Lead

Lead is a naturally occurring metal used in the production of fuels, paints, ceramic products, batteries, solder, and a variety of consumer products. The use of leaded gasoline and leadbased paint was eliminated or restricted in the United States beginning in the 1970s, resulting in substantial reductions in exposure to lead. However, children continue to be exposed to lead due to the widespread distribution of lead in the environment. For example, children are exposed to lead through the presence of lead-based paint in many older homes, the presence of lead in drinking water distribution systems, and current use of lead in the manufacture of some products.

In the United States, the major current source of early childhood lead exposure is leadcontaminated house dust.^{1,2} Exposure to lead in house dust tends to be highest for young children, due to their frequent and extensive contact with floors, carpets, window areas, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity. A major contributor to lead in house dust is deteriorated or disrupted lead-based paint.³⁻⁵ Housing units constructed before 1950 are most likely to contain lead-based paint, but any housing unit constructed before 1978 may also contain lead-based paint.⁶ As of 2000, approximately 15.5 million housing units in the United States had one or more lead dust hazards on either floors or windowsills.⁷ New lead dust hazards occur when lead in house paint is released during home renovation and remodeling activities.^{8,9}

Two other contributors to lead in house dust are lead-contaminated soil and airborne lead.¹⁰⁻¹³ Known sources of lead in soil include historical airborne emissions of leaded gasoline, emissions from industrial sources such as smelters, and lead-based paint.^{14,15} Current sources of lead in ambient air in the United States include smelters, ore mining and processing, lead acid battery manufacturing, and coal combustion activities such as electricity generation.¹⁵

Lead-contaminated house dust is not the only source of childhood lead exposure. Direct contact with lead-contaminated soil,¹³ ingestion of lead-based paint chips,¹⁶ and inhalation of lead in ambient air also contribute to childhood lead exposure. Drinking water is an additional known source of lead exposure among children in the United States, particularly from corrosion of pipes and other elements of the drinking water distribution systems.^{5,17,18} Exposure to lead via drinking water may be particularly high among very young children who consume baby formula prepared with drinking water that is contaminated by leaching lead pipes.¹⁷ Although childhood exposure to lead in the United States typically occurs through contact with contaminated environmental media; children may also be exposed through lead-contaminated toys;^{5,19} jewelry;²⁰ tobacco smoke;²¹ imported candies, spices, and condiments;^{5,22} and imported folk remedies.^{23,24}

Compared with adults, children's bodies typically absorb a much greater fraction of a given amount of ingested lead. Once absorbed, most of the lead is stored in bones, where it can stay

many years, while other lead goes into the blood and can be eliminated more quickly. Elimination of lead from the body usually occurs through urine or feces.²⁵

Childhood blood lead levels in the United States differ across groups in the population, such as those defined by socioeconomic status and race/ethnicity.²⁶ Children living in poverty and Black non-Hispanic children tend to have higher blood lead levels²⁷ and higher levels of lead-contaminated dust in the home⁶ than do other children. Blood lead levels tend to be higher for children living in older housing, most likely because older housing units are more likely to contain lead-based paint.^{6,28} Blood lead levels may vary by nutritional status: conditions such as iron deficiency have been associated with higher blood lead levels in children.¹⁵ In addition, some children who have immigrated to the United States may have been exposed to lead in their previous countries of residence. Foreign birth place and recent foreign residence have both been positively associated with the risk of elevated blood lead levels among immigrant children in the United States.^{27,29}

Childhood blood lead levels in the United States have declined substantially since the 1970s. The decline in blood lead levels is due largely to the phasing out of lead in gasoline between 1973 and 1995,³⁰ and to the reduction in the number of homes with lead-based paint hazards.⁷ Some decline was also a result of regulations reducing lead levels in drinking water, as well as legislation limiting the amount of lead in paint and restricting the content of lead in solder, faucets, pipes, and plumbing, and the elimination of lead-soldered cans for food use.⁵ In the United States, lead content is banned or limited in many products, including food and beverage containers, ceramic ware, toys, Christmas trees, polyvinyl chloride pipes, vinyl mini-blinds, and playground equipment.⁵ However, because trace levels of lead may be present in these products, normal use may still result in lead exposure.⁵

The National Toxicology Program (NTP) has concluded that childhood lead exposure is associated with reduced cognitive function.³¹ Children with higher blood lead levels generally have lower scores on IQ tests³²⁻³⁸ and reduced academic achievement.³¹ In addition to the effects on IQ and school performance, research on the effects of lead has increasingly been addressing the effects of lead on behavior. The NTP has concluded that childhood lead exposure is associated with attention-related behavioral problems (including inattention, hyperactivity, and diagnosed attention-deficit/hyperactivity disorder) and increased incidence of problem behaviors (including delinquent, criminal, or antisocial behavior).³¹ Studies have reported that lead exposure in children may contribute to decreased attention,³⁸⁻⁴³ hyperactivity-impulsivity,⁴⁴ and increased likelihood of attention-deficit/hyperactivity disorder.⁴⁴⁻⁵² Other adverse behavioral outcomes that have been associated with childhood lead exposure in some studies include conduct disorders, ^{53,54} increased risks of juvenile delinquency and antisocial behaviors,⁵⁵⁻⁵⁷ higher total arrest rates, and arrest rates for violent crimes in early adulthood.^{58,59} Socioeconomic status may also modify the effect of lead on these cognitive and behavioral changes, resulting in stronger effects in children with lower socioeconomic status.^{60,61}

