/nhood_mediation_lead. The data on which it is based are restricted-access and can only be obtained under contractual arrangements that preclude us from disseminating them. Researchers interested in obtaining these data can follow the procedures outlined at https://www.icpsr.umich.edu/web/pages/ICPSR/access/restricted/index.html for the PHDCN and at https://www.chicago.gov/city/en/depts/cdph/provdrs/health_data_and_reports.html for the BLL surveillance data. The Neighborhood Change Database can be licensed from GeoLytics, Inc. (https://geolytics.com), and it is also available through some academic library systems.

A bias-corrected estimate for the total effect can be obtained by (1) computing a bias term equal to $B(\tau) = \sum_{a'}(A - a')P(A = a')\tau$; (2) using it to construct an adjusted outcome equal to $Y^{B(\tau)} = Y - B(\tau)$; and (3) substituting the adjusted outcome into the regression used to estimate the effect of interest. The degree to which inferences about this effect are sensitive to unobserved confounding can be assessed by evaluating the bias-corrected estimates across a range of plausible values for τ.

Similarly, nonrandom selection into two point-in-time exposures, a and m, can be modeled as

$$\begin{aligned} s(a,a',m,m') &= E\Big(Y(a,m)|A=a,M=m,\mathbf{C}\Big) - E\Big(Y(a,m)|A=a',M=m',\mathbf{C}\Big) \\ &= (a-a')\phi + (m-m')\psi, \end{aligned} \tag{15}$$

where bias in point-in-time variants of the controlled direct, controlled mediator, and average joint effects is governed by ϕ, ψ, and $(\phi + \psi)$, respectively. Bias-corrected estimates of these effects can be computed by constructing a bias term, $B(\phi,\psi) = \sum_{a'}(A - a')P(A = a')\phi + \sum_{m'}(M - m')P(M = m')\psi$, and by replicating the analysis on an adjusted outcome, $Y^{B(\phi,\psi)} = Y - B(\phi,\psi)$.

We generalize this approach for the present study, in which the exposures of interest are time-varying. In this setting, we use separate selection functions to model unobserved confounding at each time point, and we modify the bias terms so that they reflect accumulated selection from baseline through the end of follow-up. Specifically, the bias terms are given by $B(\tau) = \sum_t \sum_{a_t'}(A_t - a_t')P(A_t = a_t')\tau$ and $B\big(\phi,\psi\big) = \sum_t \sum_{a_t'}(A_t - a_t')P\big(A_t = a_t'\big)\phi + \sum_t \sum_{m_t'}\big(M_t - m_t'\big)P\big(M_t = m_t'\big)\psi$. We compute adjusted outcomes as outlined previously and use them to refit Eqs. (11) and (12), from which we obtain bias-corrected estimates for the effects of interest. To facilitate interpretation, we calibrate the sensitivity parameters such that a one-unit change corresponds to the amount of bias eliminated from our focal effect estimates by virtue of adjusting for parental education. The results, then, capture sensitivity to multiples of observed confounding from nonrandom selection on this covariate.

Results

Figures 2 and 3 display the distribution of concentrated disadvantage and elevated BLLs across Chicago census tracts in 1997, when the PHDCN was concluding its first wave of data collection. Several patterns are evident. First, in an alarming number of Chicago neighborhoods, a majority of resident children have elevated BLLs. Second, disadvantaged and lead-contaminated neighborhoods are spatially concentrated on Chicago's South and West Sides, which are predominantly Black and suffer from high rates of poverty. Finally, even though neighborhood composition and lead contamination are tightly coupled in Chicago, disadvantaged neighborhoods with lower levels of lead contamination and advantaged neighborhoods with higher levels of lead contamination both exist in nontrivial numbers.

