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Environmental Inequality: The Social Causes and Consequences of Lead Exposure

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Abstract

In this article, we review evidence from the social and medical sciences on the causes and effects of lead exposure. We argue that lead exposure is an important subject for sociological analysis because it is socially stratified and has important social consequences—consequences that themselves depend in part on children's social environments. We present a model of environmental inequality over the life course to guide an agenda for future research. We conclude with a call for deeper exchange between urban sociology, environmental sociology, and public health, and for more collaboration between scholars and local communities in the pursuit of independent science for the common good.

INTRODUCTION

Scholars have long understood that lead is a dangerous neurotoxin (Hamilton 1929), but, with few exceptions, sociologists have been slow to grasp its significance for the study of inequality. This seems puzzling at first, given ample evidence that lead exposure is socially stratified and can undermine children's development. Although the Centers for Disease Control and Prevention (CDC 2012) maintains that there is no safe level of lead exposure, racial and class disparities in exposure are substantial and enduring (Reed 2011), with African Americans and the poor bearing the greatest risk. Average blood lead levels today are highest in poor communities of color (Sampson & Winter 2016). Among both individuals and communities, lead exposure in the United States follows classic lines of social stratification.

It is hard to be sure why sociologists have paid relatively little attention to lead exposure's implications for inequality, but we suspect that there are two primary reasons.[1] The first is that many sociologists, like much of the general public, supposed that the problem was mostly solved. Lead was phased out of newly manufactured gasoline and paint by the mid-1980s, eliminating two major sources of exposure (Markowitz & Rosner 2013). These landmark regulations were rightfully deemed major victories for public health (Needleman 2004). However, as the crisis in Flint, Michigan brought to light, the victory is far from complete.

In April 2014, in an effort to save money, the city of Flint began drawing water from the Flint River (Hanna-Attisha et al. 2016). Water from the river is far more corrosive than water from Lake Huron, the city's previous source. As it passed through Flint's aging water system, the river water began loosening lead from the pipes' inner walls (Pieper et al. 2017). Residents complained about the water's color, smell, and taste, but public officials assured them that it was safe to use. By the fall of 2015, researchers had vindicated residents' concerns: Many homes had lead levels well above Environmental Protection Agency (EPA) limits (Kennedy et al. 2016). In January of 2016, President Barack Obama declared a state of emergency.

The crisis in Flint alerted scholars and the general public to the continuing dangers of lead exposure, particularly in poor communities neglected by public officials (Olson & Fedinick 2016). Research showed that the highest concentrations of lead in Flint were located in its most disadvantaged neighborhoods (Hanna-Attisha et al. 2016). But only a year later, a *Reuters* investigation revealed that Flint's problems were far from unusual. *Reuters* found nearly 3,000 areas where the proportion of children with elevated lead levels exceeded that of Flint (Pell & Schneyer 2016). The evacuation of an entire neighborhood in East Chicago, Indiana, because of toxic levels of lead in the soil also made national news (Goodnough 2016). Lead exposure, once again, had captured the public's attention.

We suspect that, beyond underestimating the extent of the problem, there is a second reason for sociologists' relative lack of attention to the connection between lead exposure and inequality. The most direct mechanisms through which lead exposure affects behavior are biological. Scholars might consequently conclude that studying lead exposure is the proper domain of natural more than social science. But, for two reasons, we believe that lead exposure is an important subject for sociological inquiry.

First, as we discuss below, who is exposed to lead and why are fundamentally social questions. Like solitary confinement (Gawande 2009) and proximity to neighborhood violence (Massey 2004, Sharkey & Sampson 2015), lead exposure affects people's bodies and brains, but its causes

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[1]Exceptions include Bullard (1994), Reed (2011), Stretesky (2003), and Stretesky & Lynch (2001, 2004).

are social.[2] Historical research documenting the social, political, and industrial origins of exposure to environmental toxins challenges a tradition of biological determinism in social science that has long been used to explain and justify inequality. Second, lead exposure not only has social causes; it also has social consequences—consequences that themselves depend in part on children's social environments. As environmental sociologists have long recognized, understanding how exposure to environmental toxins such as lead might narrow or widen inequality in test scores, health, behavioral problems, or crime is an essential area of future research.

Foundational studies in urban sociology linked the health of the public to housing conditions, city infrastructure, and industrial toxins (Sampson 2012). In this article, we seek to place lead exposure and, more broadly, environmental inequality firmly within this tradition. We argue that selection into lead exposure, as into poor neighborhoods (Sampson 2012) or incarceration (Wildeman & Muller 2012), is not merely a statistical nuisance, but an important object of sociological analysis in its own right. To substantiate these claims, we first describe the primary sources of lead exposure in the United States, explaining why this dangerous toxin is so pervasive in the first place. Next, we turn to lead's distribution, which we show is socially stratified. We propose that lead exposure, because of its social and geographic concentration, is an underappreciated type of neighborhood effect: a source of inequality that is literally embodied (Krieger 2005, Massey 2004; see also Massey & Denton 1993, Sampson 2012, Sharkey 2013, Wacquant 2008, Wilson 1987). We then review the latest evidence of lead exposure's consequences.[3] Taken together, this evidence suggests that lead has detrimental effects on a number of cognitive, physical, behavioral, and social outcomes of concern to sociologists. In synthesizing this body of work, we provide a theoretical model of how lead exposure contributes to the reproduction of inequality over the life course. We close with a discussion of the implications of the latest research for sociology and public policy.