Mothers who are exposed to lead can transfer lead to the fetus during pregnancy and to the child while breast feeding.^{62,63} The NTP has concluded that there is "limited evidence" that prenatal lead exposure is associated with cognitive and behavioral effects in children.³¹ The Centers for Disease Control and Prevention (CDC) has recently published guidelines for screening pregnant and lactating mothers for possible lead exposure to better protect the fetus.⁶⁴

Many studies of the effects of lead focus on outcomes in children ages 5 years and younger. This focus reflects scientific thinking that early childhood is when children tend to experience peak exposures to lead, and also when they are most biologically susceptible to the effects of lead. Increased susceptibility to the neurodevelopmental effects of lead in the first three years of life is expected because this period is characterized by major growth and developmental events in the nervous system.¹⁵ However, lead is toxic to individuals of all ages, and children older than 5 years may also be susceptible to the neurodevelopmental effects of lead. Blood lead measurements at various ages in early childhood have been found to be strongly correlated with cognitive deficits,³⁶ and some analyses have found that effects are more strongly associated with blood lead levels at school age (i.e., 5- to 6-year-old children) compared with levels measured earlier in life.^{65,66}

Childhood lead exposures may also have lifelong effects. For instance, high childhood blood lead concentrations are associated with significant region-specific brain volume loss in adults, with greater effects seen in males.^{67,68} Childhood blood lead concentrations are also inversely associated with intellectual functioning in young adulthood.⁶⁹ In addition, lead stored in bones has the potential to be released into the bloodstream later in life. Such is the case with pregnant women, breastfeeding women, and elderly persons, as blood lead levels are comparatively elevated in these populations.^{25,70,71} Finally, childhood exposures to lead may contribute to a variety of neurological disorders and neurobehavioral effects in later life. ^{25,71-73}

Until recently, CDC defined a blood lead level of 10 micrograms per deciliter (μ g/dL) as "elevated"; this definition was used to identify children for blood lead case management.^{72,74} However, no level of lead exposure has been identified that is without risk of deleterious health effects.¹⁵ CDC's Advisory Committee on Childhood Lead Poisoning Prevention (ACCLPP) recommended in January 2012 that the 97.5th percentile of children's blood lead distribution (currently 5 μ g/dL) be defined as "elevated" for purposes of identifying children for follow-up activities such as environmental investigations and ongoing monitoring.⁷⁵ CDC has adopted the ACCLPP recommendation.⁷⁶ CDC specifically notes that "no level of lead in a child's blood can be specified as safe,"¹ and the NTP has concluded that there is sufficient evidence for adverse health effects in children at blood lead levels less than 5 μ g/dL.³¹

The following two indicators use the best nationally representative data available on blood lead levels over time in children. Indicators B1 and B2 present blood lead concentrations for children ages 1 to 5 years. Both indicators have been updated since the publication of *America's Children and the Environment, Third Edition* (January 2013) to include data from 2011–2012.

Indicator B1: Lead in children ages 1 to 5 years: Median and 95th percentile concentrations in blood, 1976–2012

Indicator B2: Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2007–2012

About the Indicators: Indicators B1 and B2 present concentrations of lead in blood of U.S. children ages 1 to 5 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of lead in the blood. Indicator B1 presents concentrations of lead in blood over time. Indicator B2 shows how blood lead levels differ by race/ethnicity and family income.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for lead. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). NHANES conducts interviews and physical examinations with approximately 10,000 people in each two-year year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals.*⁷⁷

Lead

Indicators B1 and B2 present levels of lead in children's blood. Blood lead levels are reflective of relatively recent exposure and, to a varying extent across individuals, may also incorporate contributions of long-term lead exposures.¹⁵ All values are reported as micrograms of lead per deciliter of blood (μ g/dL).

Concentrations of lead in the blood of children have been measured in NHANES beginning with the 1976–1980 survey cycle (referred to as NHANES II). For 2011–2012, NHANES collected lead biomonitoring data for 7,920 individuals ages 1 year and older, including 713 children ages 1 to 5. Lead was detected in 99% of all individuals sampled. The median blood lead level among all NHANES participants in 2011–2012 was 1.0 µg/dL and the 95th percentile was 2.9 µg/dL.