The first row of Table 3 presents estimates for the total effect of sustained exposure to disadvantaged neighborhoods on receptive vocabulary ability measured around the time of school entry. Specifically, these estimates contrast scores on the PPVT under continuous residence in a neighborhood that is 0.7 standard deviations above the

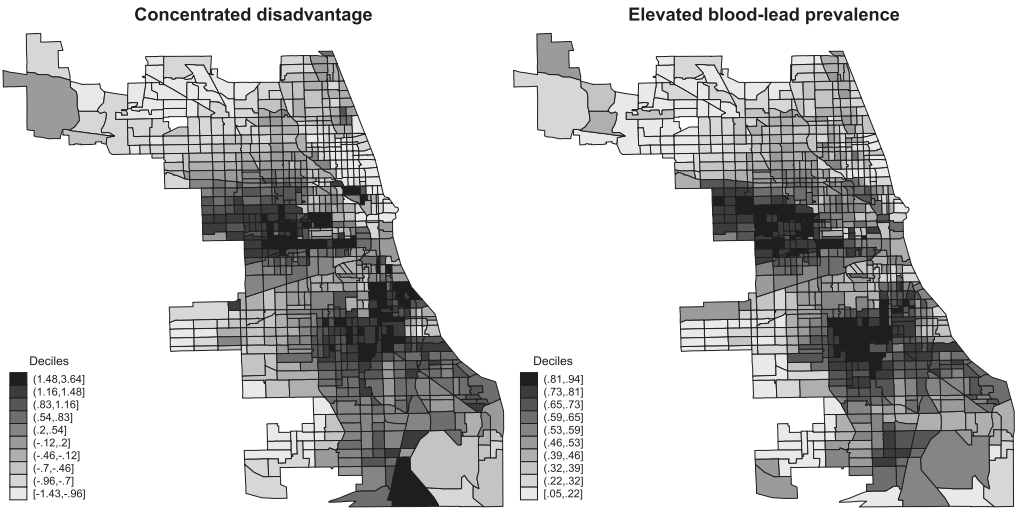

Fig. 2 Spatial distribution of concentrated disadvantage and elevated blood-lead levels in Chicago, 1997. In accordance with CDPH policy, estimates for census tracts with fewer than 10 tested children are suppressed from this figure. For visual continuity, we impute these suppressed estimates from nearby tracts with a sufficiently large number of observations.

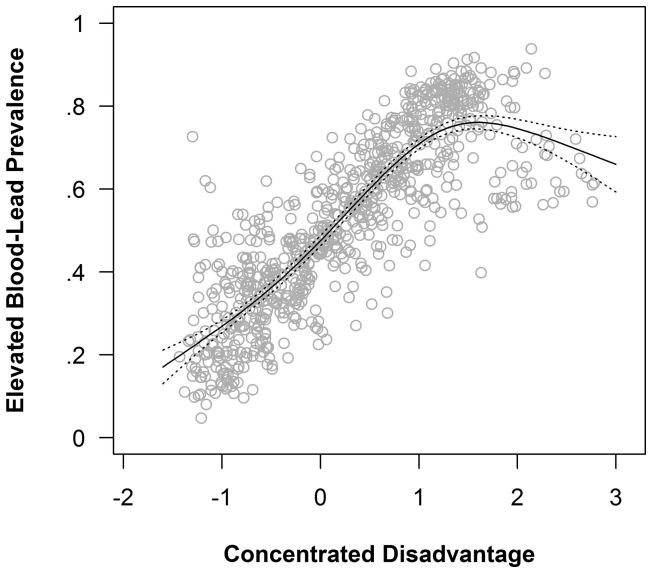

Fig. 3 Bivariate relationship between concentrated disadvantage and environmental lead contamination across Chicago census tracts, 1997. In accordance with CDPH policy, tracts with fewer than 10 tested children in 1997 are suppressed from this figure. The solid line represents fitted values from a thin plate spline, while the dashed lines represent upper and lower limits of a 95% confidence interval.

citywide mean on our index of concentrated disadvantage (the 75th percentile) rather than a neighborhood that is 0.9 standard deviations below the mean (the 25th percentile). All estimates for the total effect are substantively large and statistically significant at stringent thresholds. They indicate that living in a disadvantaged neighborhood

Table 3 Estimated effects of cumulative exposure to neighborhood disadvantage and environmental lead contamination during early childhood on end-of-study receptive vocabulary ability, Project on Human Development in Chicago Neighborhoods birth cohort (n=1,266)