SOURCES OF LEAD EXPOSURE

Since the late nineteenth century, lead exposure in the United States has come from four primary sources: water pipes, gasoline exhaust, smelting plants, and chipping paint. American cities began installing lead pipes to distribute water roughly 150 years ago (Troesken 2006). Lead pipes offered engineering advantages over iron pipes because lead is both more malleable and more durable than iron (Troesken & Beeson 2003). Cities that could afford the initial investment typically used lead pipes because they could be bent around obstacles when they were installed and, once installed, lasted longer than iron pipes. Although lead in plumbing was banned in 1986 (Weitzman et al. 2013), millions of lead service lines are still in use today (Cornwell et al. 2016). Cities using lead pipes installed before 1986 typically treat their water to prevent corrosion, in accordance with the EPA's Lead and Copper Rule (EPA 1991). But a 2016 report by the Natural Resources Defense Council found that thousands of cities had broken the rule in the previous year (Olson & Fedinick 2016).

Researchers at General Motors first put lead in gasoline in 1921. The additive prevented engine knock in automobiles, a problem whose solution had long eluded engineers. In 1924, General Motors and Standard Oil formed the Ethyl Corporation to sell tetraethyl lead, a compound so

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[2]Denno (1992, p. 385) argues, "although lead poisoning is oftentimes 'biological-looking' because it can lead to permanent physical disorders, such as neurodevelopmental delay and intellectual deficit, its origins are environmental."

[3]Owing to space constraints, we focus our review primarily, although not exclusively, on studies based in the United States. The causes and consequences of lead exposure elsewhere merit their own review.

dangerous that it could kill a person if it was absorbed through the skin (Denworth 2008). That fall, five workers died while making tetraethyl lead at a Standard Oil plant in New Jersey. Two more died at a plant in Dayton, Ohio, the same year, and more than sixty fell gravely ill, some suffering from terrifying hallucinations (Denworth 2008). After news of the deaths reached the public, the surgeon general weighed the conflicting opinions of industry and public health advocates. "Confronted with an early choice between corporate interests demanding absolute proof of harm and health experts insisting on absolute proof of safety," writes journalist Lydia Denworth (2008, p. 55), "America chose business." With few restrictions on the use of tetraethyl lead, "millions of tons of lead were put into the environment between 1925 and 1986, when it was finally taken out of gasoline." The deleading of gasoline was one of the great public health victories of the twentieth century: Blood lead fell in tandem with lead in gasoline (Markowitz & Rosner 2013). But residue from decades of emissions remains in the soil of communities across the country (Mielke et al. 2007, 2011; Zahran et al. 2013).

Industrial smelting plants have left a similar legacy (Albalak et al. 2003, Elliott & Frickel 2013, Vargas et al. 2001). Although most plants that emitted lead into the environment have been shuttered for years (Eckel et al. 2001), the soil that surrounds them still often contains dangerous amounts of lead. Soil lead is especially harmful to children because they play outdoors, crawl, and frequently put their hands and objects in their mouths (Health Impact Proj. 2017).

Americans mixed lead with paint even before they mixed it with gasoline. In 1906, National Lead founded Dutch Boy, which marketed lead paint "as the covering of choice to millions of young families" (Markowitz & Rosner 2013, Warren 2001). Household lead paint was banned in 1978, but old lead paint continues to adorn the walls and exteriors of millions of US homes, especially in low-income communities of color (Cox et al. 2011, Jacobs et al. 2002). When lead paint peels and turns to dust, children can inhale and ingest it. Today, lead paint chips and dust from paint and soil are "far and away the major source of exposure in children" (Weitzman et al. 2013, p. 190).

INEQUALITY IN LEAD EXPOSURE

In the late 1960s and early 1970s, when community groups such as the Young Lords and the Black Panthers were calling attention to lead exposure's disproportionate impact on poor communities of color (Markowitz & Rosner 2013, Nelson 2016), there were no centralized data on blood lead levels. In 1976, the National Health and Nutrition Examination Survey (NHANES) began measuring blood lead levels in a nationally representative sample of Americans. The first analysis of NHANES data collected from 1976 to 1980 confirmed community activists' suspicions: 12.2% of black children, compared with 2% of white children, had elevated blood lead levels (Mahaffey et al. 1982), then defined as 30 µg/dL or higher (six times the CDC's current monitoring threshold of 5 µg/dL). The study's authors found racial disparities at all income levels and in both urban and rural areas, as well as income disparities among both African Americans and whites.

Since the publication of these initial results, researchers have consistently documented wide racial and class inequalities in exposure to lead. As recently as 1990, air lead concentrations were highest in the counties with the greatest proportions of black youth and the lowest median incomes (Stretesky 2003). An analysis of NHANES data from 2007 to 2010 finds that 7.7% of black children and 3.2% of white children aged 1 to 2 years had blood lead levels above 5 µg/dL (Raymond et al. 2014). The percentage for children on Medicaid was six, compared with half a percent for all other children.

