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

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Abstract

Although socioeconomic disparities in cognitive ability emerge early in the life course, most research on the developmental consequences of living in a disadvantaged neighborhood focuses 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 environmental health hazards, and in particular, exposure to neurotoxic lead. To evaluate this model, we follow a cohort of 1,266 children in the Project on Human Development in Chicago Neighborhoods from birth through the time of school entry, matching them at each survey wave with information on neighborhood composition and the areal risk of lead exposure. With these data, we then estimate the joint effects of neighborhood poverty and environmental lead contamination on receptive vocabulary ability. We find that sustained exposure to disadvantaged neighborhoods substantially reduces vocabulary ability during early childhood and that nearly all of this effect may operate through a causal mechanism involving lead contamination. These findings are robust to unobserved confounding and to the use of several alternative estimation strategies, which suggests that living in a disadvantaged neighborhood impedes early childhood development because it increases exposure to environmental toxins like lead.

Keywords: neighborhoods, poverty, lead, child development, environmental health, mediation

1. Introduction

Socioeconomic disparities in cognitive development emerge among infants as young as six months (Hurt and Betancourt 2016; Tomalski et al. 2013). By the time children are two years old, these disparities become pronounced. At this age, children from more advantaged families can produce nearly three times as many words as children from disadvantaged families (Fernald, Marchman, and Weisleder 2013), and they score significantly higher on various other indicators of mental function (Noble et al. 2015). Later, by the start of kindergarten, many disadvantaged children have fallen even further behind their peers from more advantaged backgrounds, and the large disparities in academic skills present at the time of school entry persist largely unchanged as children progress through their primary education (Garcia 2015; von Hippel, Workman, and Downey 2018).

Exposure to disadvantaged neighborhoods is widely thought to be an important driver of socioeconomic disparities in cognitive skills (Jencks and Mayer 1990; Leventhal and Brooks-Gunn 2000; 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 and then become entrenched. For example, according to a recent review of the literature on neighborhood context and child development, only 42 out of 266 relevant studies included children under age six, and only seven included children under age three (Minh et al. 2017). Of these, fewer still examined effects on cognitive abilities.

Instead, studies of neighborhood effects have focused mainly on school-age children and adolescents. This is because, at least in part, the most prominent theoretical models of neighborhood effects highlight causal mechanisms, such as differences in school quality (Jencks and Mayer 1990; Wodtke and Parbst 2017), socialization by peers and extrafamilial adults

(Harding 2010; Wilson 1987), and collective supervision (Sampson 2001, 2012), that are delimited by age and primarily relevant for older children who traverse a more expansive sphere of social interaction. How, then, might spatially concentrated poverty affect cognitive ability during early childhood, when socioeconomic disparities first take root?

In this study, we contend that differences in exposure to environmental health hazards are a central pathway through which neighborhood poverty may harm early cognitive development. The most basic features of the neighborhood surrounding children include the air they breathe, the water they drink, and the buildings in which they play (Sharkey and Faber 2014). Because of the older and poorer physical condition of housing in disadvantaged neighborhoods together with their proximity to major roadways and industry, residents of these communities are disproportionately exposed to harmful pollutants and other household contaminants (Massey 2004; Muller, Sampson, and Winter 2018). Many of the chemicals present in disadvantaged neighborhoods are highly neurotoxic and associated with a variety of cognitive impairments, especially when exposure occurs during early childhood (Grandjean and Landrigan 2006; Shonkoff 2003).

One such chemical is lead, a neurotoxic heavy metal that is a major risk factor for lasting cognitive impairments among exposed children (Bellinger et al. 1991; Lanphear et al. 2005; Muller et al. 2018). In the U.S., childhood exposure to lead comes primarily from three routes of transmission: by ingesting soil contaminated with emissions from metal processing facilities or leaded gasoline, by drinking from outdated lead plumbing installed prior to the ban on its use in potable water systems, and by ingesting dust or chips from lead paint applied before its use was banned in interior construction (Agency for Toxic Substances and Disease Registry 2019). All of these sources are more common in disadvantaged communities, and as a result, blood-lead

levels for children living in poor, racially segregated neighborhoods are substantially higher than for children living in more advantaged areas (Lanphear et al. 1998; Muller et al. 2018; Sampson and Winter 2016). If neighborhood poverty harms early cognitive development through differences in exposure to environmental health hazards, neurotoxic lead may therefore play an especially important explanatory role.

To evaluate this hypothesis, we follow a cohort of children in the Project on Human Development in Chicago Neighborhoods (PHDCN; Earls et al. 2007) from birth through roughly the time of school entry. At every wave of the study, we match sampled children with data on the socioeconomic composition of their neighborhoods from the U.S. Census and with data on the areal risk of lead exposure from the Chicago Department of Public Health (CDPH). With these matched data sources, we then estimate the joint effects of sustained exposure to disadvantaged neighborhoods and environmental lead contamination throughout infancy and 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. First, consistently estimating these effects requires the strong assumption that there are not any unobserved factors that affect both child outcomes and selection into different neighborhoods. Because it is difficult to identify and measure all such factors, bias due to unobserved confounding remains a ubiquitous threat to causal inference in observational studies of neighborhood effects. Second, even if all factors that jointly affect neighborhood selection and child outcomes were observed, conventional methods of covariate adjustment, such as propensity score matching or regression control, remain biased when any of these characteristics vary over time and are themselves affected by prior neighborhood exposures (Elwert and Winship 2014; Robins 1986). This pattern of dynamic selection, where past

exposures affect time-varying confounders of future exposures, is endemic to contextual effects research and necessitates alternative methods of covariate adjustment (Wodtke, Harding, and Elwert 2011).

We attempt to overcome these challenges in two ways. First, we resolve the problem of dynamic selection on observed covariates by using a novel estimation procedure termed regression-with-residuals (RWR; Wodtke, Alaca, and Zhou 2020; Wodtke and Almirall 2017; Wodtke and Zhou 2020). RWR and its variants are capable of adjusting for time-varying confounders affected by prior neighborhood exposures without incurring the biases that afflict conventional approaches to covariate adjustment in the longitudinal setting. Second, we mitigate concerns about selection on unobserved covariates by combining RWR with a formal sensitivity analysis to construct a range of effect estimates adjusted for bias arising from different patterns of unobserved confounding.

Results from our analysis yield multiple important insights about contextual effects on child development. First, we find that sustained exposure to disadvantaged neighborhoods from birth through the time of school entry reduces receptive vocabulary ability by about one-third of a standard deviation. This suggests that neighborhood poverty has a substantively large and deleterious overall effect on cognitive development during early childhood. Second, we find that the joint effect of neighborhood poverty and environmental lead contamination is even more harmful. Specifically, sustained exposure to a neighborhood with not only a disadvantaged population but also a high level of lead contamination is estimated to reduce receptive vocabulary ability by about two-fifths of a standard deviation. Finally, we find that nearly all of the total effect of neighborhood disadvantage on receptive vocabulary ability may operate

through a causal mechanism involving lead contamination, although due to sampling uncertainty, our results are also consistent with only partial mediation.

This study makes several contributions to theory, empirics, and methods in the literature on neighborhood poverty and child development. Theoretically, we outline a model of neighborhood effects on cognitive abilities during early childhood that highlights the mediating role of environmental health hazards, and in particular, exposure to neurotoxic lead. Empirically, we provide defensible estimates for the joint effects of sustained exposure to disadvantaged and lead-contaminated neighborhoods on vocabulary skills assessed around the time of school entry. Methodologically, we introduce new and transferable methods for estimating the joint effects of contextual exposures that vary endogenously over time.