	Linear and Additive RWR		RWR + Gender Moderation		RWR + Race Moderation		RWR + Income Moderation		Residual Balancing	
Estimand	Est.	p Value	Est.	p Value	Est.	p Value	Est.	p Value	Est.	p Value
Average Total Effect (ATE)	−0.358	<.001	−0.358	<.001	−0.356	<.001	−0.352	.001	−0.345	.002
	(0.106)		(0.106)		(0.107)		(0.107)		(0.109)	
Average Joint Effect (AJE)	−0.412	<.001	−0.417	<.001	−0.412	<.001	−0.408	<.001	−0.401	<.001
	(0.103)		(0.103)		(0.104)		(0.104)		(0.109)	
Controlled Direct Effect (CDE)	−0.027	.866	−0.037	.818	−0.025	.879	−0.039	.809	−0.056	.741
	(0.160)		(0.161)		(0.161)		(0.162)		(0.169)	
Controlled Mediator Effect (CME)	−0.385	.004	−0.380	.005	−0.387	.005	−0.369	.007	−0.345	.017
	(0.135)		(0.136)		(0.137)		(0.138)		(0.144)	
Test for Mediation (ATE – CDE)	−0.331	.007	−0.321	.010	−0.331	.007	−0.312	.012	−0.289	.040
	(0.122)		(0.124)		(0.123)		(0.125)		(0.141)	

Notes: The table presents combined estimates from 50 imputations. Standard errors, shown in parentheses, are computed using the stratified cluster bootstrap with 500 replications; p values are from z tests of the null hypothesis that the focal estimand is equal to 0. RWR = regression-with-residuals.

throughout early childhood reduces receptive vocabulary ability by about one third of a standard deviation. This effect size is comparable to those reported previously in observational and quasi-experimental studies of neighborhood effects in Chicago (Burdick-Will et al. 2011) and is consistent with experimental estimates from the Chicago study site of the Moving to Opportunity demonstration program (Orr et al. 2003). However, reported estimates of neighborhood effects are heterogeneous, and ours exceed those documented at the Moving to Opportunity study sites in New York, Boston, and Los Angeles.

The middle rows of Table 3 present estimates for the controlled direct effect, the controlled mediator effect, and then their sum—that is, the average joint effect. As with the total effect, we report estimates for a controlled direct effect that compares neighborhoods 0.7 standard deviations above with those 0.9 standard deviations below the mean on our index of concentrated disadvantage. For the controlled mediator effect, we report estimates that compare neighborhoods with an elevated blood-lead prevalence of 65% rather than 30%, which correspond approximately with the upper and lower quartiles of the citywide distribution.

Estimates of the joint effect from across RWR specifications and from models fit with residual balancing weights are all similar in magnitude and statistically significant at stringent thresholds. They indicate that sustained exposure to both disadvantaged and lead-contaminated neighborhoods during early childhood reduces receptive vocabulary ability by about two fifths of a standard deviation. Moreover, estimates for the controlled direct and mediator effects suggest that the deleterious impact of neighborhood disadvantage is driven by disparate exposures to lead. Specifically, estimates for the controlled direct effect are close to 0 and fail to approach conventional thresholds for statistical significance. A failure to reject the null hypothesis of no direct effect implies that we cannot rule out the possibility that the total effect of neighborhood disadvantage may operate exclusively through lead contamination. Estimates of the controlled direct effect, however, are imprecise, which is due in part to collinearity among the exposure and mediator. Given the imprecision of these estimates, our results are also consistent with only partial mediation.

Estimates for the controlled mediator effect, by contrast, are substantively large and statistically significant. They indicate that sustained exposure to neighborhoods with higher versus lower levels of lead contamination would reduce vocabulary ability by about two fifths of a standard deviation even if all children were exposed to the same level of concentrated disadvantage. By way of reference, prior research suggests that an increase in blood-lead concentration from 1 to 10 μg/dL is linked with declines in cognitive ability ranging from one third to one half of a standard deviation among young children (Canfield et al. 2003; Lanphear et al. 2005).

