Inequitable Chronic Lead Exposure

A Dual Legacy of Social and Environmental Injustice

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Both historic and contemporary factors contribute to the current unequal distribution of lead in urban environments and the disproportionate impact lead exposure has on the health and well-being of low-income minority communities. We consider the enduring impact of lead through the lens of environmental justice, taking into account well-documented geographic concentrations of lead, legacy sources that produce chronic exposures, and intergenerational transfers of risk. We discuss the most promising type of public health action to address inequitable lead exposure and uptake: primordial prevention efforts that address the most fundamental causes of diseases by intervening in structural and systemic inequalities.

Key words: environmental justice, health inequity, lead exposure, racial inequity

ENVIRONMENTAL TOXINS continue to disproportionately affect minority populations within the United States, constituting a lingering significant public health issue. Childhood lead toxicity is of particular concern. While the full spectrum of toxicological effects of lead in the human system deserves further study, we know that the persistent presence of lead in women and children is a public health issue of a first order.[1] Recent media attention to the Freddie Gray case (a 25-year-old black man from Baltimore who died in police custody) revealed his history of severe lead poisoning as a child[2] and has drawn public attention to the wide-reaching effects of childhood lead exposure, including its potential contributions to disparities in problem behavior among adolescents and young adults. This public concern is buttressed by emerging empiric evidence of elevated criminal activity among those who were lead poisoned as children.[3-6] When paired with our knowledge of the negative educational and developmental outcomes associated with lead exposure,[7-9] the disproportionately high rates of lead toxicity in minority populations represent a prime social and environmental justice issue.

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A range of both historic and contemporary factors contributes to the current unequal distributions of lead in the urban environment. However, in practice, the issue of inequities in childhood lead toxicity continues to be treated primarily as a problem facing families and individuals living in houses with lead paint. The extant literature tells us that the issue is much broader. Here we explore 4 key considerations to addressing the inequitable lead exposure in urban areas as an environmental justice and structural inequity issue, with the goal of nudging practitioners to recognize the clustering of lead exposure as much more than the clustering of houses with lead paint. To fully investigate the issue in terms of equity, we consider (1) the geographic clustering of lead toxicity, (2) legacy sources of lead in the environment, (3) intergenerational transfers of lead exposure risk, and (4) primordial prevention from an environmental justice perspective.

BACKGROUND ON CHILDHOOD LEAD TOXICITY, HEALTH, AND WELL-BEING

There is no known "safe" level of lead exposure below which health effects do not occur,[10,11] however, the Centers for Disease Control and Prevention currently recommends that actions are taken for children with blood lead levels (BLLs) at or above 5 μg/dL.[11] Children in approximately 4 million households in the United States are being exposed to high levels of lead. In addition, of those children exposed to lead, half a million children younger than 6 years have BLLs above the Centers for Disease Control and Prevention recommendations.[11] Lead exposure often occurs with little or no distinct symptoms, particularly in lower-level chronic exposure, manifesting only once irreparable damage has been done.[11] Finally, while acute symptoms of exposure can be treated, *there is no remedy for the underlying damage* that has been done physiologically; the damage is largely irreversible.[10,12]

The effects of childhood lead toxicity can vary according to an individual's age and the level to which one is exposed.[10] The health effects influence nearly every organ system.[12] Consequences for the neurologic system alone—that is, impact on IQ, behavior, and educational performance[13,14]—alter the lifetime trajectories of exposed children and require substantial remedial education.[11] Research has shown a potential link between childhood lead poisoning and aggressive behavior, as well as an increase in criminal activity in adulthood.[3-6] One study looked at the association of air-lead levels and crime rates in numerous counties throughout the United States.[3] The results of this study indicate that elevated air-lead levels are associated with higher rates of both property and violent crimes and that this association is most prominent in areas lacking resources for the prevention and treatment of lead exposure.[3]

The negative outcomes associated with lead toxicity are not limited to childhood. The best measure of long-term effects of childhood lead toxicity is based on lead levels in bone, as bone is the primary site of lead storage in the body. Studies report that higher lead levels found in the teeth of young adults (around 18 years of age) are associated with school failure and impaired motor functioning.[15,16] Others indicate that levels of lead in bone are associated with hypertension and impaired kidney function in adolescents and adults.[17-19]

GEOGRAPHIC INEQUITY IN THE DISTRIBUTION OF LEAD TOXICITY

Given the potential negative health and social outcomes, the failure to address chronic lead exposure raises concerns about social justice and the long-term health of urban children living in environmentally disadvantaged neighborhoods. Lead poisoning in America today involves clear and substantial issues of geographic and generational health inequity. The likelihood of exposure to contaminants and the health consequences of that exposure are burdens disproportionately shouldered by racially and socioeconomically disadvantaged communities.

Housing disparities, in particular, play a major role in the disproportionality of lead exposure, as residence in substandard housing is unequally distributed in the US population.[20-22] This problem is compounded by a shortage of affordable housing, with an "estimated 9 million low-income renters (who) must compete for only 3 million available and affordable units."[21(p6)] Federal law, Residential Lead-Based Paint Hazard Reduction Act, requires property owners to disclose known sources of lead exposure including lead-based paint but does not require them to remediate the source. Low-income individuals may not have the resources to find alternative housing. Specifically, the 2005 American Housing Survey revealed that 7.5% of non-Hispanic blacks reside in moderately substandard housing and 2.9% reside in severely substandard housing.[23] In comparison, 2.8% of non-Hispanic whites reside in moderately substandard housing and 1.6% in severely substandard housing.[23,24] In other words, for every 1 white person living in substandard housing, there are more than 2 black persons living in substandard housing.

