America's Children and the Environment, Third Edition

DRAFT Indicators

Biomonitoring: Lead

EPA is preparing the third edition of *America's Children and the Environment* (ACE3), following the previous editions published in December 2000 and February 2003. ACE is EPA's compilation of children's environmental health indicators and related information, drawing on the best national data sources available for characterizing important aspects of the relationship between environmental contaminants and children's health. ACE includes four sections: Environments and Contaminants, Biomonitoring, Health, and Special Features.

EPA has prepared draft indicator documents for ACE3 representing 23 children's environmental health topics and presenting a total of 42 proposed children's environmental health indicators. This document presents the draft text, indicators, and documentation for the lead topic in the Biomonitoring section.

For more information on America's Children and the Environment, please visit www.epa.gov/ace. For instructions on how to submit comments on the draft ACE3 indicators, please visit www.epa.gov/ace/ace3drafts/.

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 lead-based paint was eliminated in the United States 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 lead-contaminated house dust.[1,2] 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 may occur when lead in house paint is released during home renovation and remodeling activities.[8,9]

A second contributor to lead in house dust is lead-contaminated soil.[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.[14-16] Airborne lead may also contribute to lead in house dust.[10] 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.[16]

Exposure to lead in house dust tends to be highest for young children, due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity. Direct contact with lead-contaminated soil,[13] ingestion of lead-based paint chips,[17] 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.[5,18,19] 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.[18]

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[20,21] and jewelry,[22] tobacco smoke,[23] imported candies, spices and condiments,[5,24] and imported folk remedies.[25,26]

Childhood blood lead levels in the United States differ across groups in the population such as those defined by socioeconomic status and race/ethnicity.[27] Children living in poverty and Black

non-Hispanic children tend to have higher blood lead levels[28] and higher levels of lead-contaminated dust in the home[6] 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.[6,29] Blood lead levels may vary by nutritional status: conditions such as iron deficiency have been associated with higher blood lead levels in children.[16 30,31] 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.[28,32]

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,[33] 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. 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. However, because trace levels of lead may be present in these products, normal use may still result in lead exposure.[5]

Exposure to lead during childhood is associated with reduced cognitive function. Children with higher blood lead levels generally have lower scores on IQ tests.[34-40] Childhood lead exposure has been associated with an increased likelihood of having a reading disability, dropping out of high school, and absenteeism.[41] 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. Studies have found that lead exposure in children may contribute to increased likelihood of conduct disorders,[42] decreased attention,[40,43-45] hyperactivity-impulsivity,[46] and attention-deficit/hyperactivity disorder.[46-53] Other adverse behavioral outcomes that have been associated with childhood lead exposure include increased risks of juvenile delinquency and antisocial behaviors,[54-56] higher total arrest rates, and arrest rates for violent crimes in early adulthood.[57]

Mothers who are exposed to lead can transfer lead to their unborn children during pregnancy.[58] Cognitive and behavioral effects of prenatal exposure to lead have been observed in young infants and children across numerous studies.[16,39,57,59]

Many studies of the effects of lead focus on outcomes in children ages 5 years and younger. This 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.[16] 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,[38] and some analyses have found that effects are more strongly associated with blood lead levels at school age (older than 5 years) compared with levels measured earlier in life.[60,61]

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.[62,63] In addition, lead stored in bones also has the potential to be re-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. Finally, childhood exposures to lead can contribute to a variety of neurological disorders in later life.[64-66]

Compared with adults, children's bodies typically absorb a much greater fraction of a given amount of ingested lead. Once absorbed, some 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.[64]

The Centers for Disease Control and Prevention (CDC) defines a blood lead level of 10 micrograms per deciliter as "elevated;" this definition is used to identify children for blood lead case management.[67,68] However, no level of lead exposure has been identified that is without risk of deleterious health effects,[16] and CDC specifically notes that "no level of lead in a child's blood can be specified as safe."[1] As concentrations of lead in blood have declined over the past three decades, there have been increasing opportunities to study the effects of lower blood lead levels, and adverse effects of lead on intelligence and behavior have been observed at blood lead levels lower than 10 micrograms per deciliter.[35-40,42,46,47,49-51,53,69] Scientific findings provide clear evidence of cognitive deficits in young children with blood-lead concentrations in the range of 5-10 µg/dL, with evidence of effects at blood lead levels as low as 2µg/dL.[16]

The following two indicators present data on blood lead levels in children ages 1 to 5 years. Indicator B1 shows changes over time in median and 95th percentile childhood blood lead levels, and Indicator B2 presents median blood lead levels among children grouped by race/ethnicity and income level.

Indicator B1: Lead in children ages 1 to 5 years: Median and 95th percentile concentrations in blood, 1976–2008

Indicator B2: Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2005–2008

Overview

Indicators B1 and B2 present levels 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, and then measures the concentration of lead in blood. Indicator B1 shows the change in blood lead levels over time. Indicator B2 shows how blood lead levels differ by race/ethnicity and family income.

NHANES

Data for these indicators come from the National Health and Nutrition Examination Survey (NHANES). NHANES is a nationally representative survey designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population, conducted by the Centers for Disease Control and Prevention (CDC). NHANES conducts interviews and physical examinations for approximately 5,000 people each year. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants.[70] 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). Indicator B1 uses data from all cycles of NHANES conducted since 1976–1980, including the most current data available (from samples collected in 2007–2008). Indicator B2 uses data from the 2005–2006 and 2007–2008 surveys. The data from these two NHANES cycles are combined to increase the statistical reliability of the estimates for each race/ethnicity and income group.

Blood Lead Measurement

In studies of exposure to lead and the effects of lead, blood lead is the most commonly used measurement of exposure. 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.[16]

Data Presented in the Indicators

Indicator B1 presents the median (50th percentile) and 95th percentile blood lead levels over time, and Indicator B2 presents the current (2005-2008) median blood lead levels for children of different races/ethnicities and levels of family income. The current 95th percentiles of blood lead by race/ethnicity and income are presented in the data tables. The median is the value in the

middle of the distribution of blood lead levels: half of the children have levels greater than the median, and half have levels below the median. The median can be thought of as representing a typical exposure. The 95th percentile is a value representing the upper range of blood lead levels: 5% of children have levels greater than the 95th percentile. This value therefore can be thought of as representing a relatively high exposure among children, but not a maximum level.

All values are reported as micrograms of lead per deciliter of blood (µg/dL).

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.

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 50th or 95th percentiles over time, since the majority of children have had detectable levels of lead in each NHANES cycle.

Race/Ethnicity and Income Groups

Median blood lead levels for four race/ethnicity groups are presented in Indicator B2: White non-Hispanic, Black non-Hispanic, Mexican-American, and "Other." The "Other" race/ethnicity category includes Asian non-Hispanic, Native American non-Hispanic, Hispanic other than Mexican-American, those reporting multiple racial categories, and those with a missing value for race/ethnicity. The data are also tabulated across three family income categories: all incomes, below the poverty level, and greater than or equal to the poverty level.