Data Presented in the Indicators

Indicator B1 presents median and 95th percentile concentrations of lead in blood over time for children ages 1 to 5 years, using NHANES data from 1976–2012.

Indicator B2 presents current median concentrations of lead in blood for children ages 1 to 5 years of different races/ethnicities and levels of family income, using NHANES data from 2009–2010 and 2011–2012.

The data from two NHANES cycles are combined to increase the statistical reliability of the estimates for each race/ethnicity and income group, and to reduce any possible influence of geographic variability that may occur in two-year NHANES data. The current 95th percentiles of blood lead by race/ethnicity and income are presented in the data tables.

Four race/ethnicity groups are presented in Indicator B2: White non-Hispanic, Black non-Hispanic, Mexican-American, and "All Other Races/Ethnicities." The "All Other Races/Ethnicities" category includes all other races and ethnicities not specified, together with those individuals who report more than one race. The limits of the sample design and sample size often prevent statistically reliable estimates for smaller race/ethnicity groups. The data are also tabulated across three income categories: all incomes, below the poverty level, and greater than or equal to the poverty level.

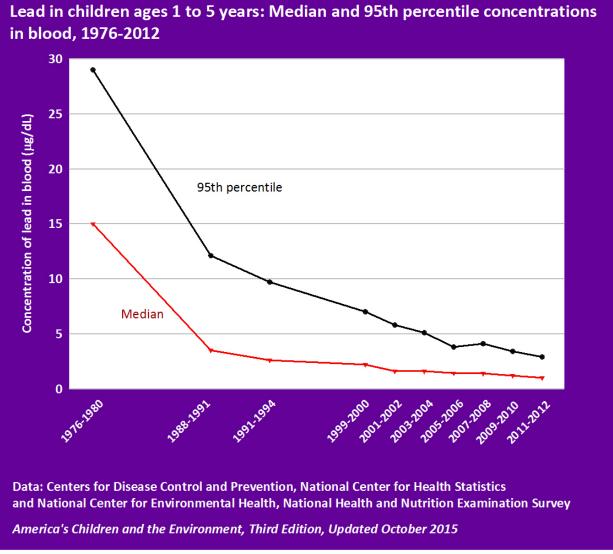
The sensitivity of measurement techniques has improved over the years spanned by Indicator B1, allowing increased detection of lower blood lead levels. These improvements do not affect the comparability of the median or 95th percentiles over time, since between 92 and 100% of children have had detectable levels of lead in each NHANES cycle.

Additional information on how median and 95th percentile blood lead levels vary among different age groups for children ages 1 to 17 years is presented in a supplementary data table. Another data table provides median blood lead levels for the same race/ethnicity and income groups in 1991–1994, for comparison with the more current data presented in Indicator B2.

The indicators focus on ages 1 to 5 years because this age range has been the focus for research, data collection, and intervention due to the elevated exposures that occur during early childhood and the sensitivity of the developing brain to the effects of lead. Blood lead data for school-age children, whose neurological development is also affected by lead exposure, are included in the data tables for this indicator.

Please see the Introduction to the Biomonitoring section for an explanation of the terms "median" and "95th percentile," and information on the statistical significance testing applied to these indicators.

Indicator B1

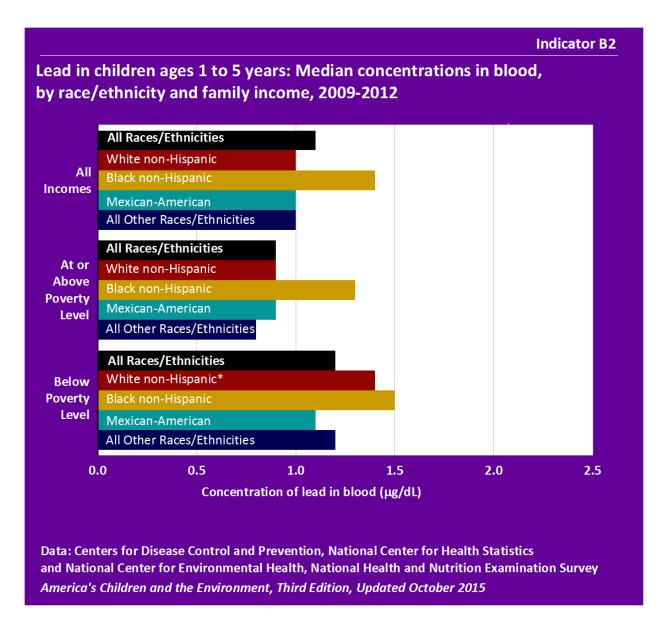


Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Lead is measured in blood samples obtained from individual survey participants.
- The median concentration of lead in the blood of children between the ages of 1 and 5 years dropped from 15 μ g/dL in 1976–1980 to 1.0 μ g/dL in 2011–2012, a decrease of 93%.
- The concentration of lead in blood at the 95th percentile in children ages 1 to 5 years dropped from 29 µg/dL in 1976–1980 to 2.9 µg/dL in 2011–2012, a decrease of 90%.
- The largest declines in blood lead levels occurred from the 1970s to the 1990s, following the elimination of lead in gasoline. The data show continuing declines in blood lead levels from

1999–2000 through 2011–2012, when the primary focus of lead reduction efforts has been on lead-based paint in homes.