The bottom row of Table 3 evaluates whether the total effect differs from the controlled direct effect, formally testing whether lead contamination explains the link between neighborhood disadvantage and vocabulary ability. Estimates of the difference are substantively large, and p values from tests of the null hypothesis that these effects are equal provide considerable evidence against this possibility. Thus, our results indicate that neighborhood effects during early childhood are at least partly explained by a causal mechanism involving lead contamination.

Figure 4 displays bias-corrected estimates from the sensitivity analysis. The upper left panel summarizes the sensitivity of estimates for the average total effect to unobserved confounding. Specifically, it displays how estimates for the total effect would

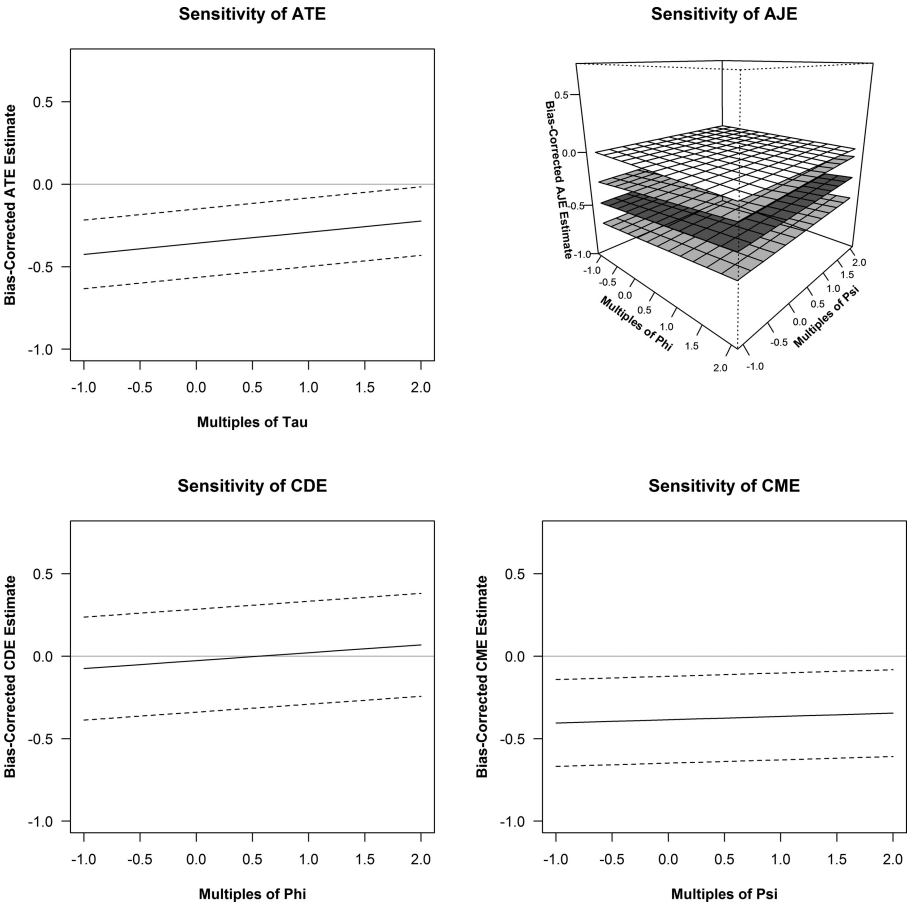

Fig. 4 Bias-corrected effect estimates, Project on Human Development in Chicago Neighborhoods birth cohort ($n = 1{,}266$). In the two-dimensional plots, dashed lines represent 95% confidence intervals based on the stratified cluster bootstrap with 500 replications. In the three-dimensional plot, the dark gray plane represents the bias-adjusted point estimates, the light gray planes represent 95% confidence intervals, and the white plane provides a reference at 0. Tau, phi, and psi are scaled to equal the bias eliminated from our focal effect estimates by virtue of adjusting for parental education. ATE = average total effect. AJE = average joint effect. CDE = controlled direct effect. CME = controlled mediator effect.

change if there were unobserved selection into neighborhoods with different levels of concentrated disadvantage. When the sensitivity parameter in this plot is equal to 0, the estimate is the same as that from our baseline RWR specification. Larger values of the sensitivity parameter—in absolute terms—represent a greater degree of unobserved selection and thus a more pronounced bias correction.