It follows that exposure to environmental toxins, such as lead, is geographically clustered as well.[25-27] Black children are 3 times more likely than white children to have dangerously elevated BLLs.[28] This raises concerns about social justice and the long-term health of these children. Between 1997 and 2001, more than 80% of all children in the United States with confirmed elevated BLLs were of a minority race or ethnicity, predominantly (60%) non-Hispanic black.[1] More recent surveillance demonstrates progress in lowering mean BLLs overall, yet racial and economic inequities persist.[29]

These racial differences in lead toxicity are not solely due to differences in housing. Socioeconomically disadvantaged populations tend to cluster geographically. In part, this clustering is the result of constrained choices available to those of low financial means, as well as it is due to the history of racial residential segregation in the United States.[30] Together, these mechanisms result in concentrations of marginalized populations living in neighborhoods with higher exposure to environmental toxins such as lead and less access to resources that directly mitigate the exposure (such as nutritious food and renovated infrastructures).[25,31] This lead exposure is due, in part, to housing inequities and also includes other systemic inequities that manifest in geographic clustering of environmental risks.

LEGACY SOURCES OF LEAD CONTAMINATION AND CHRONIC LEAD EXPOSURE

While American society as a whole contributed to the legacy of lead contamination in our communities, primarily through the use of leaded paint and gasoline, the current burden of adverse health effects is borne predominantly by low-income minority groups living in distinct geographic areas. Certain sources of lead that could contribute to *acute* lead poisoning have been the topic of substantial public health action, with focus on consumer product safety (eg, toys with lead-based paints) and seriously degraded lead-based paints in dilapidated homes.[32] Yet, there is a paucity of public health action to address chronic exposure resulting from

the legacy of lead in gasoline, pipes, and industrial manufacturing.

The legacy of leaded gasoline

In the 20th century, 2 new applications for the use of lead turned lead toxicity into a widespread problem throughout the United States. First, lead-based paints became standard use in new homes throughout the early part of the century. Second, lead additives for gasoline were developed as an antiknock engine formula in the 1920s, and the rapid growth of motor vehicle use in the middle part of the century was fueled by gasoline doped with tetraethyl lead. By the 1970s, Americans faced lead exposure from multiple sources.[33]

Overall, about 5 million metric tons of lead was deposited in the environment as a result of the combustion of leaded gasoline. Almost all of that lead was originally deposited near roadways. The spread of deposited lead then depended on the conditions of the depositional area. The impervious surfaces of busy streets that received more than 1 metric tons of lead per year[33] did not absorb the lead but, instead, contributed to continual runoff of lead down storm drains and from there directly into rivers. Instead, when lead was deposited on a grassy fringe (such as a front yard or park), the lead was effectively retained and eventually concentrated in the surface layer of soils.[34] Thus, surface soils became the repositories of lead deposited over decades—in the case of older roadways, the proximal soils retained almost all of the lead deposited on them over a period of about 60 years. Furthermore, lead from the combustion of leaded gasoline is preferentially enriched in the more readily windblown fine-size fraction of soils and so lead in dusts derived from urban soils is likely to be more potent and concentrated than in the bulk soils.[35]

The urban roadway example shows both the impact of the point source of lead deposition from leaded gasoline and the diffuse soil lead that blankets urban regions. In other words, even at distances away from the roadway beyond where direct lead deposition occurs (and far away from structures using lead-based paint), the background level for lead is significantly higher in urban areas (~500 ppm) than in suburban areas (~60 ppm).[36] Despite the increasing suburbanization of society (and even the suburbanization of poverty), it is well established that minority populations remain heavily overrepresented in urban versus suburban neighborhoods.[37] More specifically, studies have found that minority and high-poverty neighborhoods are overrepresented near high-volume roads (experiencing >2 times the level of traffic density compared with other communities).[38,39] All of this indicates that legacy sources of leaded gasoline could contribute to current racial inequities in chronic lead exposure.

The legacy of lead pipes and lead solder in plumbing

Despite the near elimination of the use of paint-related lead sources in the United States 40 years ago, and the phasing out of other potentially more harmful sources such as leaded gasoline and lead solder in plumbing phased out shortly after, the legacy of these sources remain imprinted into the fabric of urban cities in the form of soil and water contamination.[39] In addition to lead-based paint and residual lead in soil, a significant source of childhood lead exposure occurs through drinking water as a result of leaching from lead pipes, lead solder, or brass fixtures. Current estimates are that exposure through water contributes between 10% and 20% to children's BLLs, with the estimate dramatically increasing to 40% to 60% for infants who are formula-fed.[40] This is of particular concern as the breast-feeding rates are lowest among non-Hispanic black women and low-income populations,[41,42] the same populations that are particularly vulnerable to the other sources of lead exposure described earlier.