- These decreasing trends were all statistically significant, including the trend in both the median and 95th percentile over the most recent 12 years (from 1999–2000 to 2011–2012).
- In 2011–2012, median blood lead levels by age group were: 1.1 μg/dL for age 1 year; 1.0 μg/dL for age 2 years; 0.9 μg/dL for ages 3 to 5 years; 0.7 μg/dL for ages 6 to 10 years; 0.5 μg/dL for ages 11 to 15 years; and 0.5 μg/dL for ages 16 to 17. The 95th percentile blood lead levels were 3.6, 3.5, 2.7, 2.0, 1.3, and 1.5 μg/dL, respectively, for ages 1, 2, 3 to 5, 6 to 10, 11 to 15, and 16 to 17 years. (See Table B1a.)
 - The differences among age groups in median and 95th percentile blood lead levels were statistically significant.



*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- Lead is measured in blood samples obtained from individual survey participants.
- The median blood lead level in children ages 1 to 5 years in 2009–2012 was 1.1 μg/dL. The median blood lead level in Black non-Hispanic children ages 1 to 5 years in 2009–2012 was

1.4 μ g/dL, higher than the level of 1.0 μ g/dL in White non-Hispanic children, Mexican-American children, and children of "All Other Races/Ethnicities."

- The median blood lead level in Black non-Hispanic children was statistically significantly higher than the median level for each of the remaining race/ethnicity groups.
- The median blood lead level for children living in families with incomes below the poverty level was 1.2 μg/dL, and for children living in families at or above the poverty level it was 0.9 μg/dL, a difference that was statistically significant.
- The median blood lead levels in children ages 1 to 5 years were higher for those in families with incomes below the poverty level compared with those at or above the poverty level within each race/ethnicity group. (See Table B2a.)
 - The differences in median blood lead levels between income groups were statistically significant for each race/ethnicity group.
- The 95th percentile blood lead level among all children ages 1 to 5 years was 3.2 µg/dL. The 95th percentile blood lead level in Black non-Hispanic children ages 1 to 5 years in 2009–2012 was 4.1 µg/dL, compared with 3.4 µg/dL for White non-Hispanic children, and 2.6 µg/dL for children of "All Other Races/Ethnicities" and Mexican-American children. (See Table B2a.)
 - The 95th percentile blood lead level in Black non-Hispanic children was statistically significantly higher than the 95th percentile forMexican-American children and children of "All Other Races/Ethnicities."
- Among children ages 1 to 5 years in families with incomes below poverty level, the 95th percentile blood lead was 4.3 µg/dL, and among those in families at or above the poverty level, it was 2.6 µg/dL, a difference that was statistically significant. (See Table B2a.)
- The 95th percentile blood lead levels in children ages 1 to 5 years were higher for those in families with incomes below the poverty level compared with those at or above the poverty level for Black non-Hispanic children, Mexican-American children, and children of "All Other Races/Ethnicities." (See Table B2a.)
 - The difference in 95th percentile blood lead levels between income groups was statistically significant for Mexican-American children after accounting for differences by age and sex.
- Between 1991–1994 and 2009–2012, median blood lead levels among Black non-Hispanic children ages 1 to 5 years declined 67%: from 4.3 µg/dL to 1.4 µg/dL. Over the same time period, median blood lead levels among Mexican-American children ages 1 to 5 years declined 68%: from 3.1 µg/dL to 1.0 µg/dL, and median blood lead levels among White non-Hispanic children ages 1 to 5 years declined 57%: from 2.3 µg/dL to 1.0 µg/dL. The differences over time were statistically significant for each race/ethnicity. (See Table B2b.)

Lead

1. Centers for Disease Control and Prevention. 2005. Preventing Lead Poisoning in Young Children. Atlanta, GA.

2. Lanphear, B.P., R. Hornung, M. Ho, C.R. Howard, S. Eberly, and K. Knauf. 2002. Environmental lead exposure during early childhood. *The Journal of Pediatrics* 140 (1):40-7.

3. Lanphear, B.P., and K.J. Roghmann. 1997. Pathways of lead exposure in urban children. *Environmental Research* 74 (1):67-73.

4. Rabinowitz, M., A. Leviton, H. Needleman, D. Bellinger, and C. Waternaux. 1985. Environmental correlates of infant blood lead levels in Boston. *Environmental Research* 38 (1):96-107.