In the sections that follow, we begin by outlining a general theoretical framework that links environmental health hazards to disadvantaged neighborhoods. We then narrow our focus to one hazard that is especially harmful for young children by tracing the connection between exposure to lead, concentrated poverty and racial segregation, and impaired cognitive development. Next, we describe the dynamic selection process that complicates analyses of contextual effects in the longitudinal setting and present new methods to resolve these problems. Finally, we estimate the joint effects of neighborhood disadvantage and environmental lead contamination and then conclude by discussing their implications for theory, policy, and future research.

2. Place, Poverty, and Environmental Inequality: A General Framework

Environmental health hazards are often spatially clustered within disadvantaged communities (Elliott and Frickel 2013; Mohai, Pellow, and Roberts 2009; Muller et al. 2018). This pattern of

environmental inequality is a function of several interrelated factors, including the siting of toxic infrastructure, unequal housing investment, disparate regulatory enforcement, and residential sorting. In this section, we outline a set of higher-order processes that engender a link between neighborhood disadvantage and exposure to toxic substances.

For over a century, governments and corporations have confronted the dilemma of where to place necessary but noxious infrastructure, such as factories, highways, and landfills, by pursuing the “path of least political resistance” (Elliott and Frickel 2013:525). This has often involved selecting sites in or near communities with many poor and minority residents because they are not as well equipped as their more affluent counterparts to mount effective political opposition. Compared with members of more advantaged neighborhoods, those living in disadvantaged areas are less likely to have the time and resources necessary for organizing efforts, like petitioning neighbors or filing lawsuits, that might deter the placement of hazardous infrastructure. As a result, the “path of least political resistance” often leads to poor and racially segregated neighborhoods, exposing their residents to a variety of different pollutants.

Unequal patterns of housing investment also engender environmental inequalities. An ongoing flow of capital into the local housing stock is crucial for reducing exposures to environmental hazards, as older, dilapidated, or abandoned structures are more likely to expose residents to harmful materials used in their construction (Mohai et al. 2009; Muller et al. 2018). Historically, the practice of redlining and the use of covenant agreements restricted housing investment in low-income, Black, and immigrant neighborhoods by suppressing the amount of available mortgage financing and by limiting the pool of prospective buyers. Today, market forces interact with more subtle prejudices among buyers, realtors, and lenders to produce similar outcomes (Massey and Denton 1998; Trounstein 2018). Where renewed investment does

occur in low-income neighborhoods, it disproportionately comes in the form of speculative ownership, and investor-owned properties are more likely to suffer neglect and disrepair (Travis 2019), increasing the risk of exposure to environmental hazards within the home.

After the placement of toxic infrastructure and housing divestment expose residents to health risks, ongoing regulation and abatement of these hazards becomes necessary but costly. In an era of chronically strained government budgets and powerful business interests, disadvantaged neighborhoods tend to suffer from weaker enforcement of environmental health protections. For the same reasons that low-income residents may lack the power to effectively resist placement of polluting infrastructure, governments and landlords often face few consequences for failing to enforce or properly implement hazard control policies in poor communities (Markowitz and Rosner 2013; Trounstein 2018).

Unequal patterns of siting, investing, and regulating shape an ongoing process of residential sorting that reproduces and perpetuates environmental inequality (Crowder and Downey 2010). As some neighborhoods become sites of noxious infrastructure, dilapidated housing, and unabated toxins, families that are able will pay to avoid them, thereby tightening the link between neighborhood composition and environmental hazards. Moreover, these neighborhoods may endure a process of “territorial stigmatization” that renders them irredeemable in the eyes of all but perhaps their present inhabitants, precipitating further cycles of divestment, neglect, and outmigration (Besbris et al. 2015; Wacquant 2008). This self-reinforcing process of residential sorting may calcify environmental inequalities over long periods of time, exposing residents of disadvantaged neighborhoods to harmful toxins throughout the life course. Below, we implicate exposure to lead, in particular, as a key mechanism accounting for neighborhood effects on cognitive development during early childhood.

3. Disadvantaged Neighborhoods and Environmental Lead Contamination

Neighborhood poverty is associated with higher concentrations of lead in the blood of resident children throughout the U.S. (Gleason, Nanavaty, and Fagliano 2019; Griffith et al. 1998; Lanphear et al. 1998; Moody, Darden, and Pigozzi 2016; Sampson and Winter 2016). As but one example, consider Monroe County, New York, where the odds of having an elevated blood lead level ≥ 10 $\mu\text{g/dL}$ (micrograms per deciliter) are nearly 50% greater for children living in the poorest tertile of neighborhoods than for children living elsewhere (Lanphear et al. 1998). These disparities arise primarily because disadvantaged neighborhoods are disproportionately contaminated by dust or chips from lead-based paint, outdated lead plumbing, and emissions from industrial facilities that process materials containing lead or from vehicles that once burned leaded gasoline (Agency for Toxic Substances and Disease Registry 2019; Muller et al. 2018).

Household contamination by lead-based paint is the primary source of lead exposure among contemporary cohorts of American children (Agency for Toxic Substances and Disease Registry 2019). During the first half of the 20th century, lead-based paint became a popular wall covering in residential structures, and it was widely used in interior construction until the 1970s. Over time, leaded paint peels, chips, and disintegrates into dust, which then settles on floors and window sills where it can be ingested by children. In 1978, the U.S. Consumer Product Safety Commission banned the manufacture of lead-based paint 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 (Agency for Toxic Substances and Disease Registry 2019; Gleason et al. 2019; Troesken 2006). Lead came into common use in water distribution systems as early as the 19th century due to its superior

combination of malleability and durability. Lead pipes and lead solder were widely used in household plumbing as well as in the service lines that connected homes to water mains conveying the local public supply. When lead plumbing corrodes, it can contaminate drinking water, which is then ingested by residents. The use of lead in potable water systems was prohibited by the U.S. Environmental Protection Agency (EPA) in 1986. Nevertheless, over six million lead service lines remain in use across the country, and older buildings still frequently contain lead plumbing (Cornwell, Brown, and Via 2016; Troesken 2006). The dangers associated with lead plumbing were exemplified by the Flint Water Crisis, which began in 2014 when the city of Flint, MI switched its public water supply and failed to properly apply corrosion inhibitors (Masten, Davies, and McElmurry 2016). As a result, unsafe levels of lead leached into the city's drinking water from its aging service lines, and thousands of residents were exposed to high concentrations of the toxin (Pieper et al. 2018).

The association between neighborhood poverty and lead contamination is due in part to the concentration of older, neglected dwellings in low-income communities, as the presence of lead paint and lead plumbing in homes is closely linked with their age and upkeep (Cox et al. 2011; Jacobs et al. 2002). Residential structures built after lead was banned from interior paints and potable water systems are much less likely to be contaminated, but the presence of lead is common in homes built earlier, and poor neighborhoods tend to have an older housing stock. Nevertheless, the link between lead exposure and neighborhood poverty persists even after adjusting for the age of homes (Gleason et al. 2019; Lanphear et al. 1998; Moody et al. 2016; Sampson and Winter 2016). This is in part because older homes in poor communities are less likely to have their lead safely contained and also because disadvantaged neighborhoods are contaminated by other sources of lead that are not directly linked to their residential structures.

In particular, a third important source of lead exposure involves airborne emissions, and relatedly, soil contamination (Mielke et al. 2013; Muller et al. 2018). Combustion of leaded gasoline was a major source of airborne lead emissions in the U.S. until the 1970s, when restrictions were first imposed on its use as a fuel additive. Today, the majority of airborne lead emissions come from industrial facilities that process materials containing lead, such as smelters and battery manufacturers, although pollution from these sources has also declined over time as the American economy deindustrialized (Ard 2015; Eckel, Rabinowitz, and Foster 2001; Elliott and Frickel 2013).