The upper right panel of Figure 4 summarizes the sensitivity of estimates for the average joint effect to unobserved confounding. Specifically, it displays how estimates for the joint effect would change if there were unobserved selection into neighborhoods of different socioeconomic composition and with different levels of lead contamination. When both sensitivity parameters in this plot are equal to 0, the

estimate is the same as that from our baseline RWR specification. Larger multiples of ϕ represent a greater degree of unobserved selection into neighborhoods with different levels of concentrated disadvantage, whereas larger multiples of ψ represent a greater degree of unobserved selection into neighborhoods with different levels of lead contamination.

The lower panels of Figure 4 summarize the sensitivity of the controlled direct and mediator effects to each of these two forms of unobserved selection, respectively. The lower left panel displays how estimates for the controlled direct effect would change if there were unobserved selection into neighborhoods of different socioeconomic composition, and the lower right panel shows how estimates for the controlled mediator effect would change if there were unobserved selection into neighborhoods with different levels of lead contamination.

Across all four plots, our inferences appear robust to different forms of unobserved selection. Even when $\tau = \phi = \psi = 2$—that is, when the magnitude of bias due to unobserved selection is assumed to be twice as strong as the bias that would arise by virtue of omitting controls for parental education—adjusted estimates for the total, joint, and controlled mediator effects are negative, substantively large, and statistically significant, whereas estimates for the controlled direct effect remain small and insignificant. Given that parental education is among the most powerful determinants of neighborhood selection and child development, this level of confounding by unobserved factors is extreme and seems unlikely, although we cannot rule it out empirically.

As an additional assessment of possible selection bias, we also perform a falsification test by evaluating whether neighborhood effects on cognitive ability can be explained by lead contamination among older children in the PHDCN who were aged 6–12 at baseline. Children older than 6 years are less sensitive to lead contamination than infants and toddlers. If our focal mediator were truly unconfounded, we would expect any evidence of mediation among these older children to be less pronounced than for the birth cohort because of older children's comparatively lower sensitivity to lead hazards. Results from this analysis, presented in part C of the online appendix, provide little evidence of mediation via lead contamination among older children, thereby disconfirming the falsification test and further bolstering confidence in our causal inferences targeting early childhood.

Discussion

The effects of neighborhood poverty have been extensively studied among older children. However, comparatively little research has explored contextual effects during early childhood, and the mechanisms hypothesized to explain them remain shrouded in a "black box" (Sampson 2012). In this study, we investigate whether living in a disadvantaged neighborhood from birth through school entry affects vocabulary skills, focusing on the mediating role of exposure to neurotoxic lead. Using novel counterfactual methods and longitudinal data, we find that growing up in a disadvantaged neighborhood substantially reduces vocabulary ability during early childhood and that this effect operates through a causal mechanism involving lead contamination.

Our findings suggest that the genesis of cognitive disparities can be traced partly to neighborhoods and their environmental health risks. Studies have indicated that socioeconomic differences take root when children are very young and have variously implicated parental behavior, family resources, or genetics in generating these early gaps (Hurt and Betancourt 2016; Nisbett 2011). Results from the PHDCN reveal that higher order patterns of spatial stratification are also important determinants of cognitive development. Beyond characteristics of families and individuals, children's neighborhoods shape their exposure to environmental toxins, such as lead, which in turn affect their cognition.

This study also has important implications for ecological social theory, accounts of which typically focus on intermediate mechanisms that are most relevant for older children and adolescents, such as school quality, access to adult role models, and collective supervision (Jencks and Mayer 1990; Sampson 2012; Wilson 1987). Each of these mechanisms may transmit neighborhood effects on certain outcomes and at certain times, but their influence during the earliest phases of development is circumscribed by natural limitations on the social interactions of infants and toddlers. This study suggests that in the search for mechanisms connecting neighborhood poverty to child outcomes, attention to the developmental specificity of different putative mediators will be important. During early childhood, we find support for a theoretical model that views neighborhood effects as first arising from disparities in exposure to environmental health hazards, although factors ranging from limited childcare options to violent crime may also play a mediating role at this developmental stage. During more advanced developmental periods, schools, peers, and role models likely become more important.