5. Levin, R., M.J. Brown, M.E. Kashtock, D.E. Jacobs, E.A. Whelan, J. Rodman, M.R. Schock, A. Padilla, and T. Sinks. 2008. Lead exposures in U.S. Children, 2008: implications for prevention. *Environmental Health Perspectives* 116 (10):1285-93.

6. Gaitens, J.M., S.L. Dixon, D.E. Jacobs, J. Nagaraja, W. Strauss, J.W. Wilson, and P. Ashley. 2008. U.S. Children's Exposure to Residential dust lead, 1999-2004: I. Housing and Demographic Factors. *Environmental Health Perspectives* 117 (3):461-7.

7. Jacobs, D.E., R.P. Clickner, J.Y. Zhou, S.M. Viet, D.A. Marker, J.W. Rogers, D.C. Zeldin, P. Broene, and W. Friedman. 2002. The prevalence of lead-based paint hazards in U.S. housing. *Environmental Health Perspectives* 110 (10):A599-606.

8. Centers for Disease Control and Prevention. 2009. Children with elevated blood lead levels related to home renovation, repair, and painting activities - New York State, 2006-2007. *Morbidity and Mortality Weekly Report* 58 (3):55-58.

9. Centers for Disease Control and Prevention. 1997. Children with elevated blood lead levels attributed to home renovation and remodeling activities - New York, 1993-1994. *Morbidity and Mortality Weekly Report* 45 (51-52):1120-1123.

10. Adgate, J.L., G.G. Rhoads, and P.J. Lioy. 1998. The use of isotope ratios to apportion sources of lead in Jersey City, NJ, house dust wipe samples. *Science of the Total Environment* 221 (2-3):171-80.

11. Clark, S., W. Menrath, M. Chen, P. Succop, R. Bornschein, W. Galke, and J. Wilson. 2004. The influence of exterior dust and soil lead on interior dust lead levels in housing that had undergone lead-based paint hazard control. *The Journal of Occupational and Environmental Hygiene* 1 (5):273-82.

12. von Lindern, I., S. Spalinger, V. Petroysan, and M. von Braun. 2003. Assessing remedial effectiveness through the blood lead:soil/dust lead relationship at the Bunker Hill superfund site in the Silver Valley of Idaho. *Science of the Total Environment* 303 ((1-2)):139-70.

13. Lanphear, B.P., T.D. Matte, J. Rogers, R.P. Clickner, B. Dietz, R.L. Bornschein, P. Succop, K.R. Mahaffey, S. Dixon, W. Galke, et al. 1998. The contribution of lead-contaminated house dust and residential soil to children's blood lead levels. A pooled analysis of 12 epidemiologic studies. *Environmental Research* 79 (1):51-68.

14. Mielke, H.W., and P.L. Reagan. 1998. Soil is an important pathway of human lead exposure. *Environmental Health Perspectives* 106 Suppl 1:217-29.

15. U.S. Environmental Protection Agency. 2006. *Air Quality Criteria for Lead. Volume I of II*. Washington, DC: United States Environmental Protection Agency. EPA/600/R-5/144aF.

16. McElvaine, M.D., E.G. DeUngria, T.D. Matte, C.G. Copley, and S. Binder. 1992. Prevalence of radiographic evidence of paint chip ingestion among children with moderate to severe lead poisoning, St Louis, Missouri, 1989 through 1990. *Pediatrics* 89 (4 Pt 2):740-2.

17. Edwards, M., S. Triantafyllidou, and D. Best. 2009. Elevated blood lead in young children due to lead-contaminated drinking water: Washington, DC, 2001-2004. *Environmental Science and Technology* 43 (5):1618-1623.

18. Miranda, M.L., D. Kim, A.P. Hull, C.J. Paul, and M.A. Galeano. 2007. Changes in blood lead levels associated with use of chloramines in water treatment systems. *Environmental Health Perspectives* 115 (2):221-5.

19. VanArsdale, J.L., R.D. Leiker, M. Kohn, T.A. Merritt, and B.Z. Horowitz. 2004. Lead poisoning from a toy necklace. *Pediatrics* 114 (4):1096-9.

20. Weidenhamer, J.D., and M.L. Clement. 2007. Widespread lead contamination of imported low-cost jewelry in the US. *Chemosphere* 67 (5):961-5.

21. Mannino, D.M., R. Albalak, S. Grosse, and J. Repace. 2003. Second-hand smoke exposure and blood lead levels in U.S. children. *Epidemiology* 14 (6):719-27.

22. Gorospe, E.C., and S.L. Gerstenberger. 2008. Atypical sources of childhood lead poisoning in the United States: a systematic review from 1966-2006. *Clinical Toxicology (Philadelphia)* 46 (8):728-37.

23. Saper, R.B., S.N. Kales, J. Paquin, M.J. Burns, D.M. Eisenberg, R.B. Davis, and R.S. Phillips. 2004. Heavy metal content of ayurvedic herbal medicine products. *The Journal of the American Medical Association* 292 (23):2868-73.