The contemporary legacy of leaded gasoline and a formerly robust metal processing industry is lead contamination of the soil (Eckel et al. 2001; Elliott and Frickel 2013; Mielke, Laidlaw, and Gonzales 2011). Airborne lead emissions eventually settle into the ground, where they can remain indefinitely because lead does not biodegrade. For example, the West Calumet public housing complex, located near a former smelter in East Chicago, IN, was found to have soil lead concentrations as high as 91,100 parts per million before it was hastily evacuated and later demolished in 2018 (Environmental Protection Agency 2019). Although this is an extreme case, many communities have soil lead levels in residential areas that far exceed EPA safety standards. And as with interior sources of lead contamination, soil lead levels also tend to be much higher in neighborhoods with lower income levels 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).

In addition to the concentration of lead in poor neighborhoods, patterns of racial residential segregation also disproportionately expose children from certain minority groups. Both historical and contemporary patterns of discrimination have concentrated Blacks and

Latinos in older, poorer, and more isolated sections of many American cities (Massey 2004; Massey and Denton 1998). As a result, areas with a greater proportion of Black and/or Latino residents are more likely to contain environmental health hazards, including lead (Ard 2015; Crowder and Downey 2010; Sampson and Winter 2016). Moreover, in some cities, the relationship between neighborhood racial composition and environmental lead contamination persists even after adjusting for economic differences (Griffith et al. 1998; Lanphear et al. 1998; Sampson and Winter 2016). This indicates that poor and segregated minority communities face distinctive environmental health burdens.

Harmful levels of lead remain in many neighborhoods across the U.S., but environmental regulations and other abatement efforts have aided in reducing the amount of the toxin to which individuals are exposed (Markowitz and Rosner 2013; Muller et al. 2018). As a result of the bans on leaded gasoline, paint, and plumbing together with the deindustrialization of the American economy, children's blood lead levels have declined over time, as have the disparities in exposure between poor children and their higher income peers (Wheeler and Brown 2013). Nevertheless, children living in disadvantaged communities remain at significantly greater risk of lead exposure, which can impede cognitive development even at very low levels.

4. Lead Exposure and Child Cognitive Development

Although any resident of a lead-contaminated environment may encounter the toxin, young children are the most susceptible to all of the exposure pathways outlined previously. At a simple level, children drink more water and breathe more air per unit of body weight than adults, which elevates their risk of poisoning in the presence of airborne lead particles or leaded plumbing. In addition, because they often play on floors and frequently engage in hand-to-mouth behavior,

infants and toddlers are more likely to inhale or ingest the remnants of lead-based paint and lead-contaminated soil. After the media have been ingested, children's bodies also absorb lead more easily and efficiently. For example, gastrointestinal absorption of lead is estimated to be 5 to 10 times greater in infants than in adults (Agency for Toxic Substances and Disease Registry 2019). Thus, the disproportionate contamination of disadvantaged neighborhoods by multiple sources of lead places their youngest residents at the greatest risk of exposure.

Within the central nervous system, lead inhibits proper absorption and binding of calcium and zinc ions, which disrupts many different biological processes, including energy metabolism, apoptosis, neurotransmitter release, cellular signaling, protein synthesis, and genetic regulation (Lidsky and Schneider 2003). Intense, high-dose exposure (e.g., blood-lead levels $>70\text{ }\mu\text{g/dL}$) causes acute symptomatic poisoning characterized by severe neurological injury, coma, or possibly even death. Cases of acute symptomatic poisoning are rare among recent cohorts of American children, who are typically exposed to lead at much lower doses. But even low-dose exposure (e.g., blood-level levels $<10\text{ }\mu\text{g/dL}$) is associated with subclinical toxicity in children, and there is no blood-lead level that is presently known not to pose any risk of adverse neurological effects during early childhood (Agency for Toxic Substances and Disease Registry 2019; World Health Organization 2010).

Numerous studies show that blood lead concentration is inversely related to measures of general intelligence among children, even at low levels less than $10\text{ }\mu\text{g/dL}$ (Bellinger et al. 1991; Canfield et al. 2003; Chiodo, Jacobson, and Jacobson 2004; Jusko et al. 2008; Lanphear et al. 2000, 2005). In fact, several studies suggest that the relationship between blood lead levels and intelligence is nonlinear, with greater cognitive impairments arising at lower concentrations (Canfield et al. 2003; Jusko et al. 2008; Lanphear et al. 2005). For example, Canfield and

colleagues (2003) estimate that, among children under 5 years old, an increase in blood lead concentration from 1 to 10 $\mu\text{g/dL}$ is associated with a decline in scores on the Stanford-Binet Intelligence Test of about one-half of a standard deviation; an additional increase in blood lead concentration from 10 to 30 $\mu\text{g/dL}$ is associated with a further, albeit considerably smaller, decline in test scores of about one-seventh of a standard deviation. Differences in measured intelligence between children with higher versus lower blood lead levels persist well into adulthood, and they are robust to adjustments for confounding (Reuben et al. 2017).

In addition to scoring lower on tests of general intelligence, children with higher blood lead concentrations also tend to perform significantly worse on school assessments of academic achievement (Aizer et al. 2018; Evens et al. 2015; Zhang et al. 2013). For example, in Detroit, children with elevated blood lead levels are much more likely to score “less than proficient” on standardized tests of reading, math, and science achievement (Zhang et al. 2013). Moreover, the association between lead exposure and student achievement may also be nonlinear, where declines in school performance are steepest at lower blood lead concentrations (Aizer et al. 2018; Evens et al. 2015). Beyond tests of intelligence and achievement, lead exposure is associated with many other dimensions of cognitive development, including impulsivity, conduct disorders, and delayed acquisition of psychomotor skills (Goodlad, Marcus, and Fulton 2013; Sampson and Winter 2018; Winter and Sampson 2017).

Because the central nervous system has little capacity for repair, the effects of lead are largely irreversible. Nevertheless, policy intervention may still help to mitigate the worst outcomes by reducing exposures for new birth cohorts and by attenuating the risk of sustained exposures for existing cohorts of children. The U.S. Department of Housing and Urban Development (HUD) provides grants to states, counties, and cities for which 65% of the

distributed funds must be spent directly on lead abatement, interim control, and risk assessment. Sorensen and colleagues (2019) estimate that these grants reduced the prevalence of elevated blood lead levels in children by over 70% between 2000 to 2008, leading to considerable improvements on tests of math and reading achievement. Other interventions, including medical surveillance and environmental regulation, show similar promise during early childhood (Billings and Schnepel 2018; Tong et al. 1998).

In sum, disadvantaged neighborhoods are disproportionately contaminated with lead from a variety of different sources, and exposure to the toxin, even at very low levels, seriously 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 cognitive development during early childhood by increasing the risk of exposure to neurotoxic lead.

5. Prior Studies

Among the only prior studies to evaluate the hypothesized causal process linking neighborhood poverty with individual outcomes via lead contamination is Manduca and Sampson (2019). Using aggregate cross-sectional data at the level of census tracts, these authors show that lead contamination accounts for part of the association between neighborhood poverty and rates of intergenerational income mobility. But few prior studies examine the effects of neighborhood poverty during early childhood, and none evaluate whether environmental hazards like lead mediate these effects. In this study, we follow a birth cohort over time, tracking where they live and the amount of toxin present in their residential environment, to examine whether and how neighborhood poverty affects cognitive development during the early life course.

6. A Graphical Causal Model

Figure 1 presents an abridged causal graph, where each arrow between variables represents a direct causal effect of arbitrary functional form. Specifically, it depicts a theoretical model of exposure to disadvantaged neighborhoods, environmental lead contamination, and child cognitive development. In this model and henceforth, A_t denotes the socioeconomic composition of a child's neighborhood at time t , M_t denotes a measure of the degree to which a child's neighborhood is contaminated by lead at time t , and Y denotes a child's cognitive ability at the end of follow-up. In addition, the model incorporates a set of observed baseline characteristics denoted by C , such as a child's race and gender, as well as a set of observed time-varying factors, such as family income and parental marital status, which are denoted by L_t . Finally, it includes two sets of unobserved variables, denoted by U and V .