Studies of single states and cities have yielded similar results. Lanphear et al. (1996) find that black children in a sample in Rochester, New York, were exposed to more lead-contaminated house dust than white children, although white children's homes had higher water lead levels (see

also Haley & Talbot 2004). Vivier et al. (2011) and Krieger et al. (2003) show that more than 30% of children in the poorest neighborhoods in Rhode Island had lead levels exceeding 10 µg/dL. Moody et al. (2016) report that children living in predominantly black and low-income segregated neighborhoods in Detroit had the highest average blood lead levels in the city, although there was no gap between black and white children in the poorest neighborhoods (see also Kaplowitz et al. 2010). Sampson & Winter (2016), in contrast, report that the racial disparity in blood lead in Chicago persisted even after adjusting their estimates for neighborhood-level education, poverty, and the age, vacancy, and dilapidation of neighborhood housing. In some of the neighborhoods Sampson & Winter (2016) study, more than 90% of children had elevated blood lead levels in 1995 (see also Oyana & Margai 2010).

According to Markowitz and Rosner (2013), lead companies historically seized on the fact that lead is concentrated in poor communities of color to minimize their responsibility for its effects. Throughout the twentieth century, both the Ethyl Corporation and the Lead Industries Association (LIA), the lead industry's trade association, funded research that downplayed the risks of exposure (Denworth 2008, Markowitz & Rosner 2013). "When political opposition to the use of lead paint grew in the 1950s," Markowitz & Rosner (2013, p. 210) note, "the LIA leadership argued that lead poisoning was the fault of 'ignorant' 'Negro and Puerto Rican families' rather than of the companies that had sold, marketed, and profited from the decades-long pollution campaign." In recent toxic tort cases, "blaming children's genetic inheritance has become a popular strategy of lead companies" (Shostak 2013, p. 172).

Although racial disparities in lead exposure are well documented, evidence for precisely why they exist is comparatively scarce (Currie 2011). Crowder & Downey (2010) and Kravitz-Wirtz et al. (2016) show that African Americans and Latinos are exposed to much higher levels of industrial pollution than whites, even at similar levels of income. Crowder & Downey (2010) find that African Americans are both more likely to move into heavily polluted neighborhoods and less likely to leave, attributing these differences primarily to discriminatory real estate practices that restrict African Americans' and Latinos' geographical mobility. Schulz et al. (2016) find that communities of color in the Detroit metropolitan area are more likely to be surrounded by hazardous facilities and are disproportionately exposed to pollution.

Residential segregation and discriminatory housing markets may similarly increase African Americans' and poor children's exposure to lead. Aizer & Currie (2017), for instance, find that black children and children receiving free school lunch in Rhode Island were more likely to live in high-traffic neighborhoods where their risk of being exposed to gasoline emissions was greater. Sampson & Winter (2016) find that children's average blood lead levels were higher in neighborhoods that contained or were adjacent to lead smelting plants, although this source of lead accounted for only a small proportion of the racial gap in average blood lead levels in Chicago. These findings are consistent with a long tradition of research in urban sociology showing that poor black neighborhoods characterized by concentrated disadvantage are "ecologically distinct" from poor neighborhoods generally (Sampson 2012, p. 101; Wilson 1987). Sampson & Winter (2016, p. 264) use the term "toxic inequality" to describe "the magnitude of the association between the spatial isolation of African Americans and the prevalence of lead poisoning."

Low-income families often lack the power and resources to remove lead from their homes or to move away when they cannot (Aizer 2017, Brulle & Pellow 2006, Bullard 1994). "From the very first," write Markowitz & Rosner (2013, p. 36),

> lead poisoning and housing were inextricably linked. For housing officials, removing lead paint was (and still is) an expensive procedure that landlords were often unwilling to undertake. And housing officials in the few cities that passed regulations to control lead often ignored these housing codes,
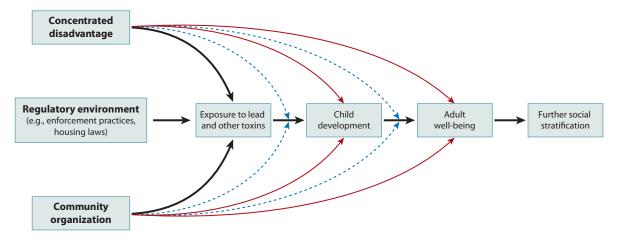
Figure 1

A model of inequality, environmental toxicity, and well-being over the life course. Thick black arrows represent the causal effects we discuss throughout the article. Thin red arrows represent the direct effects of concentrated disadvantage and community organization on child development and adult well-being. Failure to account for these effects can lead researchers to generate biased estimates of the effect of lead exposure. Dashed blue arrows indicate that the effects of lead exposure on child development, and the effects of child development on adult well-being, will vary depending on the level of concentrated disadvantage and community organization in the neighborhood where a child grows up. Concentrated disadvantage, community organization, and the regulatory environment are themselves correlated, but we omit these relationships from the figure because we do not focus on them in the text.

fearing that the expense of abatement would prompt landlords to abandon their properties. Further, effective enforcement required a huge army of inspectors, personnel that were unavailable to local departments of health with limited budgets. Finally, identifying dilapidated interiors was itself difficult because most poor tenants were unaware of their rights to a safe home even under the existing housing codes, or they were afraid they might be evicted if they filed a complaint. Even when buildings with peeling lead paint were identified, it might take months, even years for a landlord to be hauled into court, and even then the fines were generally minimal, leading landlords to forego expensive repairs and pay the eventual fine instead.

