



Legacies of inequality, legacy lead exposures, and improving population well-being

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The findings in McFarland et al. (1) are at once disturbing and reassuring. The bad news is captured in their title: "Half of US population exposed to adverse lead levels in early childhood." That striking figure, based on a welldesigned and rigorous analysis, will make readers take note, as well it should. We have known for some time now the deleterious impact of lead exposure on a wide range of features in young children's development, including the disruption of multiple organ systems, cognitive deficits, emotional dysregulation, and impaired self-control. The authors also note the convergent evidence on what they call "legacy lead exposures" which are revealed in multiple outcomes later in life such as educational attainment, income mobility, delinquent behavior, and physical health. The bundle of adversities associated with childhood lead exposure, even if individually subtle, cumulate over time and can generate long-term effects (2-4).

How can there be grounds for optimism in a study showing that half the current population of adults has been exposed to the detrimental effects of lead in childhood? The authors' (1) birth cohort design and population projections throughout the 21st century provide an answer. We know that leaded gasoline declined substantially in the United States starting in the mid-1970s and that there have been subsequent declines in children's levels of lead. But the innovation of the McFarland et al. study is to estimate cohortspecific blood-lead levels (BLLs) by age in 2015 based on the National Health and Nutrition Examination Survey (NHANES), a representative sample of US children. Constructing serial cross-sectional measures of BLLs across four decades of the NHANES merged with census and mortality data, the authors provide past, present, and estimated future trends in lead exposure, along with consequences for cognitive ability. Although levels of childhood lead exposure among current adults are alarmingly high, and the authors estimate considerable population losses over time in cognitive ability, the trends in lead exposure are nonetheless in the right direction. The decline across successive cohorts is so steep that nearly all of today's children have BLLs less than 5 µg/ dL, traditionally considered to be low even though no lead level is now considered safe. McFarland et al. project that, as we reach 2100, nearly all adults will have experienced leadfree childhoods if 2015 exposure levels hold. This means that the trajectory of the United States is on the right path, at least with respect to the fading of prior lead exposures among adult populations. That is good news indeed.

Poisoned Development

Still, legacy lead exposures live on, with cumulative and manifold consequences for children's development, many of which have yet to fully express themselves. While there is good news, then, it is qualified. We need a fuller accounting of the sources and consequences of lead exposure for other forms of cognitive functioning, physical health, problem behavior, and other aging outcomes over multiple cohorts and in the life course of individuals.

As the authors (1) argue, understanding disparities in lead exposure across time—particularly Black/White disparities—is an additional next step for legacy lead-related research. In the NHANES data, most Black adults now under age 45 y experienced much higher levels of BLLs in early life than White adults. This racial inequity is consistent with prior research and longstanding patterns of environmental hazards in communities of color (5).

Another need is to extend analyses of other sources of legacy lead exposures, including those that still pose a threat to children today. Because of missing BLL data prior to the NHANES, the estimates in McFarland et al. (1) of lead exposure from 1940 to the 1970s are based on predictions from trends in leaded gasoline. These are likely to be underestimates because lead exposure, historically and today, also comes from leaded paint in older homes, lead plumbing (6, 7), brownfields, and hazardous industrial plants. In Flint, MI, lead in the water was the culprit, and, in many cities, smelter plants, even if now defunct, spewed lead particles that remain hidden in the soil. These are sites unseen (8), or what have been called ghosts of polluters past (9). In Chicago, for example, children's average BLLs in the mid-1990s were higher in neighborhoods that contained or were adjacent to lead smelting plants, many of which were relics (10). In 2016, a year after the last estimates of BLLs in McFarland et al., a neighborhood in East Chicago, IN-Black and poor-was evacuated because of dangerous levels of lead in children, attributed to the contaminated soil from a shuttered smelter plant nearby (11). And a recent investigation of Santa Ana, CA, not typically thought of as an industrial city, found that current lead levels in the soil are elevated in poor Latino neighborhoods characterized by recycling plants and an industrial past, literally on the other side of the railroad tracks from more advantaged neighborhoods (9). These findings are consistent with a tradition of research

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showing that poor neighborhoods of color are ecologically distinct and suffer disproportionately from "toxic inequality," denoting the spatial isolation of communities proximate to hazardous industries, brownfields, and the prevalence of lead and other chemical exposures (10).

The fact that hazardous industrial plants are linked to heightened lead levels means that the projection of an effectively lead-free environment for adults of the future is not likely to hold in the developing world (12) and in advanced economies with powerful growth mandates coupled with weak environmental regulations. Cohort-based projections of adult exposure for multiple countries could provide needed information for where to target global population interventions to mitigate past exposure and where to strengthen regulatory policies on industrial lead emissions.

Analyses of environmental hazards other than lead would likewise benefit from cohort-based designs like the present study. Air pollution exposure, for example, follows some of the same contours of inequality that lead does (13), and with similar long-term negative effects on cognitive and human capital development (14, 15). Combined with legacy lead exposures and other hazardous chemicals, toxic inequality will likely remain a risk to societal well-being, both in the United States and globally.

(In)Equality by Design

Fig. 1 presents a conceptual model for the sources and consequences of lead exposure and other toxins that can serve as a potential guide for further research and the empirical challenges it faces. One implication of this model is that the burdens of lead exposure are unequally shared because of residential racial segregation, concentrated poverty, discrimination in housing markets, and neighborhood disinvestments (grouped under "concentrated disadvantage"). Another implication is that, because lead exposure is stratified, as are its consequences, it has the capacity to generate further stratification by reproducing inequality between both individuals and neighborhoods, and across generations. Lead toxicity is therefore a pathway through which environmental inequality literally gets into the mind and body, with both individual and social consequences. Its eradication is a central component of tackling broader racial and other social inequalities in human development.