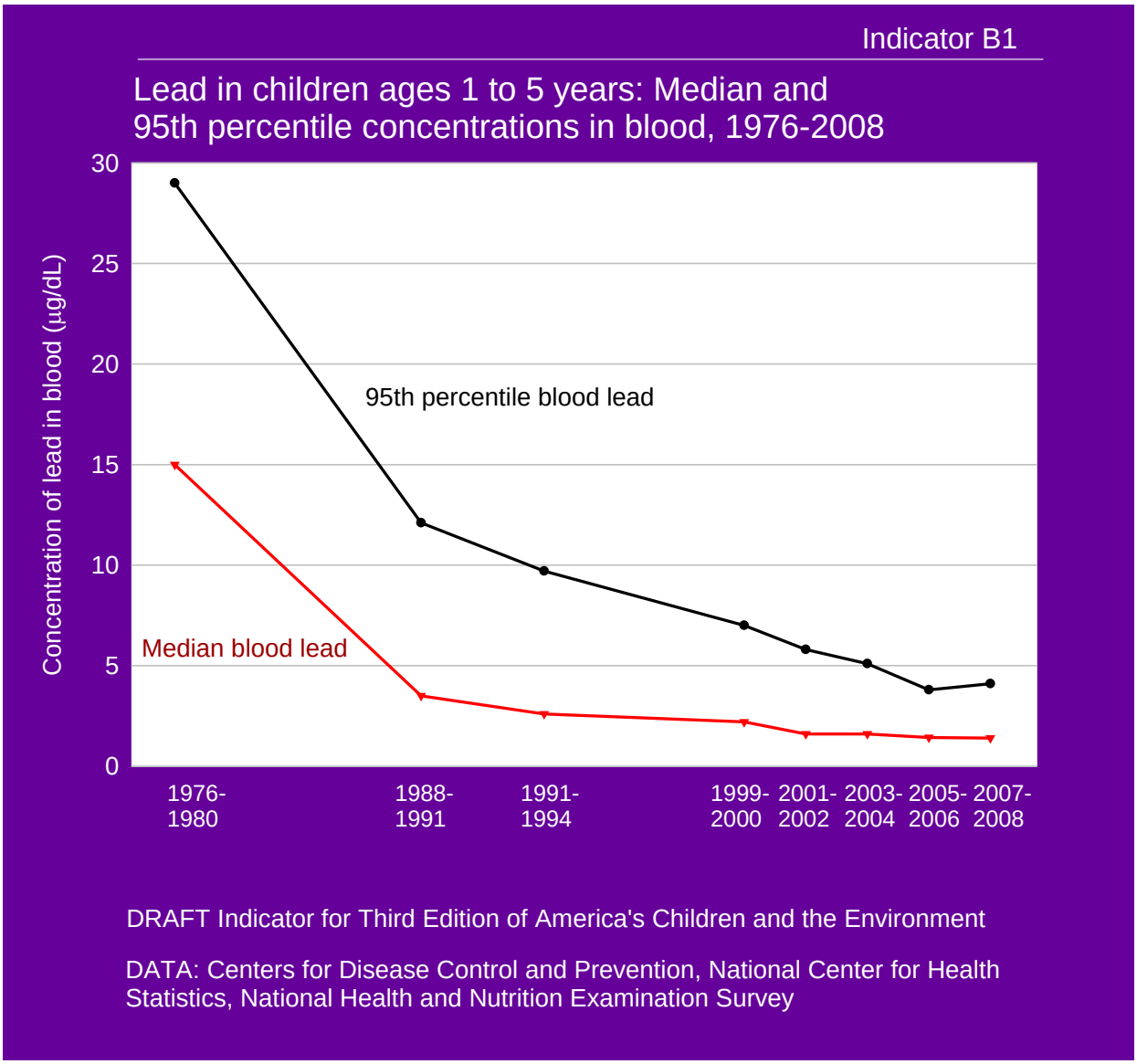
A supplementary data table also provides median blood lead levels for the same race/ethnicity and income groups in 1991–1994, for comparison with the more current data presented in Indicator B2.

Statistical Testing

Statistical analysis has been applied to the biomonitoring indicators to determine whether any changes in chemical concentrations over time, or any differences in chemical concentrations between demographic groups, are statistically significant. These analyses use a 5% significance level ($p \leq 0.05$), meaning that a conclusion of statistical significance is made only when there is no more than a 5% chance that the observed change over time or difference between demographic groups occurred randomly. It should be noted that when statistical testing is conducted for differences among multiple demographic groups (e.g., considering both race/ethnicity and income level), the large number of comparisons involved increases the probability that some differences identified as statistically significant may actually have occurred randomly.

A finding of statistical significance for a biomonitoring indicator depends not only on the numerical difference in the value of a reported statistic between two groups, but also on the

number of observations in the survey, the amount of variability among the observations, and various aspects of the survey design. For example, if two groups have different median levels of a chemical in blood or urine, the statistical test is more likely to detect a difference when samples have been obtained from a larger number of people in those groups. Similarly, if there is low variability in levels of the chemical within each group, then a difference between groups is more likely to be detected. A finding that there is or is not a statistically significant difference in exposure levels between two groups or in exposure levels over time does not necessarily suggest any interpretation regarding the health implications of those differences.



- The median concentration of lead in the blood of children between the ages of 1 and 5 years dropped from 15 micrograms per deciliter (µg/dL) in 1976–1980 to 1.4 µg/dL in 2007–2008, a decline of 91%.

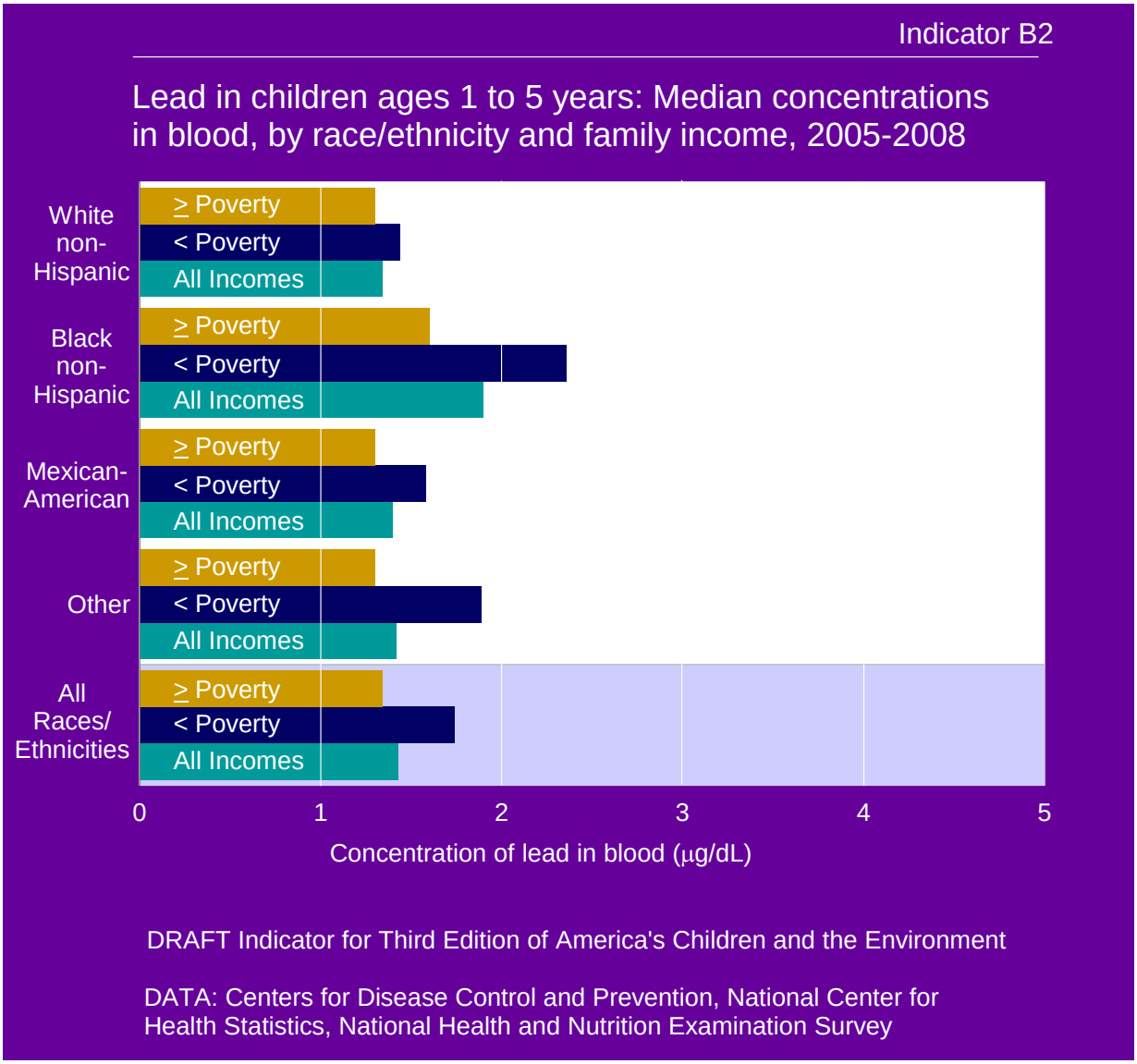
- The concentration of lead in blood at the 95th percentile in children ages 1 to 5 years dropped from 29 µg/dL in 1976–1980 to 4 µg/dL in 2007–2008, a decline of 86%.