The model in Figure 1 is quite general, as each variable observed throughout the course of development may affect all future variables. 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 . In other words, this model postulates that environmental lead contamination is both a direct cause of cognitive ability as well as a mechanism through which the effects of neighborhood poverty operate during early childhood.

Figure 1 also depicts the two challenges associated with endogenous neighborhood selection. First, it shows that there are unobserved confounding variables, U and V , that affect cognitive ability and that also affect selection into neighborhoods of different composition and with different levels of lead contamination. Second, it shows that observed time-varying

covariates confound the effects of future contextual exposures and, via the paths emanating from A_t and M_t into L_{t+1} , are also affected by prior contextual exposures. In this model, then, families select into different neighborhood environments dynamically, where past residential choices affect the time-varying determinants of future residential choices, consistent with prior research (Wodtke et al. 2011). These challenges inform our analytic strategy below.

7. Methods

7.1. Data

To investigate the joint effects of neighborhood disadvantage and environmental lead contamination, we combine and analyze data from the PHDCN, the CDPH, and the GeoLytics Neighborhood Change Database (NCDB). The PHDCN is a longitudinal survey that began in 1994 with a representative sample of over 6,000 children living within a constellation of neighborhood clusters, defined as groups of one to three census tracts in the City of Chicago. Data on sampled children and their families were collected through surveys fielded in 1994-1997 (baseline), 1997-1999, and 1999-2002. These surveys focused on children in seven different age groups, including a birth cohort under 1 year of age at baseline. Restricted-access data from the PHDCN identify the neighborhood cluster in which sample members lived at each wave of the study, which we use to match children with information on the socioeconomic composition of their local community and the degree of environmental lead contamination.¹ Our analytic sample

¹ Researchers interested in obtaining these data, which were licensed under special contractual arrangements designed to protect the anonymity of respondents, may apply for them through the Inter-university Consortium for Political and Social Research. A full set of replication files, absent the restricted data, are available from the authors upon request.

includes all $n = 1,266$ children from the birth cohort living within $j = 80$ neighborhood clusters at baseline.

Data on lead contamination come from the CDPH blood lead surveillance database. In 1993, the State of Illinois launched the Illinois Lead Program. This program mandated blood-lead screening for children who live in an area defined as “high risk” for lead poisoning, which includes the entire City of Chicago. Children are tested following a recommended schedule at 12, 24, and 36 months of age, with additional screening until age six if needed. Laboratories report all test results to the CDPH, where they are compiled into the surveillance database.² The database contains a tested child’s name, date of birth, and home address, as well as information about each test, including the laboratory where it was conducted, the date of collection, and the result in $\mu\text{g}/\text{dL}$. In total, it covers more than two million tests conducted since 1995. Access to these data is also restricted and can be obtained only via special contractual arrangements with the CDPH.³

² As part of the Illinois Lead Program, healthcare providers are legally required to order testing for children age 3 or under who reside in Chicago. Healthcare providers must also test older children between age 4 and 6 if they have not been screened previously. In addition, parents are required to provide proof of lead testing for their child upon enrollment in a daycare facility and/or kindergarten located within the city limits, and federal law mandates blood lead testing for all children receiving Medicaid. Although compliance is imperfect, these legal requirements generate blood lead testing data with extensive (80-90%) and representative coverage for the population of young children in Chicago. For example, Evens et al. (2015) find few appreciable differences when comparing the population of 3rd graders enrolled in Chicago Public Schools between 2003 and 2006 with children in the blood lead surveillance database born between 1994 and 1998.

³ Researchers interested in obtaining these data may apply for access through the CDPH. The CDPH disclaims responsibility for any analysis, interpretations, or conclusions drawn from them.

Information on the socioeconomic composition of neighborhoods comes from the NCDB, which contains harmonized tract-level data from the 1970-2010 U.S. Censuses and the 2006-2010 American Community Surveys.

We focus on Chicago because it is a large and representative urban center for which high-quality data on both cognitive ability and lead exposure during early childhood are available. The city is also highly stratified and suffers from lead contamination by all the sources outlined previously, making it an ideal case for studying the link between concentrated disadvantage, environmental health hazards, and child development in the U.S.

7.2. Measures

The exposure of interest in this study is the socioeconomic composition of a child's neighborhood cluster. 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, where higher values represent more disadvantaged neighborhoods and lower values represent less disadvantaged neighborhoods. We standardize it 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 or with additional socioeconomic indicators) as well as analyses based on the individual characteristics that compose it (e.g., the poverty rate by itself) yield nearly identical results, as all of these different measures are very highly correlated in Chicago.

To measure environmental lead contamination, we use the CDPH blood lead surveillance database. Specifically, we use these data to estimate the proportion of children under 6 years of age living in each neighborhood cluster with a blood lead level ≥ 5 $\mu\text{g/dL}$, separately for each year since 1995.⁴ We then smooth these annual estimates over time using kernel regression and match them to participants in the PHDCN at each wave of the survey. The prevalence of elevated blood lead levels in a neighborhood cluster provides an ecological proxy measure for the degree to which a local community is contaminated by lead from all sources. The threshold we use to define elevated blood lead levels is consistent with current monitoring guidelines from the U.S. Centers for Disease Control and Prevention and with prior research highlighting the danger of low-dose exposures. Parallel analyses based on thresholds of ≥ 6 $\mu\text{g/dL}$ and ≥ 10 $\mu\text{g/dL}$, as well as analyses based on the natural log of the local average blood lead level, yield similar results.⁵

The outcome of interest in this study is a child's cognitive ability, which we measure using scores on the third edition of the Peabody Picture Vocabulary Test (PPVT; Dunn 1997). The PPVT was administered as part of the third wave of the PHDCN between 1999-2002, when

⁴ We compute these estimates from the subset of tests that were conducted with venous blood samples and at labs with limits of detection < 5 $\mu\text{g/dL}$, as they are the most accurate and reliable. The median number of children per neighborhood cluster per year that are tested according to these criteria is 157, with an interquartile range of 86 to 252.

⁵ We also conducted parallel analyses based on estimates of elevated blood lead levels among children under 4 years old, under 3 years old, and under 2 years old. This was to account for the possibility that residential mobility between the time a child is exposed to lead and the time at which they are tested may distort how accurately our prevalence estimates reflect areal lead contamination. By focusing on younger children, these estimates limit the amount of time that may elapse between exposure and testing, and by extension, they also limit any distortions from moves between neighborhoods in the interim period. Results based on this set of alternative measures are similar to those we present in the main text.

most members of the birth cohort were between age four and five.⁶ The PPVT is a standardized assessment of one 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 to understand the world around them and because it is an important precursor for the acquisition of virtually all other cognitive abilities. The PPVT has desirable psychometric properties, including high validity and high reliability, even among linguistically diverse samples (Campbell 1998). Because vocabulary skills are typically the most important subcomponent of general intelligence tests, the PPVT also correlates highly with the Stanford-Binet Intelligence Scale and the Wechsler Intelligence Scale for Children (Hodapp 1993; Hodapp and Gerken 1999). In all multivariate analyses, we standardize scores to have zero mean and unit variance.

To adjust for confounding, we control for several covariates measured at baseline, including gender, race, 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 only recorded at the baseline wave of the PHDCN and thus are treated as time-invariant in this analysis. A child’s gender is coded as a binary variable, 1 for female and 0 otherwise. Race is expressed as a series of indicator variables denoting classification as “White,” “Black,” “Hispanic,” or “another race.” Family size is equal to the number of people living in a child’s household. The primary caregiver’s age at baseline is measured in years, and her education level is expressed using a series of indicator variables for “less than high school,” “high school graduate,” “some college,” and “college graduate.”

⁶ About 4% of sample members were assessed before age 4, and about 6% were assessed after age 5. We adjust for the age at which the PPVT was administered in all multivariate analyses.