24. Woolf, A.D., J. Hussain, L. McCullough, M. Petranovic, and C. Chomchai. 2008. Infantile lead poisoning from an Asian tongue powder: a case report & subsequent public health inquiry. *Clinical Toxicology (Philadelphia, PA)* 46 (9):841-4.

25. Agency for Toxic Substances and Disease Registry. 2007. *Toxicological Profile for Lead*. Atlanta, GA: ATSDR, Division of Toxicology and Environmental Medicine/Applied Toxicology Branch. http://www.atsdr.cdc.gov/ToxProfiles/tp13.pdf.

26. Pirkle, J.L., R.B. Kaufmann, D.J. Brody, T. Hickman, E.W. Gunter, and D.C. Paschal. 1998. Exposure of the U.S. population to lead, 1991-1994. *Environmental Health Perspectives* 106 (11):745-50.

27. Dixon, S.L., J.M. Gaitens, D.E. Jacobs, W. Strauss, J. Nagaraja, T. Pivetz, J.W. Wilson, and P. Ashley. 2009. U.S. Children's exposure to residential dust lead, 1999-2004: II. The contribution of lead-contaminated dust to children's blood lead levels. *Environmental Health Perspectives* 117 (3):468-74.

28. Kim, D.Y., F. Staley, G. Curtis, and S. Buchanan. 2002. Relation between housing age, housing value, and childhood blood lead levels in children in Jefferson County, Ky. *American Journal of Public Health* 92 (5):769-72.

29. Tehranifar, P., J. Leighton, A.H. Auchincloss, A. Faciano, H. Alper, A. Paykin, and S. Wu. 2008. Immigration and risk of childhood lead poisoning: findings from a case control study of New York City children. *American Journal of Public Health* 98 (1):92-7.

30. U.S. Environmental Protection Agency. 2000. *National Air Quality and Emissions Trends Report, 1998*. Research Triangle Park, NC: EPA Office of Air Quality Planning and Standards. http://epa.gov/airtrends/aqtrnd98/.

31. National Toxicology Program. 2012. *NTP Monograph on Health Effects of Low-Level Lead*. Research Triangle Park, NC: National Institute of Environmental Health Sciences, National Toxicology Program. http://ntp.niehs.nih.gov/go/36443.

32. Bellinger, D., J. Sloman, A. Leviton, M. Rabinowitz, H.L. Needleman, and C. Waternaux. 1991. Low-level lead exposure and children's cognitive function in the preschool years. *Pediatrics* 87 (2):219-27.

33. Canfield, R.L., C.R. Henderson, Jr., D.A. Cory-Slechta, C. Cox, T.A. Jusko, and B.P. Lanphear. 2003. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. *New England Journal of Medicine* 348 (16):1517-26.

34. Jusko, T.A., C.R. Henderson, B.P. Lanphear, D.A. Cory-Slechta, P.J. Parsons, and R.L. Canfield. 2008. Blood lead concentrations < 10 microg/dL and child intelligence at 6 years of age. *Environmental Health Perspectives* 116 (2):243-8.

35. Lanphear, B.P., K. Dietrich, P. Auinger, and C. Cox. 2000. Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. *Public Health Reports* 115 (6):521-9.

36. Lanphear, B.P., R. Hornung, J. Khoury, K. Yolton, P. Baghurst, D.C. Bellinger, R.L. Canfield, K.N. Dietrich, R. Bornschein, T. Greene, et al. 2005. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environmental Health Perspectives* 113 (7):894-9.

37. Schnaas, L., S.J. Rothenberg, M.F. Flores, S. Martinez, C. Hernandez, E. Osorio, S.R. Velasco, and E. Perroni. 2006. Reduced intellectual development in children with prenatal lead exposure. *Environmental Health Perspectives* 114 (5):791-7.

38. Surkan, P.J., A. Zhang, F. Trachtenberg, D.B. Daniel, S. McKinlay, and D.C. Bellinger. 2007. Neuropsychological function in children with blood lead levels <10 microg/dL. *Neurotoxicology* 28 (6):1170-7.

39. Calderon, J., M.E. Navarro, M.E. Jimenez-Capdeville, M.A. Santos-Diaz, A. Golden, I. Rodriguez-Leyva, V. Borja-Aburto, and F. Diaz-Barriga. 2001. Exposure to arsenic and lead and neuropsychological development in Mexican children. *Environmental Research* 85 (2):69-76.

40. Chiodo, L.M., C. Covington, R.J. Sokol, J.H. Hannigan, J. Jannise, J. Ager, M. Greenwald, and V. Delaney-Black. 2007. Blood lead levels and specific attention effects in young children. *Neurotoxicology and Teratology* 29:538-546.

41. Chiodo, L.M., S.W. Jacobson, and J.L. Jacobson. 2004. Neurodevelopmental effects of postnatal lead exposure at very low levels. *Neurotoxicology and Teratology* 26 (3):359-71.