As early as the mid-19th century, US citizens raised health concerns about the use of water pipes made of, or lined with, lead and many areas implemented ordinances that prohibited to varying degrees the use of lead water pipes. In response, in 1928, the Lead Industry Association (LIA) was established. Rabin states, "The LIA's 1934 annual meeting minutes record an 'intensive' effort to reverse the downward trend in the use of lead pipes…."[39(p1586)] Despite concerns, many large urban centers continued to install lead pipes as they were more durable.[39] The use of lead in water pipes and solder was not restricted until the passage of the Safe Drinking Water Act Amendment of 1986,[39] although "lead-free" materials that include up to 8% lead can still be used.[43,44] Many large urban centers continued to install lead pipe into the 1980s, with the greatest use of lead lines in the Midwest and Northeast. Since the 1980s, lead contamination has been noted in a number of urban centers. Most notably, between 2001 and 2004 in Washington, District of Columbia, when water disinfection shifted from the use of chlorine disinfection to chloramine, a sampling of houses with water delivered through lead-lined pipes demonstrated that more than half of the houses had lead levels over the Environmental Protection Agency (EPA) action levels.[39]

The exact number of lead water pipes currently in use is not clear, with more than 30% of cities

surveyed not being able to state how much lead piping exists.[39] The existence of lead-lined water pipes in large urban centers is of great concern, especially to the more vulnerable populations that reside in urban cores where all of the legacy sources exist and compound the impact of chronic lead exposure.

The legacy of industrial sites

As detailed in an excellent series in *USA Today* called "Ghost Factories,"[45] many neighborhoods were built near industrial sites, for the ease of transport for factory workers—indeed, many of these "factory neighborhoods" thrived with the worker's income supporting markets, restaurants, and myriad retail establishments. Sometimes, this colocation of industry and community had some negative effects, including, of course, the emission of harmful products into the air, water, and soil and subsequent human exposure. Although the inequitable colocation of hazardous waste treatment and dumping was at the core of the original investigations of environmental racism,[46] the historical siting of polluting industries has not been salient in discussions of inequitable lead exposure. In some instances, original zoning decisions were made in the early 1900s, with an explicit intent to support both segregation and the siting of industrial facilities in black communities. The very practical decision to site new facilities within existing industrial zones has perpetuated the concentration of polluting facilities in minority communities.[47,48]

Mohai and colleagues[49] explain that information from a conference led by sociologists at the University of Michigan brought these issues to the attention of the EPA, which established an Office of Environmental Equity. However, progress stalled when the EPA tried to rely upon Title V Provisions in the Civil Rights Act to address these historic legacies resulting in inequities. In the words of Mohai and colleagues, the policy proposed that agencies:

> needed only to show that an action by industry or government with regard to a polluting facility would lead to a disparate outcome rather than show that an action was motivated by an intent to discriminate. However, the effectiveness of applying Title VI to future environmental justice cases was later cast in doubt … and the general strategy of using legal actions to achieve justice in cases of environmental inequality has not fared well.[49(p410)]

Further complicating the issues is the fact that previous industrial facilities of many of these contaminating industries are often no longer present and the only way to know where these "Ghost Factories"[45] were located, or the type of facilities they were, is through old property records. Or, they could be stumbled upon when performing soil or water testing.[45] As many of these sites were poorly "closed" after operations ceased, they continue to be sources of fugitive dust contamination in urban areas.[50]

Original "point" sources of lead from paint, gasoline, plumbing, and industries are now widely distributed in urban soils.[35,51,52] These soils play an important but poorly quantified role in childhood lead exposure via resuspension of lead-contaminated urban dust. The very soil under urbanites' feet is now a primary exposure pathway for lead, contributing to pockets of poor health throughout these older cities. Lack of awareness, funding, and resources has limited the possibility of improving the lead-poisoning outlook for some children, particularly those of color living at or below the poverty level in these cities.

Our previous work[33,35,53,54] indicates that lead-saturated soils and the periodic resuspension of dust particles play a major role in chronic lead exposure in urban children. The spatial pattern of lead exposure in our hometown, Indianapolis, has remained remarkably similar from the early 1990s to the mid-2000s.[33,50] Our work indicates a tight spatial connection between the soil repository for environmental lead, a legacy of 100+ years of urban lead emissions, and elevated BLLs in children,[50] with indoor and outdoor dust generated from these soils as the most likely exposure mechanism. This mechanism also drives temporal patterns in BLLs, with seasonal resuspension of lead-contaminated dust matching seasonal patterns in BLLs in Indianapolis and many other major cities.[54] Legacy sources represent one of several dimensions that distinguish lead exposure among environmental health inequities for its enduring impact across both lifetimes and generations.[55]

INTERGENERATIONAL TRANSFERS OF LEAD TOXICITY

The disproportionate exposure to lead is transferred intergenerationally from parent to child through both direct and indirect means.[56] First, mothers can directly transfer lead toxicity to their children pre- and postpartum. Among women with past lead exposure, the calcium stress experienced during pregnancy and lactation can cause the release of lead stores in bone to the bloodstream, which may then be transmitted across the placenta.[10] Pregnant women with elevated BLLs are more likely to have adverse birth outcomes, such as preterm labor, low birth weight, preterm birth, and stillbirth.[10,12] The negative outcomes can continue beyond labor, with breast-feeding serving as an additional mode of intergenerational transfer.[57,58] Finally, it has been well documented that parents can cause their children

secondhand or paraoccupational exposure of lead through associated hazards at their workplace (ie, through transfer from clothing or vehicles contaminated at factories or construction sites).[59-66] This type of transfer constitutes a specific equity concern, given the "disproportionate employment of poor and minority workers in hazardous jobs."[67(p284)]

Second, parents can indirectly transfer lead exposure risk to their children through the intergenerational transfer of underprivilege, which is shaped by their own previous exposure to lead. "In the case of lead, persistent poverty is a likely route of intergenerational transfer of lead exposure."[68(p249)] In general, lead exposure falls into the wide-ranging category of illness that is shaped by the intergenerational transfer of living, social, and economic environments that contribute to health risks.[69] However, the association with lead exposure is even more symbiotic than other health risks, given the previously reviewed association between lead toxicity, neurologic development, and educational attainment. Parents' exposure to lead in childhood decreased their educational attainment and employment opportunities, increasing their children's likelihood for exposure to lead. This type of indirect transfer of the likelihood of exposure perpetuates basic inequities in lead exposure across entire populations and generations.