Homeownership is also coded as a binary variable, where 1 indicates that a child's family owns their residence at baseline and 0 otherwise.

We additionally adjust for a set of time-varying covariates measured at each wave of the PHDCN. These include total household income in the previous year, parental marital status, and whether a child's primary caregiver is employed, receives public assistance, or speaks mainly English at home. The marital and employment status of the primary caregiver are coded as binary variables. Receipt of public assistance is also expressed as a binary variable indicating whether any of the primary caregiver's income was composed of benefits from programs like Aid to Families with Dependent Children. The home language environment is captured using an indicator variable coded 1 if the primary caregiver reports speaking mainly English and 0 otherwise. Finally, the PHDCN measured household income using interval response categories, from which we impute dollar values based on the interval midpoints. For the last open-ended interval capturing the highest incomes, dollar values are estimated using 1.4 times the interval's lower bound. Nominal incomes are then adjusted for price inflation using the Consumer Price Index – Research Series and normalized using the natural log transformation. Tables 1 and 2 provide descriptive statistics for all variables considered in this analysis.

7.3. Estimands

We rely on potential outcomes notation to define the effects of time-varying contextual exposures on an end-of-study outcome (Robins 1986; Rubin 1974). Let $\underline{a} = (a_1, a_2, a_3)$ denote a sequence of exposures to different levels of neighborhood disadvantage at waves $t = 1, 2$, and 3 of the PHDCN. Next, let $Y(\underline{a})$ denote a child's receptive vocabulary ability measured at the end of follow-up had she previously been exposed to neighborhoods with levels of disadvantage

given by $\underline{a}$. The observed outcome, Y , is assumed to equal the potential outcome, $Y(\underline{a})$, for the single exposure sequence that the child did in fact experience, while 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:

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

This expression represents the expected difference in vocabulary ability if children were exposed to the sequence of neighborhood conditions defined by $\underline{a}$ rather than some other sequence $\underline{a}'$.

With a continuous variable like our index of neighborhood disadvantage, there are infinitely many contrasts between different exposure sequences. Thus, we model the average total effects using the following parametric function:

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

where $\text{avg}(\underline{a}) = \sum_t a_t/3$ and β captures the overall effect of neighborhood composition from birth through the end of follow-up.

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

$$AJE(\underline{a}, \underline{a}', \underline{m}, \underline{m}') = E \left(Y(\underline{a}, \underline{m}) - Y(\underline{a}', \underline{m}') \right), \quad (3)$$

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

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

where $\{\gamma, \theta\}$ together capture the joint effects of concentrated disadvantage and environmental lead contamination. Although this model is restrictive, experimentation with a variety of more flexible specifications, including thin plate and tensor spline functions that permit complex forms of non-linearity and non-additivity, yield similar results.

Under this model, the average joint effects can be neatly 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

$$CDE(\underline{a}, \underline{a}') = E \left(Y(\underline{a}, \underline{m}) - Y(\underline{a}', \underline{m}) \right) = \gamma[\text{avg}(\underline{a}) - \text{avg}(\underline{a}')]. \quad (5)$$

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

$$CME(\underline{m}, \underline{m}') = E \left(Y(\underline{a}, \underline{m}) - Y(\underline{a}, \underline{m}') \right) = \theta[\text{avg}(\underline{m}) - \text{avg}(\underline{m}')]. \quad (6)$$

It 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 environmental lead contamination mediates the effect of neighborhood disadvantage on vocabulary ability. Under the models outlined previously, this difference can be expressed as follows:

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

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

7.4. Identification

The average total effect can be identified from observed data under the assumption of sequential ignorability, which is formally expressed as follows:

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

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

The average joint, controlled direct, and controlled mediator effects can be identified from the observed data under the following set of two sequential ignorability assumptions:

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

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

where, as before, $\perp$ denotes statistical independence and underbars denote variable histories. In words, the first of these assumptions states that the potential outcomes of exposure to both neighborhood disadvantage and environmental lead contamination, $Y(\underline{a}, \underline{m})$, must be independent of a child's observed exposure to neighborhood disadvantage at each time point conditional on the observed past. The second states that the potential outcomes must be independent of a child's observed exposure to lead at each time point conditional on the observed past. Substantively, these two assumptions imply that there must not be any unobserved confounders for the effects of either neighborhood disadvantage or environmental lead contamination on vocabulary ability.

These are strong assumptions. We attempt to satisfy them approximately by adjusting for the most powerful joint predictors of both neighborhood selection and child cognitive outcomes that are measured in the PHDCN. Then, to deal with the likely presence of unobserved confounding, we conduct a formal sensitivity analysis that reevaluates our findings across different hypothetical patterns of nonrandom selection into neighborhoods.

7.5. Estimation

Estimating the effects of time-varying exposures is methodologically challenging. Even if all relevant confounders have been observed and the effects of interest are identified, conventional methods of covariate adjustment remain biased when any of these confounders are themselves time-varying and affected by prior exposures (Elwert and Winship 2014; Robins 1986). As mentioned previously, 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 the time-varying characteristics of families, L_t .

Adjusting naively for time-varying confounders may lead to two forms of bias. First, it may lead to bias from over-control of intermediate pathways, as adjusting for a variable that lies on the causal pathway from an exposure of interest to the outcome partials out an indirect effect operating through the time-varying confounder. Second, it may also lead to endogenous selection bias if unobserved factors affect both the time-varying confounders and the outcome. In this situation, adjusting naively for time-varying confounders would induce a spurious association between the exposures of interest and the outcome (Elwert and Winship 2014).

To avoid these problems, we estimate contextual effects using the method of regression-with-residuals (RWR; Wodtke et al. 2020; Wodtke and Almirall 2017; Wodtke and Zhou 2020). RWR 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 over-control or endogenous selection.

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

$$E(Y|C, \underline{L}, \underline{A}, \underline{M}) = \alpha_0 + \alpha_1^T C^\perp + \alpha_2^T \sum_t L_t^\perp + \beta \text{avg}(\underline{A}) + \alpha_3 \sum_t M_t^\perp, \quad (11)$$

where $C^\perp = C - E(C)$ denotes a vector of baseline covariates centered around their marginal means, $L_t^\perp = L_t - E(L_t|C, \underline{L}_{t-1}, \underline{A}_{t-1}, \underline{M}_{t-1})$ denotes a residual transformation of the time-

varying covariates at each time point, and $M_t^\perp = M_t - E(M_t|C, \underline{L}_t, \underline{A}_t, \underline{M}_{t-1})$ similarly denotes a residual transformation of the mediator. These residual terms are estimated from a set of first-stage regressions for $E(L_t|C, \underline{L}_{t-1}, \underline{A}_{t-1}, \underline{M}_{t-1})$ and $E(M_t|C, \underline{L}_t, \underline{A}_t, \underline{M}_{t-1})$. They are then substituted into the outcome regression to adjust for observed confounding of the relationship between neighborhood disadvantage and vocabulary ability. If all these regressions are correctly specified and exposure to neighborhood disadvantage is sequentially ignorable, then RWR estimates of $\beta[\text{avg}(\underline{a}) - \text{avg}(\underline{a}')]$ from equation (11) are consistent and asymptotically normal for the $ATE(\underline{a}, \underline{a}')$.

Similarly, the average joint effects are estimated from another linear regression with the following form:

$$E(Y|C, \underline{L}, \underline{A}, \underline{M}) = \eta_0 + \eta_1^T C^\perp + \eta_2^T \sum_t L_t^\perp + \gamma \text{avg}(\underline{A}) + \theta \text{avg}(\underline{M}), \quad (12)$$

where $C^\perp$ and $L_t^\perp$ are residual terms defined as above. This regression differs from that outlined previously only in that it includes $\text{avg}(\underline{M})$ as a predictor, which is computed from untransformed rather than residualized values of the mediator, in order to estimate the joint effects of interest.