We depict these sources of national, neighborhood, and household lead exposure in **Figure 1**, which illustrates our conceptual model of environmental inequality and sketches an agenda for future research. Lead is present in water, paint, and soil because of historically weak environmental regulations. Current regulations, moreover, are still insufficient to prevent children from being exposed. Poor families of color, in particular, tend to live in neighborhoods of concentrated disadvantage, where lead is prevalent. These families often lack the resources and organizational access to eliminate environmental toxins from their homes and communities.

Sociologists should devote more attention to documenting how these and other causes sustain inequality in exposure to lead. For example, given the mounting evidence that lead exposure impedes children's development, why are government soil remediation efforts not more widespread? Why do city-level lead-paint regulations vary so dramatically? How do landlords justify maintaining lead paint in their properties? Sociology is well positioned to take on these questions because of its theoretical and methodological breadth. Research in this area would also align with a renewed interest among sociologists in housing as a source of inequality (Desmond & Bell 2015, Pattillo 2013, Rosen 2014).

MECHANISMS AND CONSEQUENCES OF LEAD EXPOSURE

Lead exposure is most harmful to young children, who absorb lead more efficiently and are more sensitive to toxicants than older children or adults (Lidsky & Schneider 2003, Needleman 2004, Silbergeld 1997). Lead's effects stem in part from its ability to mimic or compete with calcium in the body and damage the central nervous system, impairing brain development and neurotransmitter systems (Dietrich et al. 2001, Finkelstein et al. 1998, Lidsky & Schneider 2003, Needleman 2004, Troesken 2006). Cecil et al. (2008) document a negative correlation between childhood blood lead and adult brain volume in regions associated with executive function and childhood behavioral problems.

The CDC previously recommended monitoring children with blood lead levels above 10 μg/dL, but subsequent research revealed that much lower levels of exposure can have harmful effects (Canfield et al. 2003, Lanphear et al. 2005). These findings prompted the CDC to declare that there is no safe level of lead exposure and to change the level at which children should be monitored to 5 μg/dL.[4]

Previous scholarship has linked lead's effects on cognition and behavior to social outcomes ranging from test scores to health and crime. We review research in each of these areas below.[5] To some extent, estimating the effect of lead exposure is more straightforward than estimating the effect of other determinants of children's development, such as education. Children are exposed to lead before they are old enough to decide where to live, and parents and health professionals are often unaware of the presence of contaminants like lead in the soil surrounding long-shuttered smelting plants. But, as the research discussed above indicates, studies consistently show that poor black and Latino children are much more likely than other children to be exposed to lead. Because these children face multiple correlated adversities (Western 2014), it is often difficult to isolate the effect of lead on their development (Bellinger 2008). **Figure 1** illustrates this problem, showing that concentrated disadvantage and the degree of community organization in a child's neighborhood affect their development both directly and indirectly by increasing their likelihood of lead exposure.[6] Failing to account for the direct effects of concentrated disadvantage and community organization can lead researchers to generate biased estimates of the effect of lead exposure. In our review of the evidence, we place the most importance on studies that have sought to confront this challenge by identifying exogenous sources of variation in lead exposure.

Test Scores

Probably the largest literature on the consequences of lead exposure examines its effects on cognition, most commonly assessed using IQ and other test scores (Bellinger et al. 1992, Needleman & Gatsonis 1990, Pocock et al. 1994, Reuben et al. 2017). Children exposed to lead score comparatively low on standard IQ tests, with the sharpest declines occurring below 10 μg/dL (Canfield et al. 2003, Jusko et al. 2008, Lanphear et al. 2000, Lanphear et al. 2005, Rothenberg & Rothenberg 2005, Schwartz 1994). Lanphear et al. (2005), for instance, find that an increase in childhood blood lead from 2.4 to 10 μg/dL was associated with a 3.9-point IQ-score decline. These differences

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[4]For a discussion of the debates over the regulatory threshold for lead, see Markowitz & Rosner (2013, pp. 87–121).

[5]Although we do not include in our review older studies of the consequences of occupational lead exposure among adults, their findings are very similar.

[6]Concentrated disadvantage, community organization, and the regulatory environment are themselves correlated, but we omit these relationships from the figure because we do not discuss them here.

can persist into adulthood: One study found that children with relatively high blood lead levels had lower IQ test scores even when they were retested at age 38 (Reuben et al. 2017).

In recent years, scholars have found that lead exposure is correlated with poor performance on school tests as well (Amato et al. 2012). Using linked blood lead and test score data for children in Detroit, Zhang et al. (2013) find that having elevated blood lead levels considerably increases children's chances of scoring less than proficient on mathematics, science, and reading tests. A follow-up analysis that limited the sample to children matched on covariates indicated that the effects of exposure were strongest among children with blood lead levels below 10 μg/dL (Elliott et al. 2015). Evens et al. (2015) also found a nonlinear relationship between blood lead levels and test scores (see also Miranda et al. 2007). Using group-level data on the test scores of Massachusetts schoolchildren from 2001 to 2009, Reyes (2015b) reports that reductions in the share of children with elevated blood lead levels reduced the share of children who scored below satisfactory.