- 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 2007–2008, when the primary focus of lead reduction efforts has been on lead-based paint in homes.

- These trends are all statistically significant.

- In 1978, about 88% of children ages 1 to 5 years (about 13.5 million children) had blood lead levels at or greater than 10 µg/dL, which is considered elevated. By 2007–2008, this number had declined to about 1% (about 250,000 children). (Data not shown).

- In 2007–2008, children ages 6 to 10 years had median blood lead levels of 1.0 µg/dL; the median for children ages 11 to 15 years was 0.8, and for ages 16 to 17 years the median was 0.7 µg/dL. The 95th percentile blood lead levels were 2.6, 2.1, and 1.7 µg/dL, respectively, for ages 6 to 10, 11 to 15, and 16 to 17 years. (See Table B1a.)



- The median blood lead level in children ages 1 to 5 years in 2005–2008 was 1.4 µg/dL. The median blood lead level for children living in families with incomes below the poverty level was 1.7 µg/dL, and for children living in families at or above the poverty level it was 1.3 µg/dL, a difference that is statistically significant.

- Black non-Hispanic children ages 1 to 5 years in families below the poverty level had median blood lead concentrations of 2.4 µg/dL, which was the highest of any group shown and 70% greater than the median for all children ages 1 to 5 years.
    - Statistical note: the difference in median blood lead level between Black non-Hispanic children below the poverty level and all other race/ethnicity and income groups is statistically significant even after accounting for other demographic differences (i.e., differences in sex or age profile).

- The 95th percentile blood lead level among all children ages 1 to 5 years was 4.1 µg/dL. Among children in families with incomes below poverty level, the 95th percentile blood lead was 6.0 µg/dL, and among Black non-Hispanic children in families with incomes below poverty level the 95th percentile blood lead was 8.6 µg/dL. (See Table B2a.)

- Between 1991–1994 and 2005–2008, median blood lead levels among Black non-Hispanic children ages 1 to 5 years declined 56%: from 4.3 µg/dL to 1.9 µg/dL. (See Table B2b.)

- Between 1991–1994 and 2005–2008, median blood lead levels among Mexican-American children ages 1 to 5 years declined 55%: from 3.1 µg/dL to 1.4 µg/dL. (See Table B2b.)

- Between 1991–1994 and 2005–2008, median blood lead levels among White non-Hispanic children ages 1 to 5 years declined 43%: from 2.3 µg/dL to 1.3 µg/dL. (See Table B2b.)

Data Tables

Table B1: Lead in children ages 1 to 5 years: Median and 95th percentile concentrations in blood, 1976-2008

	Blood lead concentration (µg/dL)							
	1976-1980	1988-1991	1991-1994	1999-2000	2001-2002	2003-2004	2005-2006	2007-2008
Median	15.0	3.5	2.6	2.2	1.6	1.6	1.4	1.4
95th percentile	29.0	12.1	9.7	7.0	5.8	5.1	3.8	4.1

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

Table B1a: Lead in children ages 1 to 17 years: Blood lead concentrations by age group, 2007–2008

	Blood lead concentration (µg/dL)						
	All ages	Age 1 to <2 years	Age 2 to <3 years	Age 3 to <6 years	Age 6 to <11 years	Age 11 to <16 years	Age 16 to <18 years
Median	0.9	1.8	1.7	1.3	1.0	0.8	0.7
95th percentile	2.9	4.6*	3.8	4.1	2.6	2.1	1.7

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

* 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).

Table B2. Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2005-2008

	Blood lead concentration (µg/dL)					
Race / Ethnicity	All Incomes	< Poverty Level	≥ Poverty Level	≥Poverty Level (Detail)		Unknown Income
				100-200% of Poverty Level	> 200% of Poverty Level	
All Races/ Ethnicities	1.4	1.7	1.3	1.5	1.3	1.8
White Non-Hispanic	1.3	1.4	1.3	1.6	1.3	1.6
Black Non-Hispanic	1.9	2.4	1.6	1.7	1.5	3.4

Race / Ethnicity	Blood lead concentration (µg/dL)					
	All Incomes	< Poverty Level	≥ Poverty Level	≥Poverty Level (Detail)		Unknown Income
				100-200% of Poverty Level	> 200% of Poverty Level	
Mexican-American	1.4	1.6	1.3	1.4	1.1	1.4
Other†	1.4	1.9	1.3	1.5	1.2	NA**

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

† "Other" includes Asian non-Hispanic; Native American non-Hispanic; Hispanic other than Mexican-American; those reporting multi-racial; and those with a missing value for race/ethnicity.

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is at least 40% (RSE = standard error divided by the estimate).

Table B2a. Lead in children ages 1 to 5 years: 95th percentile concentrations in blood, by race/ethnicity and family income, 2005-2008

Race / Ethnicity	Blood lead concentration (µg/dL)					
	All Incomes	< Poverty Level	≥ Poverty Level	≥Poverty Level (Detail)		Unknown Income
				100-200% of Poverty Level	> 200% of Poverty Level	
All Races/Ethnicities	4.1	6.0	3.4	4.1	3.1	NA**
White non-Hispanic	3.4	5.6	3.3	3.9	2.9	2.4
Black non-Hispanic	6.5	8.6	4.3	4.9*	4.2	16.3
Mexican-American	3.6	4.1	3.3	3.3	2.8	4.2
Other†	4.3	4.9	3.6	3.6	3.1*	NA**

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

† "Other" includes Asian non-Hispanic; Native American non-Hispanic; Hispanic other than Mexican-American; those reporting multi-racial; and those with a missing value for race/ethnicity.