42. Nicolescu, R., C. Petcu, A. Cordeanu, K. Fabritius, M. Schlumpf, R. Krebs, U. Kramer, and G. Winneke. 2010. Environmental exposure to lead, but not other neurotoxic metals, relates to core elements of ADHD in Romanian children: performance and questionnaire data. *Environmental Research* 110 (5):476-83.

43. Ris, M.D., K.N. Dietrich, P.A. Succop, O.G. Berger, and R.L. Bornschein. 2004. Early exposure to lead and neuropsychological outcome in adolescence. *Journal of the International Neuropsychological Society* 10 (2):261-70.

44. Nigg, J.T., G.M. Knottnerus, M.M. Martel, M. Nikolas, K. Cavanagh, W. Karmaus, and M.D. Rappley. 2008. Low blood lead levels associated with clinically diagnosed attention-deficit/hyperactivity disorder and mediated by weak cognitive control. *Biological Psychiatry* 63 (3):325-31.

45. Braun, J.M., R.S. Kahn, T. Froehlich, P. Auinger, and B.P. Lanphear. 2006. Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives* 114 (12):1904-9.

46. Eubig, P.A., A. Aguiar, and S.L. Schantz. 2010. Lead and PCBs as risk factors for attention deficit/hyperactivity disorder. *Environmental Health Perspectives* 118 (12):1654-1667.

47. Froehlich, T.E., B.P. Lanphear, P. Auinger, R. Hornung, J.N. Epstein, J. Braun, and R.S. Kahn. 2009. Association of tobacco and lead exposures with attention-deficit/hyperactivity disorder. *Pediatrics* 124 (6):e1054-63.

48. Ha, M., H.J. Kwon, M.H. Lim, Y.K. Jee, Y.C. Hong, J.H. Leem, J. Sakong, J.M. Bae, S.J. Hong, Y.M. Roh, et al. 2009. Low blood levels of lead and mercury and symptoms of attention deficit hyperactivity in children: a report of the children's health and environment research (CHEER). *Neurotoxicology* 30 (1):31-6.

49. Nigg, J.T., M. Nikolas, G. Mark Knottnerus, K. Cavanagh, and K. Friderici. 2010. Confirmation and extension of association of blood lead with attention-deficit/hyperactivity disorder (ADHD) and ADHD symptom domains at population-typical exposure levels. *The Journal of Child Psychology and Psychiatry* 51 (1):58-65.

50. Roy, A., D. Bellinger, H. Hu, J. Schwartz, A.S. Ettinger, R.O. Wright, M. Bouchard, K. Palaniappan, and K. Balakrishnan. 2009. Lead exposure and behavior among young children in Chennai, India. *Environmental Health Perspectives* 117 (10):1607-11.

51. Tuthill, R.W. 1996. Hair lead levels related to children's classroom attention-deficit behavior. *Archives of Environmental Health* 51 (3):214-20.

52. Wang, H., X. Chen, B. Yang, M. Hao, and D. Ruan. 2008. Case-Control study of blood lead levels and Attention-Deficit Hyperactivity Disorder in Chinese children. *Environmental Health Perspectives* 116 (10):1401-06.

53. Braun, J.M., T.E. Froehlich, J.L. Daniels, K.N. Dietrich, R. Hornung, P. Auinger, and B.P. Lanphear. 2008. Association of environmental toxicants and conduct disorder in U.S. children: NHANES 2001-2004. *Environmental Health Perspectives* 116 (7):956-62.

54. Marcus, D.K., J.J. Fulton, and E.J. Clarke. 2010. Lead and conduct problems: a meta-analysis. *Journal of Clinical Child and Adolescent Psychology* 39 (2):234-41.

55. Dietrich, K.N., M.D. Ris, P.A. Succop, O.G. Berger, and R.L. Bornschein. 2001. Early exposure to lead and juvenile delinquency. *Neurotoxicology and Teratology* 23 (6):511-8.

56. Needleman, H.L., C. McFarland, R.B. Ness, S.E. Fienberg, and M.J. Tobin. 2002. Bone lead levels in adjudicated delinquents. A case control study. *Neurotoxicology and Teratology* 24 (6):711-7.

57. Needleman, H.L., J.A. Riess, M.J. Tobin, G.E. Biesecker, and J.B. Greenhouse. 1996. Bone lead levels and delinquent behavior. *The Journal of the American Medical Association* 275 (5):363-9.

58. Nevin, R. 2007. Understanding international crime trends: the legacy of preschool lead exposure. *Environmental Research* 104 (3):315-36.

59. Wright, J.P., K.N. Dietrich, M.D. Ris, R.W. Hornung, S.D. Wessel, B.P. Lanphear, M. Ho, and M.N. Rae. 2008. Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. *PLoS Medicine* 5 (5):e101.