Aizer et al. (2017) studied how blood lead, even at low levels, affected children's test scores in Rhode Island. Because the authors had multiple measures of blood lead, which is often measured with error, they used one measure as an instrument for the other to reduce measurement error in their estimates. They also took advantage of the fact that the state of Rhode Island introduced a policy requiring landlords to certify that they had minimized lead hazards on their properties. The authors obtained data on all certificates issued between 1997 and 2010 and used these data to construct a predicted measure of whether a child grew up in a house with a lead-safe certificate. Using multiple measures of blood lead and predicted lead-safe certificates as instrumental variables, they find that reductions in blood lead improved students' reading and math scores. The fact that the largest improvements to children's test scores are for lead exposure reductions occurring under the former threshold of 10 μg/dL shows that the reductions in blood lead in the late twentieth century, while laudable, are still insufficient.

Other scholars have collected information about lead exposure from unusual sources. Using data on the pH of city water as a proxy for lead exposure, Ferrie et al. (2012) show that army enlistees who lived in cities with acidic water in 1930 scored comparatively low on the Army General Classification Test. Grönqvist et al. (2017) use moss samples from Sweden to measure lead in the atmosphere. They find that declines in atmospheric lead increased children's probability of completing high school and reduced their likelihood of being convicted of a crime. In sum, there is abundant evidence across time and place that lead exposure interferes with children's cognitive development.

Health

At high levels, lead exposure can lead to severe symptoms, such as convulsions, mental illness, and renal failure (Clay et al. 2014, Needleman 2004). Using data on US cities in the early twentieth century, Troesken (2008) and Clay et al. (2014) find that cities that used lead pipes and had acidic water had considerably higher infant mortality rates than comparable cities. Early research on lead exposure focused primarily on these extreme consequences of lead poisoning (Troesken 2006).

In recent years, scholars have begun studying the effects of lower levels of lead exposure on mental and physical health. One study found that girls with high blood lead levels in an Australian lead smelting community experienced more symptoms of anxiety and depression (Burns et al. 1999). The same was true of adolescents in a representative sample of children born in Chicago in the 1990s (Winter & Sampson 2017). Cumulative occupational (Lindgren et al. 1999) and environmental (Rhodes et al. 2003) exposure, measured by working-lifetime blood or bone lead levels, is also correlated with symptoms of general distress, depression, and anxiety among men.

Medical theories link immunotoxicity, to which lead contributes, to childhood asthma and obesity (Dietert 2014). But empirical work on the relationship between lead exposure and body mass

index (BMI) has yielded mixed results. Ballew et al. (1999) found no relationship and Scinicariello et al. (2013) found a negative relationship between blood lead levels and BMI in cross-sectional, nationally representative samples. Newer studies with stronger designs, in contrast, have documented positive relationships between perinatal exposure and BMI in the first two years of life (Kim et al. 2017), and between childhood blood lead levels and adolescent BMI (Winter & Sampson 2017). Finally, based on a review of the literature, Navas-Acien et al. (2007) conclude that there is a causal relationship between adults' cumulative lead exposure and hypertension. Overall, there is growing evidence that even low levels of lead exposure can undermine the physical and mental health of children and adults, but more studies using representative, longitudinal samples and credible identification strategies would provide a stronger foundation for this claim.

Impulsivity

A large body of research has shown that children exposed to lead are more likely than comparable children to suffer from a range of behavioral problems related to impulsivity and attention deficit hyperactivity disorder (ADHD) (Marcus et al. 2010). Studies have consistently documented a cross-sectional relationship between lead exposure and hyperactivity (Cho et al. 2010, Goodlad et al. 2013) in both clinical (Mendelsohn et al. 1998, Nigg et al. 2010) and nationally representative samples (Braun et al. 2006, Froehlich et al. 2009).

Recent research provides stronger causal evidence. Nigg et al. (2016) show that a genetic mutation moderates the effect of blood lead on symptoms of ADHD among children. Winter & Sampson (2017) report that, conditional on a rich set of individual and neighborhood covariates, children with comparatively high levels of blood lead were more likely to behave impulsively in adolescence, according to their caregivers. They find similar results when they restrict their sample to children who could be matched on their observed characteristics and when they use the distance of children's homes from the nearest lead smelting plant as an instrumental variable. Reyes (2015a) takes advantage of state-specific reductions in gasoline lead due to the Clean Air Act to estimate the effect of lead exposure on an array of childhood and adolescent behavioral problems. She concludes that childhood lead exposure increased behavioral problems among children, pregnancy among teenagers, and alcohol use among adolescents.