* 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).

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is at least 40% (RSE = standard error divided by the estimate).

Table B2b. Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 1991–1994

Race / Ethnicity	Blood lead concentration (µg/dL)					
	All Incomes	< Poverty Level	≥ Poverty Level	≥Poverty (Detail)		Unknown Income
				100-200% of Poverty Level	> 200% of Poverty Level	
All Races/ Ethnicities	2.6	4.0	2.2	2.7	2.0	3.7
White Non-Hispanic	2.3	3.2	2.1	2.4	2.0	NA**
Black Non-Hispanic	4.3	5.1	3.5	3.8	3.1	5.0
Mexican-American	3.1	3.7	2.6	2.8	2.1	3.0
Other†	2.5	3.3	2.0	3.8	1.9	NA**

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey

† "Other" includes Asian non-Hispanic; Native American non-Hispanic; Hispanic other than Mexican-American; those reporting multi-racial; and those with a missing value for race/ethnicity.

** The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is at least 40% (RSE = standard error divided by the estimate).

References

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* doi: 10.1289/ehp.11917.

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. Franko, E.M., J.M. Palome, M.J. Brown, C.M. Kennedy, and L.V. Moore. 2009. Children with elevated blood lead levels related to home renovation, repair, and painting activities - New York State, 2006-2007. *Morbidity and Mortality Weekly Review* 58 (3):55-58.

9. Franko, E.M., W.N. Stasiuk, and R.W. Stevenson. 1997. Children with elevated blood lead levels attributed to home renovation and remodeling activities - New York, 1993-1994. *Morbidity and Mortality Weekly Review* 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, M. Rabinowitz, M. Farfel, C. Rohde, J. Schwartz, P. Ashley, and D.E. Jacobs. 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. 1986. *Air Quality Criteria for Lead. Volume I of IV*. Washington, DC: US Environmental Protection Agency.

16. 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.

17. 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.

18. 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.

19. 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.

20. Centers for Disease Control and Prevention. 1991. *Preventing Lead Poisoning in Young Children*. Atlanta, GA.

21. 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.

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

23. 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.

24. 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.

25. 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.

26. 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.

27. 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.

28. Dixon, S.L., J.M. Gaitens, D.E. Jacobs, W. Strauss, J. Nagaraja, T. Pivetz, J.W. Wilson, and P. Ashley. 2008. 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* doi:10.1289/ehp.11918.

29. 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.

30. Wright, R.O., M.W. Shannon, R.J. Wright, and H. Hu. 1999. Association between iron deficiency and low-level lead poisoning in an urban primary care clinic. *American Journal of Public Health* 89 (7):1049-53.

31. Wright, R.O., S.W. Tsaih, J. Schwartz, R.J. Wright, and H. Hu. 2003. Association between iron deficiency and blood lead level in a longitudinal analysis of children followed in an urban primary care clinic. *The Journal of Pediatrics* 142 (1):9-14.

32. 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.

33. 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://www.epa.gov/oar/aqtrnd98/toc.html.

34. 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.

35. 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.

36. 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.

37. 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.

38. Lanphear, B.P., R. Hornung, J. Khoury, K. Yolton, P. Baghurst, D.C. Bellinger, R.L. Canfield, K.N. Dietrich, R. Bornschein, T. Greene, S.J. Rothenberg, H.L. Needleman, L. Schnaas, G. Wasserman, J. Graziano, and R. Roberts. 2005. Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. *Environmental Health Perspectives* 113 (7):894-9.

39. 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.

40. 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.

41. Needleman, H.L., A. Schell, D. Bellinger, A. Leviton, and E.N. Allred. 1990. The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *New England Journal of Medicine* 322 (2):83-8.

42. 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.

43. 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.

44. 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.

45. 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.

46. 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.

47. 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.

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

49. 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* doi:10.1289/ehp.11400.

50. 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.

51. 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, and S.J. Jo. 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.

52. 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.

53. 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.

54. 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.

55. 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.

56. 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.

57. 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.

58. 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.

59. Patel, A.B., M.R. Mamtani, T.P. Thakre, and H. Kulkarni. 2006. Association of umbilical cord blood lead with neonatal behavior at varying levels of exposure. *Behavioral and Brain Functions* 2:22.

60. Chen, A., K.N. Dietrich, J.H. Ware, J. Radcliffe, and R. W.J. 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.

61. 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* doi:10.1289/ehp.0800426.

62. 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.

63. 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, and B.P. Lanphear. 2008. Decreased brain volume in adults with childhood lead exposure. *PLoS Medicine* 5 (5):e112.

64. 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.

65. 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.

66. 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.

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

68. 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.

69. 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.

70. Centers for Disease Control and Prevention. 2010. *Fourth National Report on Human Exposure to Environmental Chemicals*. Atlanta, GA: CDC. http://www.cdc.gov/exposurereport/.

Metadata

Metadata for	National Health and Nutrition Examination Survey (NHANES)
Brief description of the data set	The National Health and Nutrition Examination Survey (NHANES) is a program of studies designed to assess the health and nutritional status of adults and children in the United States, using a combination of interviews, physical examinations, and laboratory analysis of biological specimens.
Who provides the data set?	Centers for Disease Control and Prevention, National Center for Health Statistics.
How are the data gathered?	Laboratory data are obtained by analysis of blood and urine samples collected from survey participants at NHANES Mobile Examination Centers. Health status is assessed by physical examination. Demographic and other survey data regarding health status, nutrition and health-related behaviors are collected by personal interview, either by self-reporting or, for children under 16 and some others, as reported by an informant.
What documentation is available describing data collection procedures?	See http://www.cdc.gov/nchs/nhanes.htm for detailed survey and laboratory documentation by survey period.
What types of data relevant for children's environmental health indicators are available from this database?	Concentrations of environmental chemicals in urine, blood, and serum. Body measurements. Health status, as assessed by physical examination, laboratory measurements and interview responses. Demographic information.
What is the spatial representation of the database (national or other)?	NHANES sampling procedures provide nationally-representative data. Analysis of data for any other geographic area (region, state, etc.) is possible only by special arrangement with the NCHS Research Data Center, and such analyses may not be representative of the specified area.
Are raw data (individual measurements or survey responses) available?	Individual laboratory measurements and survey responses are generally available. Individual survey responses for some questions are not publicly released.
How are database files obtained?	http://www.cdc.gov/nchs/nhanes.htm
Are there any known data quality or data analysis concerns?	Some environmental chemicals have large percentages of values below the detection limit. Data gathered by interview, including demographic information, and responses regarding health status, nutrition and health-related behaviors are self-reported, or (for individuals age 16 years and younger)