60. Bellinger, D.C. 2008. Lead neurotoxicity and socioeconomic status: conceptual and analytical issues. *Neurotoxicology* 29 (5):828-32.

61. Weiss, B., and D.C. Bellinger. 2006. Social ecology of children's vulnerability to environmental pollutants. *Environmental Health Perspectives* 114 (10):1479-1485.

62. Chuang, H.Y., J. Schwartz, T. Gonzales-Cossio, M.C. Lugo, E. Palazuelos, A. Aro, H. Hu, and M. Hernandez-Avila. 2001. Interrelations of lead levels in bone, venous blood, and umbilical cord blood with exogenous lead exposure through maternal plasma lead in peripartum women. *Environmental Health Perspectives* 109 (5):527-32.

63. Ettinger, A.S., M.M. Tellez-Rojo, C. Amarasiriwardena, T. Gonzalez-Cossio, K.E. Peterson, A. Aro, H. Hu, and M. Hernandez-Avila. 2004. Levels of lead in breast milk and their relation to maternal blood and bone lead levels at one month postpartum. *Environmental Health Perspectives* 112 (8):926-31.

64. Advisory Committee on Childhood Lead Poisoning Prevention. 2010. *Guidelines for the Identification and Management of Lead Exposure in Pregnant and Lactating Women*. Atlanta, GA: Centers for Disease Control and Prevention. http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf.

65. Chen, A., K.N. Dietrich, J.H. Ware, J. Radcliffe, and W.J. Rogan. 2005. IQ and blood lead from 2 to 7 years of age:are the effects in older children the residual of high blood lead concentration in 2-year-olds? *Environmental Health Perspectives* 113:597-601.

66. Hornung, R.W., B.P. Lanphear, and K.N. Dietrich. 2009. Age of greatest susceptibility to childhood lead exposure: A new statistical approach. *Environmental Health Perspectives* 117 (8):1309-12.

67. Brubaker, C.J., K.N. Dietrich, B.P. Lanphear, and K.M. Cecil. 2010. The influence of age of lead exposure on adult gray matter volume. *Neurotoxicology* 31 (3):259-66.

68. Cecil, K.M., C.J. Brubaker, C.M. Adler, K.N. Dietrich, M. Altaye, J.C. Egelhoff, S. Wessel, I. Elangovan, R. Hornung, K. Jarvis, et al. 2008. Decreased brain volume in adults with childhood lead exposure. *PLoS Medicine* 5 (5):e112.

69. Mazumdar, M., D.C. Bellinger, M. Gregas, K. Abanilla, J. Bacic, and H.L. Needleman. 2011. Low-level environmental lead exposure in childhood and adult intellectual function: a follow-up study. *Environmental Health* 10 (1):24.

70. Gulson, B.L., K.J. Mizon, M.J. Korsch, J.M. Palmer, and J.B. Donnelly. 2003. Mobilization of lead from human bone tissue during pregnancy and lactation--a summary of long-term research. *Science of the Total Environment* 303 (1-2):79-104.

71. Stein, J., T. Schettler, B. Rohrer, and M. Valenti. 2008. *Environmental Threats to Health Aging: With a Closer Look at Alzheimer's and Parkinson's Diseases*. Boston, MA: Greater Boston Physicians for Social Responsibility and Science and Environmental Health Network.

 $http://www.agehealthy.org/pdf/GBPSRSEHN_HealthyAging1017.pdf.$

72. Centers for Disease Control and Prevention. 2002. *Managing Elevated Blood Lead Levels Among Young Children: Recommendations from the Advisory Committee on Childhood Lead Poisoning Prevention*. Atlanta, GA.

73. Tellez-Rojo, M.M., D.C. Bellinger, C. Arroyo-Quiroz, H. Lamadrid-Figueroa, A. Mercado-Garcia, L. Schnaas-Arrieta, R.O. Wright, M. Hernandez-Avila, and H. Hu. 2006. Longitudinal associations between blood lead concentrations lower than 10 microg/dL and neurobehavioral development in environmentally exposed children in Mexico City. *Pediatrics* 118 (2):e323-30.

74. Centers for Disease Control and Prevention. 1997. *Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials*. Atlanta, GA.

75. Advisory Committee on Childhood Lead Poisoning Prevention. 2012. *Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention*. Atlanta, GA: Centers for Disease Control and Prevention. http://www.cdc.gov/nceh/lead/ACCLPP/Final_Document_030712.pdf.

76. Centers for Disease Control and Prevention. 2012. *CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention*. Atlanta, GA: Centers for Disease Control and Prevention. http://www.cdc.gov/nceh/lead/acclpp/cdc_response_lead_exposure_recs.pdf.

77. Centers for Disease Control and Prevention. 2009. *Fourth National Report on Human Exposure to Environmental Chemicals*. Atlanta, GA: CDC. http://www.cdc.gov/exposurereport/.