ENVIRONMENTAL JUSTICE AS PRIMORDIAL PREVENTION

All of these issues raise classic questions in the area of environmental justice. Specifically, are inequities in lead toxicity a case of discrimination or are they a natural outcome of the free housing and business markets? The intergenerational and legacy evidence indicates that current racial differentials are the result of discrimination, even if not primarily based on current, active discrimination. As a whole, the literature suggests that disproportionate exposure and uptake of lead by minority urban populations are at least partly the result of past discriminatory actions, in addition to more recent *inaction*.

If we enumerate all of the specific inactions evaluated thus far, they include (1) unwillingness to require landlords to remediate lead paint, (2) unwillingness to replace city-owned lead pipes, and (3) unwillingness to apply the Civil Rights Act provisions when siting polluting facilities. These potential actions are excellent examples of the most promising type of public health initiatives to address inequitable lead exposure and uptake: primordial prevention efforts. Primordial prevention refers to addressing the most fundamental causes of diseases by implementing "intervention at the most distal point in the chain of causality."[70(p454)] It is concerned with preventing social and environmental conditions that constitute risk factors for illness.[71]

Attention to primordial prevention has gained ground within certain public health spheres, for example, cardiovascular disease. There is a large literature on primordial prevention of cardiovascular disease (especially to address racial inequities[72]), and primordial prevention has even been integrated into the American Heart Association's strategic plan.[73] Numerous chronic disease prevention efforts could benefit from this type of view, and, given the previous review, its application to the issue of chronic lead exposure is even more direct and essential than other chronic illness risks.

Effective primordial prevention necessitates a discussion of structural racism and the role it plays in the continued cycle of lead poisoning among minority populations living in already disadvantaged circumstances. In 1998, leading researcher Dr Herbert Needleman published an article in the *American Journal of Public Health* that clearly stated the failures of the government and national agencies to make appropriate strides toward prevention of lead poisoning after the "Strategic Plan for the Elimination of Childhood Lead Poisoning"[74] wavered and was eventually forgotten.[75] Needleman perhaps best summed up his frustration over the failure by stating "as the current attitude of indifference toward problems of the poor and minorities developed, the attack on lead exposure lost its urgency."[75(p1875)] What's more, although this strategic plan had the potential to make an enormous impact on the eradication of childhood lead poisoning throughout the United States—while also being cost-effective—it was met with opposition from national agencies, pediatricians, landlords, realtors, and the lead industry. These groups claimed that lead abatement was too expensive and that universal childhood lead screenings were too cumbersome.[75]

The long-term effects of structural racism have led to the hypersegregation of black communities, which further drives the perpetuation of the disproportionate burden of lead poisoning.[76] This, coupled with the lack of resources and interest at a governmental level, has created a serious environmental justice issue that begs for action. The race-related concerns raised by researchers such as Herbert Needleman and Max Weintraub in the late 1990s still largely ring true today, as many efforts to reduce or eliminate childhood lead poisoning falter, are geared toward the wrong audience, or come once the damage has already been done.

Gee and Payne-Sturges[77] propose a model for understanding environmental health disparities that emphasizes the need to investigate pivotal

mechanisms through which vulnerability to environmental hazards is modified at both the individual and community levels. To successfully address geographically concentrated lead exposure (as one of many environmental justice imperatives), it is critical to understand and mobilize change in community-level social and behavioral dynamics that contribute to and mediate environmental susceptibility to chronic lead exposure. The shift toward primary prevention called for by Gee and Payne-Sturges—and advocated by various public health practitioners—is already occurring. This approach can result in many positive outcomes through increasing awareness and modifying behaviors and, perhaps, even increasing access to quality housing and suburban environments. However, the former 2 actions place an unwarranted burden on specific, marginalized populations. The latter leaves entire areas as contaminated and dangerous places to live, play, and grow. Both of these residual effects should be taken into account when deciding upon the most effective avenues for action.[78] It is, therefore, essential to simultaneously create change within the policy, business, and legal realms.[79]

CONCLUSION

Given all of these considerations—the well-documented geographic concentrations, legacy sources rooted in historic inequities, and intergenerational transfers of risk that are predicated upon fundamental social inequities—public health practitioners should begin to consider primordial prevention efforts that emphasize systems changes that span beyond the health systems. Our previous review provides several examples of initial public health awareness campaigns that led to effective primary prevention outside of the health realm (eg, limiting the use of lead paint and leaded gas). However, the effectiveness of awareness was consistently stalled when moving on to primordial prevention of some of the fundamental causes of disproportionate lead toxicity in our most vulnerable urban populations. Interdisciplinary, collaborative, and deliberate social action efforts are needed to implement primordial-level prevention efforts.