Metadata for	**National Health and Nutrition Examination Survey (NHANES)**
	reported by an adult informant.
What documentation is available describing QA procedures?	http://www.cdc.gov/nchs/nhanes.htm includes detailed documentation on laboratory and other QA procedures. Data quality information is available at http://www.cdc.gov/nchs/about/policy/quality.htm.
For what years are data available?	Some data elements were collected in predecessors to NHANES beginning in 1959; collection of data on environmental chemicals began with measurement of blood lead in NHANES II, 1976-1980. The range of years for measurement of environmental chemicals varies; apart from lead and cotinine (initiated in NHANES III), measurement of environmental chemicals began with 1999-2000 or later NHANES.
What is the frequency of data collection?	Data are collected on continuous basis, but are grouped into NHANES cycles: NHANES II (1976-1980); NHANES III phase 1 (1988-1991); NHANES III phase 2 (1991-1994); and continuous two-year cycles beginning with 1999-2000 and continuing to the present.
What is the frequency of data release?	Data are released in two-year cycles (e.g. 1999-2000); particular data sets from a two-year NHANES cycle are released as available.
Are the data comparable across time and space?	Detection limits can vary across time, affecting some comparisons. Some contaminants are not measured in every NHANES cycle. Within any NHANES two-year cycle, data are generally collected and analyzed in the same manner for all sampling locations.
Can the data be stratified by race/ethnicity, income, and location (region, state, county or other geographic unit)?	Data are collected to be representative of the U.S. population based on age, sex, and race/ethnicity. The public release files allow stratification by these and other demographic variables, including family income range and poverty income ratio. Data cannot be stratified geographically except by special arrangement with the NCHS Research Data Center.

1

Methods

Indicator

B1. Lead in children ages 1 to 5 years: Median and 95th percentile concentrations in blood, 1976-2008

B2. Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2005-2008

Summary

Since the 1970s, the National Center for Health Statistics, a division of the Centers for Disease Control and Prevention, has conducted the National Health and Nutrition Examination Surveys (NHANES), a series of U.S. national surveys of the health and nutrition status of the noninstitutionalized civilian population. The National Center for Environmental Health at CDC measures environmental chemicals in blood and urine samples collected from NHANES participants.[i] Indicators B1 and B2 use blood lead measurements in children ages 1 to 5 years. NHANES II (1976-1980) included blood lead data for children from six months to 5 years. NHANES III (1988-1994) and the NHANES 1999-2000, 2001-2002, 2003-2004, 2005-2006, and 2007-2008 surveys included blood lead data for children ages 1 to 5 years.

Indicator B1 gives the median and 95th percentile concentrations of blood lead for children ages 1 to 5 years for each NHANES survey period. The median is the estimated concentration such that 50% of all noninstitutionalized civilian children ages 1 to 5 years during the survey period have blood lead concentrations below this level. The 95th percentile is the estimated concentration such that 95% of all noninstitutionalized civilian children ages 1 to 5 years during the survey period have blood lead concentrations below this level. Indicator B1 gives the median and 95th percentile concentrations of blood lead for children ages 1 to 5 years for each NHANES survey period.

Indicator B2 gives the median concentrations of blood lead for children ages 1 to 5 years for 2005-2008, stratified by race/ethnicity and family income. Table B1a gives the median and 95th percentile concentrations of blood lead for children ages 1 to 17 years for 2005-2008, stratified by age group. Table B2a gives the 95th percentile concentrations of blood lead for children ages 1 to 5 years for 2005-2008, stratified by race/ethnicity and family income. Table B2b gives the median concentrations of blood lead for children ages 1 to 5 years for 1991-1994, stratified by race/ethnicity and family income. The survey data were weighted to account for the complex multi-stage, stratified, clustered sampling design.

Data Summary

[i] Centers for Disease Control and Prevention. 2009. Fourth National Report on Human Exposure to Environmental Chemicals. Atlanta, GA. Available at: www.cdc.gov/exposurereport.

Indicator	B1. Lead in children ages 1 to 5 years: Median and 95th percentile concentrations in blood, 1976-2008. B2. Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2005-2008.							
Time Period	1976-2008							
Data	Blood lead							
Years	1976-1980	1988-1991	1991-1994	1999-2000	2001-2002	2003-2004	2005-2006	2007-2008
Limits of Detection (µg/dL)*	Not reported	1	1	0.3	0.3	0.3	0.3 or 0.25	0.25
Number of Non-missing Values**	2,345	2,203	2,367	723	898	911	968	817
Number of Missing Values	1,425	805	347	362	432	356	442	414
Percentage Below Limit of Detection***		4	8	0	1	0	0	0

* The Limit of Detection (LOD) is defined as the level at which the measurement has a 95% probability of being greater than zero.
**Non-missing values include those below the analytical LOD, which are reported as LOD/√2.
***This percentage is survey-weighted using the NHANES survey weights for the given period.

Overview of Data Files

The following files are needed to calculate this indicator. All these files together with the survey documentation and SAS programs for reading in the data are available at the NHANES website: www.cdc.gov/nchs/nhanes.htm.

- NHANES II: Hematology and Biochemistry file DU5411.txt. This text file contains the measured blood lead (N2LB0409), age in years (N2LB0190), the survey weight for lead data (N2LB0060), the pseudo-stratum (N2LB0324), and the pseudo-PSU (N2LB0326).

- NHANES III: Laboratory file LAB.DAT. This text file contains the measured blood lead (PBP), age in months (MXPAXTMR), the laboratory survey weights (WTPFEX1 for Phase 1 and WTPFEX2 for Phase 2), the pseudo-stratum codes (SDPSTRA1 for Phase 1 and SDPSTRA2 for Phase 2), and the pseudo-PSU codes (SDPPSU1 for Phase 1 and SDPPSU2 for Phase 2).

- NHANES 1999-2000: Demographic file demo.xpt. Laboratory file lab06.xpt. The demographic file demo.xpt is a SAS transport file that contains the subject identifier (SEQN), age (RIDAGEYR), laboratory survey weight (WTMEC2YR), pseudo-stratum (SDMVSTRA) and the pseudo-PSU (SDMVPSU). The laboratory file lab06.xpt contains SEQN and the blood lead (LBXBPB). The two files are merged using the common variable SEQN.

- NHANES 2001-2002: Demographic file demo_b.xpt. Laboratory file l06_b.xpt. The demographic file demo_b.xpt is a SAS transport file that contains the subject identifier (SEQN), age (RIDAGEYR), laboratory survey weight (WTMEC2YR), pseudo-stratum (SDMVSTRA) and the pseudo-PSU (SDMVPSU). The laboratory file l06_b.xpt contains SEQN and the blood lead (LBXBPB). The two files are merged using the common variable SEQN.

- NHANES 2003-2004: Demographic file demo_c.xpt. Laboratory file l06bmt_c.xpt. The demographic file demo_c.xpt is a SAS transport file that contains the subject identifier (SEQN), age (RIDAGEYR) and the laboratory survey weight (WTMEC2YR), pseudo-stratum (SDMVSTRA) and the pseudo-PSU (SDMVPSU). The laboratory file l06bmt_c.xpt contains SEQN and the blood lead (LBXBPB). The two files are merged using the common variable SEQN.

- NHANES 2005-2006: Demographic file demo_d.xpt. Laboratory file pbcd_d.xpt. The demographic file demo_d.xpt is a SAS transport file that contains the subject identifier (SEQN), age (RIDAGEYR), race/ethnicity (RIDRETH1), poverty income ratio (INDFMPIR), laboratory survey weight (WTMEC2YR), pseudo-stratum (SDMVSTRA), and the pseudo-PSU (SDMVPSU). The laboratory file pbcd_d.xpt contains SEQN and the blood lead (LBXBPB). The two files are merged using the common variable SEQN.