Under the assumptions of correct model specification and sequential ignorability for both neighborhood disadvantage and lead exposure, RWR estimates of $\gamma[\text{avg}(\underline{a}) - \text{avg}(\underline{a}')]$ and $\theta[\text{avg}(\underline{m}) - \text{avg}(\underline{m}')] from equation (12) are consistent and asymptotically normal for the $CDE(\underline{a}, \underline{a}')$ and $CME(\underline{m}, \underline{m}')$, respectively. By extension, their sum is consistent for the $AJE(\underline{a}, \underline{a}', \underline{m}, \underline{m}')$.$

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 levels of all covariates. They also constrain the partial associations between the covariates

and outcome to be additive and stable over time. In a set of ancillary analyses, we relax some of these constraints by including two-way interactions between elements of $C^\perp$ and $L_t^\perp$, on the one hand, with measures of neighborhood poverty and environmental lead contamination, on the other. This allows for certain patterns of effect moderation by characteristics of children and their families, such as race, gender, and income.

In addition, we also estimate the total and joint effects of interest 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 that satisfy the following two conditions: (i) the residualized confounders are orthogonal to future exposures, past exposures, and past confounders in the weighted sample, and (ii) the entropy of the weights is minimized. This method eliminates any correlation of the confounders with future exposures, conditional on the past, using weights with minimum variability. Regressions similar to those outlined previously but that omit the residualized covariates as predictors can then be fit to the weighted sample to estimate the effects of neighborhood disadvantage and environmental lead contamination. In this way, residual balancing properly adjusts for dynamic selection while obviating the need for restrictive modeling assumptions. This improved robustness, however, may come at the cost of lower efficiency compared with RWR.

We fit the regressions outlined previously by the method of least squares, and we compute standard errors for all effect estimates using the stratified cluster bootstrap with 500 replications to adjust for the PHDCN's complex sample design, where children were selected from within neighborhood clusters that were themselves selected from within a set of predefined

sampling strata.⁷ This analysis is then repeated across 50 complete datasets with missing values for all variables simulated via multiple imputation, and estimates are combined across datasets following Rubin (1987).⁸ Overall, the proportion of missing information in this analysis is about 17 percent, which is mostly due to panel attrition from the PHDCN over time.

7.6. Sensitivity Analysis

To account for the possibility that our effect estimates suffer from bias due to unobserved confounding, 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 (e.g., Brumback et al. 2004; Wodtke, Elwert, and Harding 2016).

Consider, for example, an analysis of a point-in-time neighborhood exposure, a . In this setting, bias in estimates of the average total effect would occur if

$$E(Y(a)|A = a, C) \neq E(Y(a)|A = a', C), \quad (13)$$

as the observed mean outcome is not exchangeable with the counterfactual mean outcome. The form and magnitude of this bias can be modeled with the following selection function:

$$s(a, a') = E(Y(a)|A = a, C) - E(Y(a)|A = a', C) = (a - a')\tau, \quad (14)$$

⁷ Following Rao and Wu (1988), we implement the nonparametric bootstrap for complex survey data by randomly selecting with replacement $j_k - 1$ neighborhood clusters from within each sampling stratum, where j_k denotes the total number of clusters in stratum k . In a set of ancillary analyses, we also attempted to improve precision under the PHDCN's complex sample design by fitting our models using generalized estimating equations with an exchangeable covariance structure, but the efficiency gains were trivial and the results nearly identical to those based on least squares.

⁸ We bootstrap within each multiply imputed dataset, which yields valid standard errors when performed with at least 20 imputations (Schomaker and Heumann 2018).

where τ is an unknown sensitivity parameter. Under this model, τ is equal to the bias in estimates of the average total effect. When $\tau > 0$, 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. When $\tau < 0$, 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. Theory and prior research on neighborhood selection suggest that $\tau < 0$ is more plausible than $\tau > 0$ in our application.

With this approach, a bias-corrected estimate for the average total effect can be obtained by (i) computing a bias term equal to $B(\tau) = \sum_{a'} (A - a') P(A = a') \tau$, (ii) using it to construct an adjusted outcome equal to $Y^{B(\tau)} = Y - B(\tau)$, and (iii) substituting the adjusted for the observed outcome into the regression used to estimate the effect of interest. These calculations effectively subtract the selection parameter τ from the naïve estimate of the total effect. Thus, 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, non-random selection into two point-in-time exposures, a and m , can be modeled as follows:

$$\begin{aligned} s(a, a', m, m') &= E(Y(a, m) | A = a, M = m, C) - E(Y(a, m) | A = a', M = m', C) \\ &= (a - a')\phi + (m - m')\psi. \end{aligned} \quad (16)$$

Under this selection model, the bias terms for point-in-time variants of the controlled direct, controlled mediator, and average joint effects are equal to ϕ , ψ , and $(\phi + \psi)$, respectively. Bias-corrected estimates of these effects can be computed by constructing a bias term equal to $B(\phi, \psi) = \sum_{a'} (A - a') P(A = a') \phi + \sum_{m'} (M - m') P(M = m') \psi$ and by replicating the

analysis on an adjusted outcome equal to $Y^{B(\phi, \psi)} = Y - B(\phi, \psi)$. As before, negative values of the sensitivity parameters, ϕ and ψ , are more consistent with theory and research on neighborhood selection than are positive values.

We generalize this approach for the present study where 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 here given by $B(\tau) = \sum_t \sum_{a_t'} (A_t - a_t') P(A_t = a_t') \tau$ and $B(\phi, \psi) = \sum_t \sum_{a_t'} (A_t - a_t') P(A_t = a_t') \phi + \sum_t \sum_{m_t'} (M_t - m_t') P(M_t = m_t') \psi$. Adjusted outcomes are computed as outlined previously and then used to refit equations (11) and (12), from which we can 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. Results are then reported in terms of sensitivity to multiples of observed confounding from nonrandom selection on this covariate.

8. Results

8.1. *The Distribution of Neighborhood Disadvantage and Environmental Lead Contamination*

Figures 2 and 3 describe the distribution of concentrated disadvantage and elevated blood lead levels 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 blood levels. Second, disadvantaged and lead-contaminated neighborhoods are spatially concentrated on Chicago's South and West Sides, which are predominantly Black and highly segregated. Consequently,

these contextual measures are highly correlated. For example, in neighborhoods that are one standard deviation above the citywide mean on the disadvantage index, an estimated 70% of children have elevated blood levels. In neighborhoods that are one standard deviation below the mean, by contrast, only about 20% of children are estimated to have elevated blood lead levels. Finally, even though neighborhood composition and environmental lead contamination are tightly coupled in Chicago, disadvantaged neighborhoods with relatively lower levels of lead contamination, and advantaged neighborhoods with relatively higher levels of lead contamination, both exist in nontrivial numbers.

8.2. The Joint Effects of Neighborhood Poverty and Environmental Lead Contamination

The upper panel of Table 3 presents estimates for the average total effect of sustained exposure to disadvantaged neighborhoods during early childhood on receptive vocabulary ability measured at about 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 citywide mean on the index of concentrated disadvantage rather than a neighborhood that is 0.9 standard deviations below the mean, which roughly correspond to the 75th and 25th percentiles, respectively. The first column contains estimates from our baseline RWR specification; the middle columns present RWR estimates from models that accommodate several different forms of effect moderation; and the final column presents residual balancing estimates. All of these approaches yield estimates for the total effect that are substantively large and statistically significant at stringent thresholds. They consistently indicate that living in a disadvantaged neighborhood throughout early childhood reduces receptive vocabulary ability by about one-third of a standard deviation.