Aggression and Crime

Given lead's effects on cognition and behavior, many studies have considered whether it also influences aggression, violence, and crime. One of the earliest studies of lead exposure noted that lead-poisoned children were often impulsive and violent (Byers & Lord 1943). A later influential study by Needleman et al. (1996) found that children with high levels of lead in their bones had higher rates of reported aggression and delinquency (see also Needleman et al. 2002). Other studies document a positive relationship between lead and delinquency among socioeconomically disadvantaged children (Denno 1990, Sciarillo et al. 1992, Wasserman et al. 1998), socioeconomically advantaged children (Bellinger et al. 1994), and among children with similar IQ test scores (Chen et al. 2007). Lead exposure and crime rates are also positively correlated across neighborhoods (Boutwell et al. 2016), community areas (Barrett 2017), cities (Mielke & Zahran 2012), suburbs (Taylor et al. 2016), counties (Stretesky & Lynch 2001, Stretesky & Lynch 2004), states (Reyes 2007), and countries (Nevin 2007).

Like early research on test scores, most studies of lead and delinquency use cross-sectional data, but more recent scholarship has drawn on longitudinal cohort samples or panel data. These studies document a positive correlation between lead exposure and various indicators of delinquency

(Amato et al. 2013, Dietrich et al. 2001, Reyes 2015a, Wright et al. 2008). One recent study based on a 1972 birth cohort in Dunedin, New Zealand, with lead levels ranging from 4 to 31 μg/dL, reports that lead exposure in childhood, measured at age 11 years, had a weak relationship with official criminal convictions and self-reported offending from ages 15 to 38 years (Beckley et al. 2018).[7]

Four recent studies use different sources of exogenous variation in lead exposure to identify its effect on crime. Feigenbaum & Muller (2016) use several strategies to isolate the effect of lead exposure in US cities in the late nineteenth and early twentieth centuries, including instrumenting a city's use of lead pipes with its distance from the nearest lead refinery and comparing cities with more or less acidic water. They find that cities that used lead pipes in the late nineteenth century had higher homicide rates in the early twentieth century than cities that used iron pipes. Aizer & Currie (2017) collect data on children's blood lead, home addresses, school suspensions, and juvenile detention in Rhode Island. Exploiting the fact that reductions in gasoline lead from 1990 to 2004 had the largest effect on children living near roads, they find that declines in lead exposure in Rhode Island considerably reduced both school suspensions and the probability that boys were detained as juveniles. Billings & Schnepel (2018) study the effects of a CDC-recommended intervention for children exposed to lead. The intervention includes education for caregivers and, as applicable, home environment investigations and referrals to lead remediation services.[8] The authors note that in order to receive the intervention, children needed to have two consecutive tests reporting blood lead levels above 10 μg/dL.[9] They find that children above the threshold for intervention (whose second test was above 10 μg/dL) were less likely to be suspended from school and arrested for a violent crime than children just below the intervention threshold (whose second test was between 5 μg/dL and 10 μg/dL). Sampson & Winter (2018) instrument children's blood lead using the distance of their home from the nearest smelting plant. They find that children's rates of delinquency, as reported by their parents, increased with their blood lead level, but that their rates of official arrest did not.

Whether lead exposure has any effect on crime is a different question than whether its decline was an important cause of the national crime decline in the late twentieth century. Scholars should take care to distinguish these questions. Beyond providing an estimate of the effect of lead exposure on crime, Reyes (2007) estimates how much that effect contributed to the crime drop. Based on her baseline estimates of the effect of lead exposure on violent crime, she concludes that the phase-out of lead "was responsible for approximately a 56% decline in violent crime" (Reyes 2007, p. 2). Nevin (2007) also argues that the reduction in lead exposure in the 1970s was responsible for a significant portion of the drop in crime in the 1990s. Several scholars have challenged these conclusions. Lauritsen et al. (2016), for instance, show that while a lagged national time series of gasoline lead consumption is strongly correlated with the national time series in serious violence, as recorded in the Uniform Crime Reports (UCR), it is weakly correlated with national trends in homicide and serious violence, rape, robbery, and aggravated assault, as recorded in the National Crime Victimization Survey (NCVS). There are several possible explanations for the weakness of these latter correlations. One is that crime has many causes: If causes other than lead exposure pushed national crime rates up at a time when lead exposure was relatively low, the national correlation between gasoline lead consumption and crime would be weak, even if the two are

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[7]In a commentary on this study, Farrington (2018) argues that while imprecise, the magnitude of the association between lead and delinquency is similar to or larger than other major predictors.

[8]Billings and Schnepel note that the intervention could work by reducing children's blood lead levels or by improving children's health without reducing their blood lead levels. Although they could not separately identify these two mechanisms, they report evidence suggesting that both were important.

[9]Second blood lead tests above 15 μg/dL or 20 μg/dL could trigger more intensive interventions.

causally related. Another is that the correlation between lead exposure and the NCVS measures of crime was stronger in the late years of the series than in the early years. Because there were several overlapping sources of lead in these early years—water and paint, in addition to gasoline, for instance—the national series of gasoline lead consumption could understate the total prevalence of lead in the mid-twentieth century.

Other scholars have pointed out that if the deleading of gasoline was a major cause of the crime drop, it should primarily have affected cohorts born after the policy change. Cook & Laub (2002) suggest instead that the rise and fall of homicide in the late twentieth century followed a similar pattern for all cohorts. Kim et al. (2016), in contrast, conclude that the drop in the felony arrest rate in New York State was mostly due to the decline in arrests among birth cohorts born after the 1970s. Another paper uses age-period-cohort models to study the relationship between blood lead levels and age-specific UCR homicide arrest rates (McCall & Land 2004). It finds no strong correlation between these measures of lead exposure and violent crime.