- NHANES 2007-2008: Demographic file demo_e.xpt. Laboratory file pbcd_e.xpt. The demographic file demo_e.xpt is a SAS transport file that contains the subject identifier (SEQN), age (RIDAGEYR), race/ethnicity (RIDRETH1), poverty income ratio (INDFMPIR), laboratory survey weight (WTMEC2YR), pseudo-stratum (SDMVSTRA), and the pseudo-PSU (SDMVPSU). The laboratory file pbcd_e.xpt contains SEQN and the blood lead (LBXBPB). The two files are merged using the common variable SEQN.

National Health and Nutrition Examination Surveys (NHANES)

Since the 1970s, the National Center for Health Statistics, a division of the Centers for Disease Control and Prevention, has conducted the National Health and Nutrition Examination Surveys (NHANES), a series of U.S. national surveys of the health and nutrition status of the noninstitutionalized civilian population. The National Center for Environmental Health at CDC measures environmental chemicals in blood and urine samples collected from NHANES participants. Indicators B1 and B2 use blood lead measurements in children ages 5 and under. NHANES II (1976-1980) included blood lead data for children from six months to 5 years. NHANES III (1988-1994) and the NHANES 1999-2000, 2001-2002, 2003-2004, 2005-2006, and 2007-2008 surveys included blood lead data for children ages 1 to 5 years. The NHANES data were obtained from the NHANES website: http://www.cdc.gov/nchs/nhanes.htm Following the CDC recommended approach, values below the analytical limit of detection (LOD) were replaced by LOD/√2.[ii]

[ii] See Hornung RW, Reed LD. 1990. Estimation of average concentration in the presence of nondetectable values. *Appl Occup Environ Hyg* 5:46–51.

The NHANES use a complex multi-stage, stratified, clustered sampling design. Certain demographic groups were deliberately over-sampled, including Mexican-Americans and Blacks. Oversampling is performed to increase the reliability and precision of estimates of health status indicators for these population subgroups. The publicly released data includes survey weights to adjust for the over-sampling, non-response, and non-coverage. The statistical analyses used the laboratory survey weights (WTMEC2YR) to re-adjust the blood lead data to represent the national population.

Race/Ethnicity and Family Income

For Indicator B2, the percentiles were calculated for demographic strata defined by the combination of race/ethnicity and family income.

The family income was characterized based on the INDFMPIR variable, which is the ratio of the family income to the poverty level. The National Center for Health Statistics used the U.S. Census Bureau Current Population Survey to define the family units, and the family income for the respondent was obtained during the interview. The U.S. Census Bureau defines annual poverty level money thresholds varying by family size and composition. The poverty income ratio (PIR) is the family income divided by the poverty level for that family. Family income was stratified into the following groups:

- Below Poverty Level: PIR < 1
- Between 100% and 200% of Poverty Level: 1 ≤ PIR ≤ 2
- Above 200% of Poverty level: PIR > 2
- Above Poverty Level: PIR ≥ 1 (combines the previous two groups)
- Unknown Income: PIR is missing

Race/ethnicity was characterized using the RIDRETH1 variable. The possible values of this variable are:

- 1. Mexican American
- 2. Other Hispanic
- 3. Non-Hispanic White
- 4. Non-Hispanic Black
- 5. Other Race – Including Multi-racial
- "." Missing

Category 5 includes: all Non-Hispanic single race responses other than White or Black; and multi-racial responses.

For indicator B2, the RIDRETH1 categories 2, 5, and missing were combined into a single "Other" category. This produced the following categories:

- White non-Hispanic: RIDRETH1 = 3

- Black non-Hispanic: RIDRETH1 = 4
- Mexican-American: RIDRETH1 = 1
- Other: RIDRETH1 = 2 or 5 or missing

The "Other" category includes Asian non-Hispanic; Native American non-Hispanic; Hispanic other than Mexican-American; those reporting multi-racial; and those with a missing value for race/ethnicity.

Calculation of Indicator

Indicator B1 is the median and 95th percentile for blood lead in children of ages 1 to 5 years. Table B1a is the median and 95th percentile for blood lead in children of ages 1 to 17 years, stratified by age group. Indicator B2 is the median of blood lead in children of ages 1 to 5 years stratified by race/ethnicity and family income. Table B2a is the 95th percentile of blood lead in children of ages 1 to 5 years stratified by race/ethnicity and family income. Table B2b is the median of blood lead in children of ages 1 to 5 years, stratified by race/ethnicity and family income for 1991-1994. The median is the estimated concentration such that 50% of all noninstitutionalized civilian children ages 1 to 5 years during the survey period have blood lead concentrations below this level. The 95th percentile is the estimated concentration such that 95% of all noninstitutionalized civilian children ages 1 to 5 years during the survey period have blood lead concentrations below this level.

To simply demonstrate the calculations, we will use the NHANES 2007-2008 blood lead values for children ages 1 to 5 years as an example. We have rounded all the numbers to make the calculations easier:

We begin with all the non-missing NHANES 2007-2008 blood lead values for children ages 1 to 5 years. Assume for the sake of simplicity that valid data on blood lead were available for every sampled child. Each sampled child has an associated survey weight WTMEC2YR that estimates the annual number of U.S. children represented by that sampled child. For example, the lowest blood lead measurement for a child between 1 and 5 years of age is 0.39 μg/dL with a survey weight of 60,000, and so represents 60,000 children between 1 and 5 years of age. The total of the survey weights for the sampled children equals 20 million, the total U.S. population of children between 1 and 5 years of age. The second lowest measurement is 0.42 μg/dL with a survey weight of 10,000, and so represents another 10,000 U.S. children between 1 and 5 years of age. The highest measurement was 31 μg/dL, with a survey weight of 8,000, and so represents another 8,000 U.S. children between 1 and 5 years of age.

To calculate the median, we can use the survey weights to expand the data to the entire U.S. population of 20 million children ages 1 to 5 years. We have 60,000 values of 0.39 μg/dL from the lowest measurement, 10,000 values of 0.42 μg/dL from the second lowest measurement, and so on, up to 8,000 values of 31 μg/dL from the highest measurement. Arranging these 20 million values in increasing order, the 10 millionth value is 1.4 μg/dL. Since half of the values are below 1.4 and half of the values are above 1.4, the median equals 1.4 μg/dL. To calculate the 95th

percentile, note that 95% of 20 million equals 19 million. The 19 millionth value is 4.1 μg/dL. Since 95% of the values are below 4.1, the 95th percentile equals 4.1 μg/dL.

In reality, the calculations need to take into account that blood lead measurements were not available for every respondent, and to use exact rather than rounded numbers. There were blood lead measurements for only 817 of the 1,231 sampled children ages 1 to 5 years. The survey weights for all 1,231 sampled children add up to 20.6 million, the U.S. population of children ages 1 to 5 years. The survey weights for the 817 sampled children with blood lead data add up to 13.7 million. Thus the available data represent 13.7 million values and so represent only 66% of the U.S. population of children ages 1 to 5 years. The median and 95th percentiles are given by the 6.85 millionth (50% of 13.7 million) and 13.02 millionth (95% of 13.7 million) U.S. child's value. These calculations assume that the sampled children with valid blood lead data are representative of the children without valid blood lead data.