The middle panels of Table 3 present estimates for the average joint effect, the controlled direct effect, and the controlled mediator effect. Specifically, like the total effect discussed previously, they contain estimates for a controlled direct effect that compare neighborhoods 0.7 standard deviations above, rather than 0.9 standard deviations below, the citywide mean on our index of concentrated disadvantage—a contrast that roughly corresponds with the 75th versus 25th percentiles. For the controlled mediator effect, they contain estimates that compare neighborhoods with an elevated blood lead prevalence of 65% rather than 30%, which also roughly correspond with the upper and lower quartiles of the citywide distribution. By extension, our estimates for the average joint effect compare neighborhoods with a more disadvantaged population and high levels of lead contamination to those with a less disadvantaged population and low levels of lead contamination.

According to estimates for the average joint effect, sustained exposure to a neighborhood where most residents are socioeconomically disadvantaged and 65% of children have elevated blood lead levels is predicted to reduce receptive vocabulary ability by about two-fifths of a standard deviation. Estimates for this 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 uniformly indicate that exposure to both disadvantaged and lead-contaminated neighborhoods during early childhood has devastating consequences for the development of language skills.

Moreover, estimates for the controlled direct and mediator effects suggest that, in Chicago, the deleterious impact of neighborhood disadvantage during early childhood may be driven almost entirely by disparate exposures to neurotoxic lead. Although they are imprecise, estimates for the controlled direct effect indicate that living in a more versus less disadvantaged

neighborhood would hardly reduce vocabulary abilities at all, if children were exposed to the same levels of environmental lead contamination. These estimates are all close to zero, and they universally fail to approach conventional thresholds for statistical significance. A failure to reject the null hypothesis that the controlled direct effect is zero here implies that we cannot rule out the possibility that the overall effect of neighborhood disadvantage may operate exclusively through environmental lead contamination. By contrast, estimates for the controlled mediator effect are all substantively large and statistically significant. They indicate that 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 children were all exposed to the same level of concentrated disadvantage.

The lower panel of Table 3 formally evaluates whether the average total effect differs from the controlled direct effect, which represents a test of the degree to which environmental lead contamination may explain the effect of neighborhood disadvantage on vocabulary ability. Estimates of the difference between the total and direct effect are all large in practical terms, and p-values from tests of the null hypothesis that these effects are equal provide considerable evidence against this possibility. Dividing the differences by estimates for the average total effect suggests the important mediating role of lead contamination: essentially all of the total effect is explained by a causal mechanism involving community exposure to lead. Owing to the imprecision of these estimates, however, our results are also consistent with only partial mediation. Taken together, these results indicate that, in Chicago, the effect of living in a disadvantaged neighborhood on vocabulary ability during early childhood operates largely, and perhaps even exclusively, through differences in environmental lead contamination.

Figure 4 provides a graphical summary of key findings by presenting a set of dose-response relationships computed from our baseline RWR specification. These plots relate PPVT scores with cumulative exposure to neighborhood disadvantage and environmental lead throughout early childhood. They show that sustained exposure to disadvantaged neighborhoods reduces vocabulary ability overall. They also show that sustained exposure to lead-contaminated neighborhoods reduces vocabulary ability, net of concentrated disadvantage and other covariates. And finally, they show that differences in lead contamination account almost entirely for the link between neighborhood composition and language development during early childhood.

8.3. *Sensitivity to Unobserved Confounding*

Figure 5 displays bias-corrected estimates from the sensitivity analysis described previously. The upper left panel summarizes the sensitivity of estimates for the average total effect to unobserved selection into neighborhoods with different levels of concentrated disadvantage. The upper right panel summarizes the sensitivity of estimates for the average joint effect to unobserved selection into neighborhoods of different composition and with different levels of environmental lead contamination. Similarly, the lower panels provide two-dimensional profiles showing the sensitivity of the controlled direct and mediator effects to these forms of selection. When the sensitivity parameters in these plots are equal to zero, they reproduce estimates from our baseline RWR specification. Larger values of the sensitivity parameters in absolute terms represent a greater degree of unobserved selection and thus a more pronounced bias correction.

Across all of the different plots, results indicate that our inferences about both the total and joint effects are highly robust to unobserved confounding. Even in the scenario where $\tau = \phi = \psi = 2$ and thus unobserved confounding is assumed to be twice as strong as that due to

parental education, bias-corrected estimates for the total, joint, and controlled mediator effects are all negative, substantively large, and statistically significant, while 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 vocabulary skills, this level of confounding by unobserved factors—above and beyond that due to characteristics we have measured and controlled—is extreme and seems unlikely, although we cannot rule it out empirically.

9. Discussion

The developmental effects of neighborhood poverty have been extensively studied among older children, but there is comparatively little research on contextual effects during early childhood, and the mechanisms hypothesized to explain them remain shrouded in a “black box” (Leventhal and Brooks-Gunn 2000; Sampson 2012). In this study, we investigate whether living in a disadvantaged neighborhood from birth through approximately the time of school entry affects child vocabulary skills, focusing on the mediating role of exposure to neurotoxic lead. Using novel counterfactual methods and unique longitudinal data, we find that growing up in a disadvantaged neighborhood substantially reduces vocabulary ability during early childhood and that the deleterious effects of spatially concentrated poverty among young children may operate largely through a causal mechanism involving environmental lead contamination.

These findings suggest an important role for neighborhoods in the genesis of cognitive disparities. Prior studies of child development indicate that socioeconomic differences emerge among children when they are still very young, with most implicating parental behavior, family resources, or genetics in generating these early gaps (Hurt and Betancourt 2016; Nisbett 2011).

Our results suggest that higher-order patterns of spatial stratification are also important determinants of cognitive development during early childhood. Above and beyond parenting and other characteristics of families, the neighborhoods in which children reside 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. The most influential perspectives linking neighborhood poverty to child development focus on intermediate mechanisms that are most relevant for older children and adolescents, even though disparities in many different outcomes become entrenched much earlier in the course of development. These mechanisms include, among others, differences in school quality, peer subcultures, access to adult role models, collective supervision, and informal social control (Harding 2010; Jencks and Mayer 1990; Sampson 2012; Wilson 1987). Each of these mechanisms may be important for transmitting neighborhood effects on certain outcomes and at certain times, but their influence during the most critical early phases of development is circumscribed by natural limitations on the social interactions of infants and toddlers. This study supports an alternative theoretical model that views neighborhood effects as first arising from disparities in exposure to environmental health hazards during early childhood. It also suggests that, in the search for mechanisms connecting the socioeconomic composition of neighborhoods to child outcomes, attention to the developmental specificity of different putative mediators will be 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 it reveals little about intermediate mechanisms that might serve as points of effective intervention (Sampson 2012; Sharkey and Faber 2014). Short of conducting a sequentially randomized field

experiment, our analysis provides some of the more credible evidence that neighborhood disadvantage affects cognitive development during early childhood. In addition, we also identify a cogent mechanism—environmental lead contamination—that explains these effects and is amenable to intervention. Thus, our study has implications for policy. It suggests that programs to abate lead in homes, replace lead plumbing, and remove lead-contaminated soil will not only improve child outcomes overall but may also mitigate neighborhood effects on cognitive development.

Finally, this study contributes to methods for research on contextual effects by introducing new procedures for estimating the effects of time-varying exposures. RWR and residual balancing avoid the problems that afflict conventional methods in the presence of dynamic selection. And compared with other methods that similarly avoid these problems, such as inverse probability weighting, RWR and its variants are more robust and efficient, especially when used with continuous exposures (Wodtke et al. 2020; Zhou and Wodtke 2020). Because dynamic selection and continuous exposures are ubiquitous in the social sciences, we expect these methods to find wide application.

Although this study makes important contributions to theory, methods, and policy, it is not without limitations. First, despite our best 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 but one dimension of cognitive development—receptive vocabulary ability. Although the formation of language skills is critically important, many other abilities may also be sensitive to neighborhood conditions during early childhood, and we are unable to assess them. Third, we only analyze data on the areal risk of lead exposure, as we lack

the information needed to match children in the PHDCN with their individual blood lead levels in the CDPH surveillance database. Even if it were possible, however, analyses of individual test results present their own challenges. In particular, they are subject to measurement error of a considerably greater magnitude than areal aggregates, which would function to understate the explanatory role of lead exposure. Finally, we rely on data from a cohort of children born in Chicago nearly three decades ago, even though the degree to which lead exposure explains neighborhood effects may vary across time and place.