These efforts may require partnering with legal action teams (which is not a new concept, given Medical-Legal Partnerships).[80] They may require engaging in knowledge mobilization efforts[81] and partnering with intermediary organizations.[82] Overall, prevention efforts must move beyond individual-, population-, and even community-level surveillance approaches that are currently implemented. The environmental chronic exposure context (ie, particulate-borne exposure pathway for lead) is relatively novel but well-grounded in research[35,50,53,83,84] and indicates that public health surveillance has been missing a substantial component of urban populations' community-level lead exposure. This absence becomes even more obvious as the BLL standards are reduced and the extent of chronic, lower-level lead exposure becomes more apparent in terms of educational outcomes, criminological outcomes, and adult health outcomes. Conservative cost-benefit analyses reveal that the monetary cost of prevention efforts in the United States (including lead abatement) far outweighs the economic cost of long-term treatment of children exposed to lead.[75,85]

REFERENCES

1. Meyer P, Pivertz T, Dignam T, Homa D, Schoonover J, Brody D. Surveillance for elevated blood lead levels among children—United States, 1997-2001. *MMWR Morb Mortal Wkly Rep.* 2003;52(SS-10):1-21.
2. McCoy T. Freddie Gray's life a study on the effects of lead paint on poor blacks. http://www.washingtonpost.com/local/freddie-grays-life-a-study-in-the-sad-effects-of-lead-paint-on-poor-blacks/2015/04/29/0be898e6-eea8-11e4-8abc-d6aa3bad79dd_story.html. Published April 2015. Accessed June 1, 2015.
3. Stretesky P, Lynch M. The relationship between lead and crime. *J Health Soc Behav.* 2004;45(2):214-229.
4. Wright J, Dietrich K, Rae M, et al. Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. *PLoS Med.* 2008;5(5):e101.
5. Needleman H, McFarland C, Ness R, Fienberg S, Tobin M. Bone lead levels in adjudicated delinquents: a case control study. *Neurotoxicol Teratol.* 2002;24(6):711-717. Available from: PsycINFO, Ipswich, MA.
6. Nevin R. Understanding international crime trends: the legacy of preschool lead exposure. *Environ Res.* 2007;104(3):315-336.
7. Lanphear B, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents … including commentary by Landrigan PJ. *Public Health Rep.* 2000;115(6):521-529.
8. Chandramouli K, Steer C, Ellis M, Emond A. Effects of early childhood lead exposure on academic performance and behaviour of school age children. *Arch Dis Child.* 2009;94(11):844-848.
9. Amato M, Moore C, Kanarek M, et al. Lead exposure and educational proficiency: moderate lead exposure and educational proficiency on end-of-grade examinations. *Ann Epidemiol.* 2012;22(10):738-743.
10. Agency for Toxic Substances and Disease Registry. Environmental Medicine & Environmental Health Education—CSEM. Lead (Pb) toxicity: what are the physiologic effects of lead exposure? http://www.atsdr.cdc.gov/csem/csem.asp?csem=7&po=10. Published 2010. Accessed June 1, 2015.
11. Centers for Disease Control and Prevention. Response to the Advisory Committee on Childhood Lead Poisoning Prevention report, low level lead exposure harms children: a renewed call for primary prevention. *MMWR Morb Mortal Wkly Rep.* 2012;61(20):383. Available from: Academic Search Premier, Ipswich, MA. Accessed June 10, 2015.
12. Flora G, Gupta D, Tiwari A. Toxicity of lead: a review with recent updates. *Interdiscip Toxicol.* 2012;5(2):47-58.