Equations

These percentile calculations can also be given as the following mathematical equations, which are based on the default percentile calculation formulas from Statistical Analysis System (SAS) software. Exclude all missing blood lead values. Suppose there are n children of ages 1 to 5 years with valid blood lead values. Arrange the blood lead concentrations in increasing order (including tied values) so that the lowest concentration is x(1) with a survey weight of w(1), the second lowest concentration is x(2) with a survey weight of w(2), …, and the highest concentration is x(n) with a survey weight of w(n).

1. Sum all the survey weights to get the total weight W:

$$W = \Sigma[1 \le i \le n]\, w(i)$$

2. Find the largest number i so that the total of the weights for the i lowest values is less than or equal to W/2.

$$\Sigma[j \le i]\, w(j) \le W/2 < \Sigma[j \le i + 1]\, w(j)$$

3. Calculate the median using the results of the second step. We either have

$$\Sigma[j \le i]\, w(j) = W/2 < \Sigma[j \le i + 1]\, w(j)$$

or

$$\Sigma[j \le i]\, w(j) < W/2 < \Sigma[j \le i + 1]\, w(j)$$

In the first case we define the median as the average of the i'th and i + 1'th values:

$$\text{Median} = [x(i) + x(i + 1)]/2 \text{ if } \Sigma[j \le i]\, w(j) = W/2$$

In the second case we define the median as the i + 1'th value:

$$\text{Median} = x(i + 1) \text{ if } \Sigma[j \leq i]\, w(j) < W/2$$

(The estimated median does not depend upon how the tied values of x(j) are ordered).

A similar calculation applies to the 95th percentile. The first step to calculate the sum of the weights, W, is the same. In the second step, find the largest number i so that the total of the weights for the i lowest values is less than or equal to 0.95W.

$$\Sigma[j \leq i]\, w(j) \leq 0.95W < \Sigma[j \leq i + 1]\, w(j)$$

In the third step we calculate the 95th percentile using the results of the second step. We either have

$$\Sigma[j \leq i]\, w(j) = 0.95W < \Sigma[j \leq i + 1]\, w(j)$$

or

$$\Sigma[j \leq i]\, w(j) < 0.95W < \Sigma[j \leq i + 1]\, w(j)$$

In the first case we define the 95th percentile as the average of the i'th and i + 1'th values:

$$95^{th} \text{ Percentile} = [x(i) + x(i + 1)]/2 \text{ if } \Sigma[j \leq i]\, w(j) = 0.95W$$

In the second case we define the 95th percentile as the i + 1'th value:

$$95^{th} \text{ Percentile} = x(i + 1) \text{ if } \Sigma[j \leq i]\, w(j) < 0.95W$$

Relative Standard Error

The uncertainties of the median and 95th percentile values were calculated using a revised version of the CDC method given in CDC 2005,[iii] Appendix C, and the SAS® program provided by CDC. The method uses the Clopper-Pearson binomial confidence intervals adapted for complex surveys by Korn and Graubard (see Korn and Graubard, 1999,[iv] p. 65). The following text is a revised version of the Appendix C.

> **Step 1:** Use SAS® Proc Univariate to obtain a point estimate P_{SAS} of the percentile value. Use the Weight option to assign the exact correct sample weight for each chemical result.
>
> **Step 2:** Use SUDAAN® Proc Descript with Taylor Linearization DESIGN = WR (i.e., sampling with replacement) and the proper sampling weight to estimate the proportion (p) of subjects with results less than and not equal to the percentile estimate P_{SAS} obtained in Step 1 and to obtain the standard

[iii] CDC Third National Report on Human Exposure to Environmental Chemicals. 2005
[iv] Korn E. L., Graubard B. I. 1999. *Analysis of Health Surveys.* Wiley.

error (se_p) associated with this proportion estimate. Compute the degrees-of-freedom adjusted effective sample size

$$n_{df} = (t_{num}/t_{denom})^2 \, p(1 - p)/(se_p{}^2)$$

where t_{num} and t_{denom} are 0.975 critical values of the Student's t distribution with degrees of freedom equal to the sample size minus 1 and the number of PSUs minus the number of strata, respectively. Note: the degrees of freedom for t_{denom} can vary with the demographic sub-group of interest.

Step 3: After obtaining an estimate of p (i.e., the proportion obtained in Step 2), compute the Clopper-Pearson 95% confidence interval ($P_L(x,n_{df})$, $P_U(x,n_{df})$) as follows:

$$P_L(x,n_{df}) = v_1F_{v1,v2}\,(0.025)/(v_2 + v_1F_{v1,v2}(0.025))$$
$$P_U(x,n_{df}) = v_3F_{v3,v4}\,(0.975)/(v_4 + v_3F_{v3,v4}(0.975))$$

where x is equal to p times n_{df}, $v_1 = 2x$, $v_2 = 2(n_{df} - x + 1)$, $v_3 = 2(x + 1)$, $v_4 = 2(n_{df} - x)$, and $F_{d1,d2}(\beta)$ is the β quantile of an F distribution with d1 and d2 degrees of freedom. (Note: If n_{df} is greater than the actual sample size or if p is equal to zero, then the actual sample size should be used.) This step will produce a lower and an upper limit for the estimated proportion obtained in Step 2.

Step 4: Use SAS Proc Univariate (again using the Weight option to assign weights) to determine the chemical percentile values P_{CDC}, L_{CDC} and U_{CDC} that correspond to the proportion p obtained in Step 2 and its lower and upper limits obtained in Step 3. Do not round the values of p and the lower and upper limits. For example, if p = 0.4832, then P_{CDC} is the 48.32'th percentile value of the chemical. The alternative percentile estimates P_{CDC} and P_{SAS} are not necessarily equal.

Step 5: Use the confidence interval from Step 4 to estimate the standard error of the estimated percentile P_{CDC}:

$$\text{Standard Error } (P_{CDC}) = (U_{CDC} - L_{CDC}) / (2t_{denom})$$

Step 6: Use the estimated percentile P_{CDC} and the standard error from Step 4 to estimate the relative standard error of the estimated percentile P_{CDC}:

$$\text{Relative Standard Error (\%)} = [\text{Standard Error } (P_{CDC}) / P_{CDC}] \times 100\%$$

The tabulated estimated percentile is the value of P_{SAS} given in Step 1. The relative standard error is given in Step 6, using P_{CDC} and its standard error.

The relative standard error depends upon the survey design. For this purpose, the public release version of NHANES includes the variables SDMVSTRA and SDMVPSU, which are the Masked Variance Unit pseudo-stratum and pseudo-primary sampling unit (pseudo-PSU). For approximate variance estimation, the survey design can be approximated as being a stratified random sample with replacement of the pseudo-PSUs from each pseudo-stratum; the true stratum and PSU variables are not provided in the public release version to protect confidentiality.

Percentiles with a relative standard error less than 30% were treated as being reliable and were tabulated. Percentiles with a relative standard error greater than or equal to 30% but less than 40% were treated as being unstable; these values were tabulated but were flagged to be interpreted with caution. Percentiles with a relative standard error greater than or equal to 40%,

or without an estimated relative standard error, were treated as being unreliable; these values were not tabulated and were flagged as having a large uncertainty.