Aizer & Currie (2017, p. 35), finally, find evidence consistent with Reyes's (2007) original estimates. They conclude that "reductions in blood lead levels may have been responsible for a significant part of the observed decline in antisocial behavior among youths and young adults in recent decades." Because lead exposure is only one among many possible causes of crime documented by social scientists, assessing whether the elimination of gasoline lead was an important cause relative to the other causes of the crime decline (Sharkey 2018) is an important area for future work. Collecting multiple measures of crime—both official and self-reported—and data on lead exposure from paint, smelting plants, and water pipes in addition to gasoline will be especially important in this effort.

SOCIAL-ENVIRONMENTAL INTERACTIONS

Lead exposure is socially stratified. This fact alone makes it an appropriate object of sociological analysis. But there is also evidence that both the effects of lead exposure on child development and the effects of child development on adult well-being vary with children's social environments (Morello-Frosch et al. 2011, Troesken 2006). We depict these social-environmental interactions using blue dashed arrows in **Figure 1**. Because lead exposure is unequally distributed and unevenly consequential, it has the capacity to generate further stratification by reproducing inequality between both individuals and neighborhoods.

Even if the homes of poor and rich children contained similar amounts of lead paint, children who spend more time indoors to avoid neighborhood violence would face higher rates of exposure (Newkirk 2017). Violent neighborhoods, moreover, not only keep children inside but also make their daily lives more stressful (Western 2015). Violence and other sources of neighborhood or household stress might increase children's susceptibility to lead's effects (Sharkey 2010, Sharkey & Sampson 2015). For example, Gump et al. (2008) show that pre- and postnatal lead exposure was associated with increased cortisol levels following an acute stressor in an experimental setting involving children around 10 years old. Aizer et al. (2017) find that the effects of the decline in lead exposure in Rhode Island were largest among the most disadvantaged children. Still, because low-income black and Latino children bear multiple burdens, the observed marginal effect of lead exposure could be smaller for them than it is for middle- and high-income white children. Evens et al. (2015), for instance, find that lead exposure had a larger effect on the test scores of white children than black or Latino children in Chicago.

The same behavior can also have different consequences depending on children's social environment (Sampson 2016). Children who grow up in supportive environments with ready access to resources may score comparatively low on tests if they are exposed to lead, but they are unlikely

to follow a developmental trajectory leading to severe social deprivation. And whereas in some settings a child's impulsivity may be muted or even rewarded, in others it may be punished or met with violence (Collins 2008, Western 2015).

As we illustrate in **Figure 1**, lead's effects, even if initially small in magnitude, can have long-term consequences for development and ultimately socioeconomic attainment. In a representative New Zealand birth cohort, for instance, childhood lead exposure was associated not only with lower IQ test scores and socioeconomic status in adulthood but also with downward social mobility (Reuben et al. 2017). Lead can also be transmitted directly from parents to children: It is stored in bones and mobilized in blood "at times of high bone resorption (e.g., during pregnancy, aging, postmenopause)" (Hu et al. 2007, p. 456).

We suspect that social-environmental interactions will become an increasingly important area of sociological research more generally. Stressful social environments can exacerbate the social consequences of pollutants of many kinds (Underwood 2017). For example, Alshire et al. (2017) found that the association between air pollution and cognitive errors was stronger among adults aged 55 and older living in high stress neighborhoods. "Those living in socioeconomically disadvantaged neighborhoods, where social stressors and environmental hazards are more common," they conclude, "may be particularly susceptible to adverse health effects of social and physical environmental exposures" (Alshire et al. 2017, p. 56).

DISCUSSION

In this review, we have emphasized the importance of studying the causes as well as the effects of lead exposure and other environmental toxins. We argue that these causes are fundamentally social. This means that they require social solutions.

A fundamental cause of lead exposure and other environmental toxins is a weak regulatory environment that favors the interests and profits of business over the health of the public. This pathway is represented in **Figure 1**. As we write this article, radical changes are afoot in EPA funding, the appointment of key administrators in regulatory agencies, and the study, regulation, and testing of toxins. The EPA has proposed deep cuts in the testing of children for lead exposure (Mooney & Eilperin 2017) and dozens of environmental laws have either been rescinded or been targeted for rollback (Popovich et al. 2018). In August of 2017, the Department of the Interior abruptly directed the National Academy of Sciences to "cease all work" on a study of the health effects of a common mining technique believed to deposit waste containing toxic minerals in ground waters (Wallace et al. 2017). More generally, the deep ties of top regulators of the environment in the Trump administration with conflicting business interests appear to be unprecedented (Ivory & Faturechi 2017). It is no exaggeration to say that political manipulation of the regulation and testing of toxins poses a direct threat to America's health. But a legal framework for eliminating lead does exist (Markowitz & Rosner 2013) and is currently being pursued in California (Rosner 2014). The most promising place to pursue removal and regulation may be the courts (Friedmandec 2017).