Research on neighborhood effects is frequently criticized for having limited capacity to inform policy either because of concerns about the credibility of causal inferences or because this body of work reveals little about intermediate mechanisms that might serve as points of intervention (Sampson 2012). Short of conducting a sequentially randomized field experiment, our analysis provides some of the more credible evidence that neighborhood disadvantage causally affects cognitive development. Further, we identify a cogent mechanism that explains these effects. Thus, our study has implications for policy. It suggests that programs to abate lead paint in homes, replace lead plumbing, and remove lead-contaminated soil not only will improve child outcomes overall but may also mitigate the consequences of spatially concentrated poverty.

Finally, this study contributes to methods for research on contextual effects by introducing new procedures for analyzing time-varying exposures. RWR and residual balancing avoid the problems that afflict conventional methods in the presence of dynamic selection. Additionally, compared with other methods designed to sidestep these concerns, such as inverse probability weighting, RWR and its variants are more robust and efficient (Wodtke 2020; Wodtke et al. 2020). We therefore expect these methods to find wide application in the social sciences.

Although this study makes important contributions to theory, policy, and methods, it is not without limitations. First, despite our efforts to mitigate unobserved confounding, it remains possible that we failed to control for important covariates or that any lingering bias is stronger than assumed in the sensitivity analysis, in which case our causal inferences would be mistaken. Second, we focus on only one dimension of cognitive development—receptive vocabulary skills—but many other abilities may also be sensitive to neighborhood conditions

during early childhood. Third, we analyze cumulative effects, but information on sensitive exposure periods would be valuable. In part D of the online appendix, we report point-in-time effects to explore whether exposures at certain periods (e.g., infancy vs. preschool) matter more than others. Unfortunately, we lack the data needed to precisely estimate these effects and cannot draw firm conclusions about differential sensitivity across early childhood. Fourth, we analyze only the areal risk of lead exposure because we cannot match children in the PHDCN with their individual BLLs in the CDPH surveillance database. Finally, we rely on data from a cohort of children born in Chicago nearly three decades ago, and the degree to which lead exposure explains neighborhood effects may differ across time and place.

Indeed, BLLs among children in Chicago have declined since the launch of the PHDCN. In the online appendix, Figure E.1 shows that elevated blood-lead prevalence rates declined steadily over time in Chicago. By 2010, few Chicago neighborhoods had prevalence rates over 30%, even though much higher rates were typical only a decade earlier. The steep decline in lead exposure partly reflects the success of surveillance, regulation, and abatement efforts expanded as part of the Illinois Lead Program and other such initiatives administered by the U.S. Department of Housing and Urban Development (Billings and Schnepel 2018; Sorensen et al. 2019). It follows that lead contamination may no longer be as powerful a mediator as indicated by our study and that neighborhood effects on early cognitive development may have attenuated over time.

The explanatory role of lead exposure may also be particular to certain cities or regions. Urban areas in the Midwest and Northeast tend to suffer the highest levels of lead contamination because of their widespread use of lead plumbing, their metal processing industry, and their aging housing stock (Jacobs et al. 2002; Pell and Schneyer 2016). Thus, what makes Chicago an ideal case for evaluating our theoretical model might also make it a special case. Consistent with this cautionary perspective on generalizability, studies focused on Chicago often yield some of the largest estimates of neighborhood effects (Burdick-Will et al. 2011).

Nevertheless, contemporary cohorts of children in disadvantaged neighborhoods remain at greater risk of lead exposure in many American cities, and even at the lower doses that are now more common, lead can harm their developing brains. Moreover, lead is just one of many neurotoxins concentrated in poor communities. Arsenic, mercury, manganese, and other chemicals that are known or suspected to interfere with the central nervous system are also more pervasive in disadvantaged, minority neighborhoods (Hamblin 2014; Israel 2012). As social scientists begin to illuminate the black box of neighborhood effects, they should therefore prioritize research on the role of environmental health hazards. Its limitations notwithstanding, this study provides considerable evidence that growing up in a disadvantaged neighborhood inhibits cognitive development because these environments are literally toxic for children. ■

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