13. Lanphear B, Hornung R, Khoury J, et al. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environ Health Perspect.* 2005;113(7):894-899.
14. Canfield R, Henderson C, Cory-Slechta D, Cox C, Jusko T, Lanphear B. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. *N Engl J Med.* 2003;348(16):1517-1526.
15. Needleman H, Schell A, Bellinger D, Leviton A, Allred E. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. *N Engl J Med.* 1990;322(2):83-88.
16. Fergusson D, Horwood L, Lynskey M. Early dentine lead levels and educational outcomes at 18 years. *Child Psychol Psychiatry.* 1997;38(4):471-478.
17. Staessen J, Nawrot T, Roels H, et al. Renal function, cytogenetic measurements, and sexual development in adolescents in relation to environmental pollutants: a feasibility study of biomarkers. *Lancet.* 2001;357(9269):1660-1669.
18. Fadrowski J, Navas-Acien A, Tellez-Plaza M, Guallar E, Weaver V, Furth S. Blood lead level and kidney function in US adolescents: the third National Health and Nutrition Examination Survey. *Arch Intern Med.* 2010;170(1):75-82.
19. Hu H, Aro A, Rotnitzky A, et al. The relationship of bone and blood lead to hypertension: the Normative Aging Study. *JAMA.* 1996;275(15):1171-1176.
20. Krieger J, Higgins D. Housing and health: time again for public health action. *Am J Public Health*. 2002;92(5):758-768
21. Robert Wood Johnson Foundation. Housing and health. http://www.rwjf.org/content/dam/farm/reports/issue_briefs/2011/rwjf70451. Published May 2011. Accessed June 1, 2015.
22. US Department of Housing and Urban Development and the Bureau of the Census. *American Housing Survey for the United States: 2013, Current Housing Reports, Series H150/99*. Washington, DC: US Government Printing Office.
23. Jacobs D. Environmental health disparities in housing. *Am J Public Health.* 2011;101(S1):S115-S122.
24. Cubbin C, Egerter S, Braveman P, Pedregon V; Robert Wood Johnson Foundation. Where we live matters for our health: neighborhoods and health. https://folio.iupui.edu/bitstream/handle/10244/638/commissionneighborhood102008.pdf. Published 2008. Accessed June 1, 2015.
25. Schulz A, Williams D, Israel B, Lempert L. Racial and spatial relations as fundamental determinants of health in Detroit. *Milbank Q.* 2002;80(4):677-707. Available from: Academic Search Premier, Ipswich, MA. Accessed June 10, 2015.
26. Joint Center for Political and Economic Studies. Place matters for health in Bernalillo County: ensuring opportunities for good health for all. http://jointcenter.org/sites/default/files/Place%20Matters%20for%20Health%20in%20Bernalillo%20County%20-%20English%20Summary.pdf. Published 2012. Accessed June 2015.
27. Bernard S, McGeehin M. Prevalence of blood lead levels $\geq$5 μg/dL among US children 1 to 5 years of age and socioeconomic and demographic factors associated with blood of lead levels 5 to 10 μg/dL, Third National Health and Nutrition Examination Survey, 1988-1994. *Pediatrics.* 2003;112(6):1308-1313. Available from: Academic Search Premier, Ipswich, MA. Accessed June 10, 2015.
28. Raymond J, Wheeler W, Brown M. Lead Screening and prevalence of blood lead levels in children aged 1-2 years—Child Blood Lead Surveillance System, United States, 2002-2010 and National Health and Nutrition Examination Survey, United States, 1999-2010. *MMWR Morb Mortal Wkly Rep.* 2014;63:36-42. Available from: Academic Search Premier, Ipswich, MA. Accessed June 10, 2015.
29. Braveman P, Egerter S, Mockenhaupt R. Broadening the focus: the need to address the social determinants of health. *Am J Prev Med.* 2011;40:S4-S18. Available from: Academic Search Premier, Ipswich, MA. Accessed June 10, 2015.
30. MacArthur Foundation Research Network on SES and Health. Reaching for a healthier life: facts on socioeconomic status and health in the U.S. http://www.nhpf.org/library/handouts/Adler.Kawachi.slides_03-14-08.pdf. Published September 2008. Accessed June 1, 2015.
31. Lanphear BP, Matte TD, Rogers J, et al. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. *Environ Res.* 1998;79(1):51-68. Available from: PubMed, Bethesda, MD. Accessed June 10, 2015.
32. Mielke H. Lead in New Orleans soils: new images of an urban environment. *Environ Geochem Health.* 1994;16(3-4):123-128. Available from: MEDLINE, Ipswich, MA.
33. Filippelli GM, Laidlaw MAS, Latimer JC, Raftis R. Urban lead poisoning and medical geology: an unfinished story. *GSA Today.* 2005;15(1):4-11.
34. Young T, Heerman D, Sirin G, Ashbaugh L. Resuspension of soil as a source of airborne lead near industrial facilities and highways. *Environ Sci Technol.* 2002;36(11):2484. Available from: GreenFILE, Ipswich, MA. Accessed June 10, 2015.
35. Laidlaw M, Filippelli G. Resuspension of urban soils as a persistent source of lead poisoning in children: a review and new directions. *Appl Geochem.* 2008;23(8):2021-2039. Available from: Academic Search Premier, Ipswich, MA. Accessed June 10, 2015.
36. Howell A, Timberlake J. Racial and ethnic trends in the suburbanization of poverty in U.S. metropolitan areas, 1980-2010. *J Urban Aff.* 2014;36(1):79-98. Available from: SocINDEX with Full Text, Ipswich, MA. Accessed June 10, 2015.
37. Houston D, Jun W, Ong P, Winer A. Structural disparities of urban traffic in southern California: implications for vehicle-related air pollution exposure in minority and high-poverty neighborhoods. *J Urban Aff.* 2004;26(5):565-592. Available from: SocINDEX with Full Text, Ipswich, MA. Accessed June 10, 2015.
38. Rowangould GM. A census of the US near-roadway population: public health and environmental justice considerations. *Transportation Res Part D Transport Environ*. 2013;25:59-67.
39. Rabin R. The lead industry and lead water pipes "a modest campaign." *Am J Public Health.* 2008;98(9):1584-1592. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
40. US Environmental Protection Agency. Lead in drinking water—basic information. http://www.epa.gov/safewater/lead/basicinformation.html#tapwater. Accessed June 9, 2015.
41. Allen JA, Li R, Scanlon KS, et al. Progress in increasing breastfeeding and reducing racial/ethnic differences—United States, 2000-2008 births. *MMWR Morb Mortal Wkly Rep.* 2013;62:77-80. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
42. Li R, Darling N, Emmanuel M, Barker L, Grummer-Strawn L. Breastfeeding rates in the United States by characteristics of the child, mother, or family: the 2002 National Immunization Survey. *Pediatrics*. 2005;115(1):e31-e37.
43. Levin R, Brown M, Sinks T, et al. Lead exposures in U.S. children, 2008: implications for prevention. *Environ*