Lead is still present in water, paint, and soil, in part, because of historically weak regulatory environments, which, over time, further harm racially segregated and poor communities the most. Poor families alone lack the resources and organizational access to eliminate environmental toxins from their homes and communities, begging the question of why government soil remediation, code enforcement, and housing reinvestment efforts are not more widespread. For example, the remediation of lead paint in rental properties is something that can be influenced by city governments, should they choose to do so. Aizer et al. (16) showed that a program in Rhode Island requiring landlords to mitigate lead hazards on their properties significantly reduced children's BLLs and, as a result, markedly improved their later test scores. By investing in disadvantaged neighborhoods, improving access to safe housing for all residents, and increasing regulatory controls, we can thus reduce exposure to lead and other toxins by policy redesign. Community organizing for environmental health and locally based interventions can also have an impact (4, 9, 17). All three social causes of lead exposure in Fig. 1 are important because even small changes early in childhood can cascade and cumulate over the life course, generating substantial long-term effects.

Finally, an integrated life course, cohort, and population framework highlights the fundamental causes of wellbeing. As a function of when and where they were born, children have been differentially exposed to poisonous environments, not just from lead but also from violence and extreme forms of racial and economic segregation, all of which influence the quality of children's development. Accordingly, the nurturing of human capital and capacity is very much a function of growing up in favorable circumstances and times, motivating a more expansive conception of the sources of individual development and character (15, 18). Environmental hazards are historically variable and subject to policy choices, motivating proactive investments and regulations that strive to mitigate the damage wrought by past toxic inequalities and create more equitable futures.

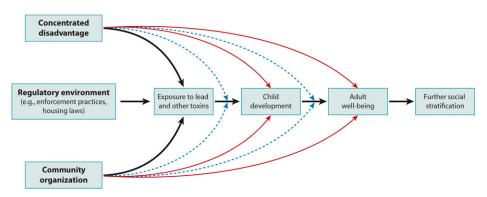


Fig. 1. A model of inequality, environmental toxicity, and well-being over the life course. Thick black arrows represent the causal effects of main interest. Thin red arrows represent the direct effects of concentrated disadvantage and community organization on child development and adult well-being. 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 not highlighted. Image credit: Reprinted with permission from *Annual Review of Sociology*, volume 44; copyright 2018 Annual Reviews, https://www.annualreviews.org/.

- M. J. McFarland, M. E. Hauer, A. Reuben, Half of US population exposed to adverse lead levels in early childhood. Proc. Natl. Acad. Sci. U.S.A. 119, 10.1073/pnas.2118631119 (2022). D. C. Bellinger, Childhood lead exposure and adult outcomes. JAMA 317, 1219–1220 (2017). 1.
- 2
- A. Reuben et al., Association of childhood blood lead levels with cognitive function and socioeconomic status at age 38 years and with IQ change and socioeconomic mobility between childhood and adulthood. 3. JAMA 317, 1244-1251 (2017).
- C. Muller, R. J. Sampson, A. Winter, Environmental inequality: The social causes and consequences of lead exposure. Annu. Rev. Sociol. 44, 263-282 (2018). 4
- R. D. Bullard, Handbook of Black American Health, I. L. Livingston, Ed. (Greenwood Press, Westport, CT, 1994). 5.
- 6. J. J. Feigenbaum, C. Muller, Lead exposure and violent crime in the early twentieth century. Explor. Econ. Hist. 62, 51-86 (2016).
- 7. J. M. Gibson et al., Early life lead exposure from private well water increases juvenile delinquency risk among US teens. Proc. Natl. Acad. Sci. U.S.A. 119, e2110694119 (2022).
- 8. S. Frickel, J. R. Elliott, Sites Unseen: Uncovering Hidden Hazards in American Cities (Russell Sage Foundation, New York, NY, 2018).
- Y. Cabrera, Ghosts of polluters past. Grist (2022). https://grist.org/project/equity/california-lead-soil/. Accessed 23 February 2022. 9.
- 10. R. J. Sampson, A. Winter, The racial ecology of lead poisoning: Toxic inequality in Chicago neighborhoods, 1995-2013. Du Bois Rev. 13, 261-283 (2016).
- 11. A. Goodnough, Their soil toxic, 1,100 Indiana residents scramble to find new homes. NY Times, 30 August 2016, Section A, p. 31. 12. B. Ericson et al., Blood lead levels in low-income and middle-income countries: A systematic review. Lancet Planet. Health 5, e145-e153 (2021).
- 13. N. Kravitz-Wirtz, K. Crowder, A. Hajat, V. Sass, The long-term dynamics of racial/ethnic inequality in neighborhood air pollution exposure, 1990-2009. Du Bois Rev. 13, 237-259 (2016).
- 14. R. Manduca, R. J. Sampson, Childhood exposure to polluted neighborhood environments and intergenerational income mobility, teenage birth, and incarceration in the USA. Popul. Environ. 42, 501–523 (2021). 15. J. G. Zivin, M. Neidell, Air pollution's hidden impacts. Science 359, 39-40 (2018).
- 16. A. Aizer, J. Currie, P. Simon, P. Vivier, Do low levels of blood lead reduce children's future test scores? Am. Econ. J. Appl. Econ. 10, 307-341 (2018).
- 17. R. J. Brulle, D. N. Pellow, Environmental justice: Human health and environmental inequalities. Annu. Rev. Public Health 27, 103-124 (2006).
- 18. R. J. Sampson, L. A. Smith, Rethinking criminal propensity and character: Cohort inequalities and the power of social change. Crime Justice 50, 13-74 (2021).