Residential segregation, concentrated poverty, discrimination in housing markets, neighborhood disinvestment, and a limited array of options for tenants seeking to remove lead from their environments are also fundamental causes of exposure to lead and other toxins. In **Figure 1**, we group these causes under the heading "concentrated disadvantage." Reyes (2007) proposed that environmental policy can be considered social policy, but these causes suggest that the reverse is true as well. By investing in neighborhoods and improving access to safe housing for all residents, we can reduce exposure to lead and other toxins.

In the meantime, community organizing and smaller interventions can have an impact (Brulle & Pellow 2006). Scientists at the University of Pennsylvania are collaborating with local community

groups to reduce lead exposure in Philadelphia's disadvantaged neighborhoods (Schein 2017). "Communities that are politically organizing around [lead accountability]," argues historian David Rosner, "are going to be the ones who really make the change" (quoted in Cabrera 2017). Aizer et al. (2017) show that a program in Rhode Island requiring landlords to mitigate lead hazards on their properties significantly reduced children's blood lead levels and, as a result, markedly improved their later test scores. For children already exposed, Billings & Schnepel (2018) find that CDC-recommended interventions can largely eliminate the negative effects of lead exposure on school suspensions and arrests. Community organizing that mobilizes legal and health-related interventions and collective efficacy (Sampson et al. 1997) are important mechanisms for reducing toxic exposures. Crucial to these efforts, the public's perception of toxic risk, or lack thereof (Auyero & Swistun 2008), and the social forces that shape these perceptions are essential areas for further research.

All three causes of lead exposure depicted in **Figure 1** are important because even small changes early in childhood can cascade and cumulate over the life course, generating substantial long-term effects. Research on allostatic load and stress, in particular, has demonstrated the power of early toxic exposures to undermine life chances (McEwen & McEwen 2017) and reproduce inequality across generations. Lead removal is no panacea—and scholars should resist the conclusion that lead exposure is the sole cause of any of the outcomes we discuss here—but the evidence we have reviewed suggests that lead removal would have positive effects on many domains of life. As recent assessments have shown, the benefits far outweigh the costs (Health Impact Proj. 2017).

As public awareness about the continuing dangers of lead exposure has grown, so have sources of data with precise geographical coordinates.[10] Sociologists should draw on these data to study both the causes and effects of lead exposure, as conceptualized in **Figure 1**. Qualitative research among landlords, regulators, and policymakers would illuminate why exposure to lead and other environmental toxins persists and how it might ultimately be eliminated.

Finally, the growing body of scholarship on lead's social causes and consequences raises questions even more fundamental than how best to reduce or eliminate exposure. Much of the political debate over American social provision takes for granted "oppositions between the morally 'deserving' and the less deserving" (Skocpol 1995, p. 149). But when traits such as impulse control and judgment are affected by environmental toxins, it becomes especially difficult to divide social adversities into those that are deserved and undeserved (Currie 2011). What we call character, Rawls (1999, p. 89) observed, "depends in good part upon fortunate family and social circumstances early in life for which we can claim no credit." Empirical support for Rawls's claim has accumulated in recent years. There is now abundant evidence that some cohorts, "simply as a function of when they were born, have been exposed as children to severely disadvantaged contexts (e.g., violence, crack epidemic, lead toxicity, extreme racial and poverty segregation; labor market precariousness of caretakers) that influence character development" (Sampson 2016, p. 507). Addressing the problem of lead exposure entails turning our attention away from explanations based on character deficiencies and toward durable investments (Sharkey 2013) that will undo the damage wrought by the lead industry.

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[10]The Toxics Release Inventory Program of the EPA currently provides detailed geocoded and time-specific data on toxic releases, brownfields, toxic cleanup sites, and more (see also Eckel et al. 2001). Since the crisis in Flint, many American cities have started to provide data on lead piping infrastructure and water quality. For example, household-level exposure to lead piping is now available for Washington, D.C. (**https://geo.dcwater.com/Lead/**). Temporally and geographically refined data on toxic exposure and its multiple sources present new opportunities for sociological research on environmental inequality.

Uniting Science and the Common Good

The crisis in Flint highlighted the dangers of perverse incentives in science and government. But it also provided a powerful example of collective efficacy in action, showing how scientists and local communities can collaborate to uncover a public health emergency and prevent it from getting worse (Brown 2013, Corburn 2005, Edwards & Roy 2016, Markowitz & Rosner 2013, Rosner & Markowitz 2016). Chicago, a city long recognized for its marked racial inequality, also demonstrated how change can happen: Persistent testing and regulatory enforcement by the city's health department over the past 20 years helped to reduce children's lead levels dramatically (Sampson & Winter 2016). A universal program, this public health effort disproportionately benefited African American children. Sustained collaboration that spans geographic, community, and political networks will be needed to not only eliminate exposure to lead but also to solve other environmental problems (Bodin 2017), such as widespread air pollution (Zivin & Neidell 2018) and toxic yet "less visible, *relict*" sites of accumulated industrial contamination (Elliott & Frickel 2013, p. 539), whose impact is socially stratified at both local and global scales. Sociologists should look to these and other examples of partnerships between scholars and the general public for lessons in how to pursue independent science for the common good.

DISCLOSURE STATEMENT

AUTHOR CONTRIBUTIONS

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