Health Perspect. 2008;116(10):1285-1293. Available from: Health Business Full Text, Ipswich, MA. Accessed June 10, 2015.
44. US Environmental Protection Agency. Safe Drinking Water Act Amendment of 1986. http://nepis.epa.gov/Exe/ZyNET.exe/20012LJB.txt?ZyActionD=ZyDocument&Client=EPA&Index=1986%20Thru%201990&Docs=&Query=&Time=&EndTime=&SearchMethod=1&TocRestrict=n&Toc=&TocEntry=&QField=&QFieldYear=&QFieldMonth=&QFieldDay=&UseQField=&IntQFieldOp=0&ExtQFieldOp=0&XmlQuery=&File=D%3A\ZYFILES\INDEX%20DATA\86THRU90\TXT\00000015\20012LJB.txt&User=ANONYMOUS&Password=anonymous&SortMethod=h|-&MaximumDocuments=1&FuzzyDegree=0&ImageQuality=r75g8/r75g8/x150y150g16/i425&Display=p|f&DefSeekPage=x&SearchBack=ZyActionL&Back=ZyActionS&BackDesc=Results%20page&MaximumPages=1&ZyEntry=3. Published 1986. Accessed June 10, 2015.
45. Ghost Factories: poison in the ground. *USA Today*. http://www.usatoday.com/topic/B68DCD3E-7E3F-424A-BDA4-41077D772EA1/ghostfactories. Published 2012. Accessed December 23, 2014.
46. Bullard RD. *Dumping in Dixie: Race, Class, and Environmental Quality*. Boulder, CO: Westview; 1990.
47. Roberts JT, Toffolon-Weiss M. *Chronicles From the Environmental Justice Frontline*. Cambridge, MA: Cambridge University Press; 2001.
48. Cole LW, Foster SR. *From the Ground Up: Environmental Racism and the Rise of the Environmental Justice Movement*. New York, NY: NYU Press; 2001.
49. Mohai P, Pellow D, Roberts JT. Environmental justice. *Ann Rev Environ Resour.* 2009;34:405-430.
50. Morrison D, Lin Q, Filippelli G, et al. Spatial relationships between lead sources and children's blood lead levels in the urban center of Indianapolis (USA). *Environ Geochem Health.* 2013;35(2):171-183. Available from: MEDLINE, Ipswich, MA. Accessed June 10, 2015.
51. Mielke H, Reagan P. Soil is an important pathway of human lead exposure. *Environ Health Perspect Suppl.* 1998;106:217. Available from: MasterFILE Premier, Ipswich, MA. Accessed June 10, 2015.
52. Zahran S, Laidlaw M, McElmurry S, Filippelli G, Taylor M. Linking source and effect: resuspended soil lead, air lead, and children's blood lead levels in Detroit, Michigan. *Environ Sci Technol.* 2013;47(6):2839-2845. Available from: Applied Science & Technology Source, Ipswich, MA. Accessed June 10, 2015.
53. Laidlaw M, Mielke H, Filippelli G, Johnson D, Gonzales C. Seasonality and children's blood lead levels: developing a predictive model using climatic variables and blood lead data from Indianapolis, Indiana, Syracuse, New York, and New Orleans, Louisiana (USA). *Environ Health Perspect.* 2005;113(6):793-800. Available from: GreenFILE, Ipswich, MA. Accessed June 10, 2015.
54. Laidlaw M, Zahran S, Mielke H, Taylor M, Filippelli G. Re-suspension of lead contaminated urban soil as a dominant source of atmospheric lead in Birmingham, Chicago, Detroit and Pittsburgh, USA. *Atmos Environ.* 2012;49:302-310. Available from: GreenFILE, Ipswich, MA. Accessed June 10, 2015.
55. Morello-Frosch R, Shenassa E. The environmental "riskscape" and social inequality: implications for explaining maternal and child health disparities. *Environ Health Perspect.* 2006;114(8):1150-1153. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
56. Serbin L, Karp J. The intergenerational transfer of psychosocial risk: mediators of vulnerability and resilience. *Ann Rev Psychol.* 2004;55:333-363. Available from: PsycINFO, Ipswich, MA. Accessed June 10, 2015.
57. Ettinger A, Roy A, Hernandez-Avila M, et al. Maternal blood, plasma, and breast milk lead: lactational transfer and contribution to infant exposure. *Environ Health Perspect.* 2014;122(1):87-92. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
58. Lozoff B, Jimenez E, Pino P, et al. Higher infant blood lead levels with longer duration of breastfeeding. *J Pediatr.* 2009;155(5):663-667. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
59. Chan J, Sim M, Golec R, Forbes A. Predictors of lead absorption in children of lead workers. *Occup Med* (Oxford, England). 2000;50(6):398-405. Available from: MEDLINE, Ipswich, MA. Accessed June 10, 2015.
60. Hipkins K, Materna B, Payne S, Kirsch L. Family lead poisoning associated with occupational exposure. *Clin Pediatr.* 2004;43(9):845-849. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
61. Kar-Purkayastha I, Balasegaram S, Cordery R, et al. Lead: ongoing public and occupational health issues in vulnerable populations: a case study. *J Public Health.* 2012;34(2):176-182. Available from: CINAHL Complete, Ipswich, MA. Accessed June 10, 2015.
62. Centers for Disease Control and Prevention. Occupational and take-home lead poisoning associated with restoring chemically stripped furniture—California, 1998. *MMWR Morb Mortal Wkly Rep.* 2001;50(13):246-248. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
63. Centers for Disease Control and Prevention. Childhood lead poisoning associated with lead dust contamination of family vehicles and child safety seats—Maine, 2008. *MMWR Morb Mortal Wkly Rep.* 2009;58(32):890-893. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
64. Piacitelli G, Whelan E, Sieber W, Gerwel B. Elevated lead contamination in homes of construction workers. *Am Ind Hyg Assoc J.* 1997;58(6):447-454. Available from: MEDLINE, Ipswich, MA. Accessed June 10, 2015.
65. Roscoe R, Gittleman J, Deddens J, Petersen M, Halperin W. Blood lead levels among children of lead-exposed workers: a meta-analysis. *Am J Ind Med.* 1999;36(4):475-481. Available from: MEDLINE, Ipswich, MA. Accessed June 10, 2015.
66. Whelan E, Piacitelli G, Matte T, et al. Elevated blood lead levels in children of construction workers. *Am J Public Health.* 1997;87(8):1352-1358. Available from: Business Source Premier, Ipswich, MA. Accessed June 10, 2015.
67. Rauh V, Landrigan P, Claudio L. Housing and health. *Ann N Y Acad Sci.* 2008;1136(1):276-288.
68. Dilworth-Bart J, Moore C. Mercy Mercy Me: social injustice and the prevention of environmental pollutant exposures among ethnic minority and poor children. *Child Dev.* 2006;77(2):247-265. Available from: SocINDEX with Full Text, Ipswich, MA. Accessed June 10, 2015.
69. Lawlor D, Andersen A, Batty G. Birth cohort studies: past, present and future. *Int J Epidemiol.* 2009;38(4):897-902. Available from: Family & Society Studies Worldwide, Ipswich, MA. Accessed June 10, 2015.
70. Starfield B. Basic concepts in population health and health care. *J Epidemiol Community Health.* 2001;55(7):452-454. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
71. Starfield B, Hyde J, Gérvas J, Heath I. The concept of prevention: a good idea gone astray? *J Epidemiol Community Health.* 2008;62(7):580-583. Available from: PsycINFO, Ipswich, MA. Accessed June 10, 2015.