Indeed, blood levels among children in Chicago have declined substantially since the launch of the PHDCN. Figure 6 shows the distribution of elevated blood lead prevalence rates across Chicago neighborhood clusters from 1995 to 2010, revealing a remarkable reduction in exposure over time. By 2010, few neighborhoods in Chicago exhibit an elevated blood lead prevalence over 30%, even though much higher rates were typical only a decade earlier. The steep decline in lead exposure in part reflects the success of surveillance, regulation, and abatement efforts expanded as part of the Illinois Lead Program since the 1990s, affirming the amenability of this mechanism to intervention. It also suggests that, at present, environmental lead contamination may be a less powerful mediator of neighborhood effects that it was in the past.

Similarly, urban areas in the Midwest and Northeast tend to be more contaminated by lead than other parts of the country owing to their widespread use of lead plumbing, their history as home to a robust metal processing industry, and the timing of much of their residential construction (Jacobs et al. 2002; Pell and Schneyer 2016). The Chicago water supply, in particular, is thought to have the most lead service lines currently in use across the entire U.S. (Spielman 2020), and historically, the city housed a large number of lead smelters. As a result,

what makes Chicago an ideal case for an initial evaluation of our theoretical model might also make it a special case, where the mediating role of environmental lead contamination is more pronounced than in other communities.

Nevertheless, children in disadvantaged neighborhoods remain at greater risk of lead exposure not only in Chicago but also in many other cities across the contemporary U.S., and even at very low doses, lead causes serious harm to their developing brains. Moreover, lead is just one of many different neurotoxins that disproportionately contaminate poor communities. Arsenic, mercury, manganese, and polychlorinated biphenyls, among other chemicals known or suspected to interfere with the central nervous system, are also more likely to be found in neighborhoods with lower income levels and with greater numbers of racial minorities (Hamblin 2014; Israel 2012). And although exposures to these chemicals have declined over time due in part to effective policy intervention, the U.S. federal government has recently revoked or otherwise rolled back a large number of regulations governing the emission or disposal of toxic substances (Popovich, Albeck-Ripka, and Pierre-Louis 2020). 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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Figures

Figure 1. Hypothesized causal relationships between baseline covariates (C), time-varying confounders (L_t), neighborhood disadvantage (A_t), environmental lead contamination (M_t), cognitive ability (Y), and unobserved factors (U, V) among children.

Figure 2. Spatial Distribution of Concentrated Disadvantage and Elevated Blood Lead Levels in Chicago, 1997.

Notes: In accordance with CDPH policy, estimates for census tracts with fewer than 10 tested children are suppressed from this figure. We impute these suppressed estimates from nearby tracts with a sufficiently large number of observations for visual continuity.

Figure 3. Bivariate Relationship between Concentrated Disadvantage and Environmental Lead Contamination across Chicago Census Tracts, 1997.

Notes: In accordance with CDPH policy, tracts with fewer than 10 tested children in 1997 are suppressed from this figure.

Figure 4. Dose-response Relationship of Neighborhood Disadvantage and Environmental Lead Contamination with Receptive Vocabulary Ability, Project on Human Development in Chicago Neighborhoods birth cohort (n=1266).

Notes: Dashed lines represent 95% confidence intervals based on the stratified cluster bootstrap with 500 replications.

Figure 5. Bias-corrected effect estimates, Project on Human Development in Chicago Neighborhoods birth cohort (n=1266).

Notes: In the two-dimension plots, dashed lines represent 95% confidence intervals based on the stratified cluster bootstrap with 500 replications. In the three-dimensional plot, the dark grey plane represents the bias-adjusted point estimates, the light grey planes represent 95% confidence intervals, and white plane provides a reference at zero. Tau, phi, and psi are scaled to equal the bias eliminated from our focal effect estimates by virtue of adjusting for parental education.

Figure 6. Temporal Changes in Environmental Lead Contamination among Chicago Neighborhoods, 1995-2010.

Notes: This plot contains kernel densities computed over all neighborhood clusters in Chicago for a given year.

Tables

Table 1. Time-invariant sample characteristics measured at baseline, Project on Human Development in Chicago Neighborhoods birth cohort (n=1266)

Variable	Mean	SD
<i>Child characteristics</i>		
Female	0.49	-
Race/ethnicity		
White	0.16	-
Black	0.30	-
Hispanic	0.49	-
Other	0.05	-
<i>Family characteristics</i>		
Family size	5.26	2.08
Homeowner	0.20	-
<i>Primary caregiver characteristics</i>		
Age	27.36	6.92
Education		
Less than high school	0.44	-
High school graduate	0.13	-
Some college	0.31	-
College graduate	0.12	-

Notes: Results are combined estimates from 50 imputations.

Table 2. Time-varying sample characteristics, Project on Human Development in Chicago Neighborhoods birth cohort (n=1266)

Variable	Wave 1 (1994-1997)		Wave 2 (1997-1999)		Wave 3 (1999-2002)	
	Mean	SD	Mean	SD	Mean	SD
<i>Contextual measures</i>						
Concentrated disadvantage	-0.06	0.74	-0.08	0.78	-0.13	0.77
Elevated blood lead prevalence (≥ 5 $\mu\text{g/dl}$)	0.54	0.17	0.48	0.18	0.39	0.16
<i>Child outcomes</i>						
Peabody Picture Vocabulary Test scores	-	-	-	-	41.91	27.33
<i>Primary caregiver characteristics</i>						
Family income (log)	9.65	1.08	9.85	1.05	10.09	0.94
Employed	0.42	-	0.52	-	0.58	-
Receives public assistance	0.40	-	0.28	-	0.18	-
Married	0.51	-	0.55	-	0.57	-
Speaks primarily English	0.67	-	0.72	-	0.73	-

Notes: Results are combined estimates from 50 imputations.

Table 3. Estimated effects of neighborhood disadvantage and environmental lead contamination on receptive vocabulary ability, Project on Human Development in Chicago Neighborhoods birth cohort (n=1266)

Estimand	Linear and additive RWR		RWR + gender moderation		RWR + race moderation		RWR + income moderation		Residual balancing	
	est.	p-value	est.	p-value	est.	p-value	est.	p-value	est.	p-value
Average total effect (ATE)	-0.358 (0.106)	<0.001	-0.358 (0.106)	<0.001	-0.356 (0.107)	<0.001	-0.352 (0.107)	0.001	-0.345 (0.109)	0.002
Average joint effect (AJE)	-0.412 (0.103)	<0.001	-0.417 (0.103)	<0.001	-0.412 (0.104)	<0.001	-0.408 (0.104)	<0.001	-0.401 (0.108)	<0.001
Controlled direct effect (CDE)	-0.027 (0.160)	0.866	-0.037 (0.161)	0.818	-0.025 (0.161)	0.879	-0.039 (0.162)	0.809	-0.056 (0.169)	0.741
Controlled mediator effect (CME)	-0.385 (0.135)	0.004	-0.380 (0.136)	0.005	-0.387 (0.137)	0.005	-0.369 (0.138)	0.007	-0.345 (0.144)	0.016
Test for mediation (ATE - CDE)	-0.331 (0.122)	0.007	-0.321 (0.124)	0.010	-0.331 (0.123)	0.007	-0.312 (0.125)	0.012	-0.289 (0.141)	0.040

Notes: Results are combined estimates from 50 imputations. Standard errors in parentheses are computed using the stratified cluster bootstrap with 500 replications, and p-values are from z-tests of the null hypothesis that the focal estimand is equal to zero.