72. Weintraub W, Daniels S, Whitsel L, et al. Value of primordial and primary prevention for cardiovascular disease: a policy statement from the American Heart Association. *Circulation.* 2011;124(8):967-990. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
73. Huffman M, Capewell S, Ning H, Shay C, Ford E, Lloyd-Jones D. Cardiovascular health behavior and health factor changes (1988-2008) and projections to 2020: results from the National Health and Nutrition Examination Surveys. *Circulation.* 2012;125(21):2595-2602. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
74. Binder S, Falk H. *Strategic Plan for the Elimination of Childhood Lead Poisoning*. Atlanta, GA: Centers for Disease Control and Prevention (DHHS/PHS); 1991. Available from: ERIC, Ipswich, MA. Accessed June 10, 2015.
75. Needleman H. Childhood lead poisoning: the promise and abandonment of primary prevention. *Am J Public Health.* 1998;88(12):1871-1877. Available from: Business Source Premier, Ipswich, MA. Accessed June 10, 2015.
76. Weintraub M. Racism and lead poisoning. *Am J Public Health.* 1997;87(11):1871-1872. Available from: MEDLINE, Ipswich, MA. Accessed June 10, 2015.
77. Gee G, Payne-Sturges D. Environmental health disparities: a framework integrating psychosocial and environmental concepts. *Environ Health Perspect.* 2004;112(17):1645-1653. Available from: GreenFILE, Ipswich, MA. Accessed June 10, 2015.
78. Quigley R, den Broeder L, Furu P, Bond A, Cave B, Bos R. *Health Impact Assessment International Best Practice Principles*. Fargo, ND: International Association for Impact Assessment; 2006:1-4. Special Publication Series No. 5.
79. Hodge J, Fuse Brown E, Scanlon M, Corbett A. Legal review concerning the use of health impact assessments in non-health sectors. *J Allied Health.* 2012;41(2):52. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
80. Sandel M, Hansen M, Zuckerman B, et al. Medical-legal partnerships: transforming primary care by addressing the legal needs of vulnerable populations. *Health Aff.* 2010;29(9):1697-1705. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.
81. Bennet A, Bennet D, Fafard K, et al. *Knowledge Mobilization in the Social Sciences and Humanities*. Frost, WV: MQI Press; 2007.
82. Minkler M, Blackwell A, Thompson M, Tamir H. Community-based participatory research: implications for public health funding. *Am J Public Health.* 2003;93(8):1210-1213. Available from: Business Source Premier, Ipswich, MA. Accessed June 10, 2015.
83. Dong S, Yao M. Exposure assessment in Beijing, China: biological agents, ultrafine particles, and lead. *Environ Monit Assess.* 2010;170(1-4):331-343. Available from: MEDLINE, Ipswich, MA. Accessed June 10, 2015.
84. Filippelli G, Laidlaw M. The elephant in the playground: confronting lead-contaminated soils as an important source of lead burdens to urban populations. *Perspect Biol Med.* 2010;53(1):31-45. Available from: PsycINFO, Ipswich, MA. Accessed June 10, 2015.
85. Gould E. Childhood lead poisoning: conservative estimates of the social and economic benefits of lead hazard control. *Environ Health Perspect.* 2009;117(7):1162-1167. Available from: CINAHL Plus with Full Text, Ipswich, MA. Accessed June 10, 2015.