Questions and Comments

Questions regarding these methods, and suggestions to improve the description of the methods, are welcome. Please use the "Contact Us" link at the bottom of any page in the America's Children and the Environment website.

Statistical Comparisons

Statistical analyses of the percentiles were used to determine whether the differences between percentiles for different demographic groups were statistically significant. For these analyses, the percentiles and their standard errors were calculated for each combination of age group, sex, income group (below poverty, at or above poverty, unknown income), and race/ethnicity group using the method described in the "Relative Standard Error" section. In the notation of that section, the percentile and standard error are the values of P_{CDC} and Standard Error (P_{CDC}), respectively. These calculated standard errors account for the survey weighting and design and, for women, for the age-specific birthrate.

Using a weighted linear regression model, the percentile was assumed to be the sum of explanatory terms for age, sex, income and/or race/ethnicity and a random error term; the error terms were assumed to be approximately independent and normally distributed with a mean of zero and a variance equal to the square of the standard error. Using this model, the difference in the value of a percentile between different demographic groups is statistically significant if the difference between the corresponding sums of explanatory terms is statistically significantly different from zero. A p-value at or below 0.05 implies that the difference is statistically significant at the 5% significance level. No adjustment is made for multiple comparisons.

For each type of comparison, we present unadjusted and adjusted analyses. The unadjusted analyses directly compare a percentile between different demographic groups. The adjusted analyses add other demographic explanatory variables to the statistical model and use the statistical model to account for the possible confounding effects of these other demographic variables. For example, the unadjusted race/ethnicity comparisons use and compare the percentiles between different race/ethnicity pairs. The adjusted race/ethnicity comparisons use the percentiles for each age/sex/income/race/ethnicity combination. The adjusted analyses add age, sex, and income terms to the statistical model and compare the percentiles between different race/ethnicity pairs after accounting for the effects of the other demographic variables. For example, if White non-Hispanics tend to have higher family incomes than Black non-Hispanics, and if the blood lead level strongly depends on family income only, then the unadjusted differences between these two race/ethnicity groups would be significant but the adjusted difference (taking into account income) would not be significant.

Comparisons between pairs of race/ethnicity groups are shown in Tables 1 and 2 for children ages 1 to 5 years. In Table 1, for the unadjusted "All incomes" comparisons, the only explanatory variables are terms for each race/ethnicity group. For these unadjusted comparisons, the statistical tests compare the percentiles for each pair of race/ethnicity groups. For the adjusted "All incomes (adjusted for age, sex, income)" comparisons, the explanatory variables are terms for each race/ethnicity group together with terms for each age, sex, and income group. For these adjusted comparisons, the statistical test compares the pair of race/ethnicity groups after accounting for any differences in the age, sex and income distributions between the race/ethnicity groups.

In Table 1, for the unadjusted "Below Poverty Level" and "At or Above Poverty Level" comparisons, the only explanatory variables are terms for each of the twelve

race/ethnicity/income combinations (combinations of four race/ethnicity groups and three income groups). For example, in row 1, the p-value for "Below Poverty Level" compares White non-Hispanics below the poverty level with Black non-Hispanics below the poverty level. The same set of explanatory variables are used in Table 2 for the unadjusted comparisons between one race/ethnicity group below the poverty level and the same or another race/ethnicity group at or above the poverty level. The corresponding adjusted analyses include extra explanatory variables for age and sex, so that race/ethnicity/income groups are compared after accounting for any differences due to age or sex.

Additional comparisons are shown in Table 3 for children ages 1 to 5 years. The AGAINST = "income" unadjusted p-value compares the blood lead levels for those below poverty level with those at or above poverty level, using the explanatory variables for the three income groups (below poverty, at or above poverty, unknown income). The adjusted p-value includes adjustment terms for age, sex, and race/ethnicity in the model. The AGAINST = "yearnum" p-value examines whether the linear trend in blood lead levels is statistically significant (using the percentiles for each NHANES period regressed against the midpoint of that period); the adjusted model for trend adjusts for demographic changes in the populations from year to year by including terms for age, sex, income, and race/ethnicity.

Table 4 shows comparisons between blood lead levels in children ages 1 to 5 years in 1991-1994 and the blood lead levels in children ages 1 to 5 years in 2005-2008. The AGAINST = "yearnum" p-value examines whether the change in the percentiles is statistically significant (using the percentiles for the periods 1991-1994 and 2005-2008 regressed against the midpoints of those two periods); the adjusted model adjusts for demographic changes in the populations from year to year by including terms for age, sex, income, and race/ethnicity. The rows for SUBSET is not missing show the p-values for different race/ethnicity groups.

The age groups used were 1, 2, 3, 4, and 5.

For more details on these statistical analyses, see the memorandum by Cohen (2010).[v]

Table 1. Statistical significance tests comparing the percentiles of blood lead levels in children ages 1 to 5 years, between pairs of race/ethnicity groups, for 2005-2008.

				P-VALUES					
Variable	**Percentile**	**RACE1**	**RACE2**	**All incomes**	**All incomes (adjusted for age, sex, income)**	**Below Poverty Level**	**Below Poverty Level (adjusted for age, sex)**	**At or Above Poverty Level**	**At or Above Poverty Level (adjusted for age, sex)**
lead	50	White non-Hispanic	Black non-Hispanic	< 0.0005	< 0.0005	< 0.0005	< 0.0005	0.021	0.003
lead	50	White non-	Mexican-	0.469	0.007	0.443	0.075	0.908	0.115

[v] Cohen, J. 2010. *Selected statistical methods for testing for trends and comparing years or demographic groups in ACE NHIS and NHANES indicators.* Memorandum submitted to Dan Axelrad, EPA, 21 March, 2010.

				P-VALUES					
Variable	Percentile	RACE1	RACE2	All incomes	All incomes (adjusted for age, sex, income)	Below Poverty Level	Below Poverty Level (adjusted for age, sex)	At or Above Poverty Level	At or Above Poverty Level (adjusted for age, sex)
		Hispanic	American						
lead	50	White non-Hispanic	Other	0.660	0.204	0.104	0.372	0.604	0.235
lead	50	Black non-Hispanic	Mexican-American	< 0.0005	< 0.0005	< 0.0005	< 0.0005	0.042	< 0.0005
lead	50	Black non-Hispanic	Other	0.010	< 0.0005	0.050	< 0.0005	0.039	< 0.0005
lead	50	Mexican-American	Other	0.948	0.258	0.183	0.567	0.581	0.773
lead	95	White non-Hispanic	Black non-Hispanic	0.002	< 0.0005	0.085	< 0.0005	0.155	< 0.0005
lead	95	White non-Hispanic	Mexican-American	0.783	0.005	0.407	0.026	0.904	0.483
lead	95	White non-Hispanic	Other	0.206	< 0.0005	0.831	0.077	0.809	< 0.0005
lead	95	Black non-Hispanic	Mexican-American	0.002	< 0.0005	0.003	< 0.0005	0.032	< 0.0005
lead	95	Black non-Hispanic	Other	0.050	< 0.0005	0.054	< 0.0005	0.348	< 0.0005
lead	95	Mexican-American	Other	0.243	< 0.0005	0.592	< 0.0005	0.700	< 0.0005

Table 2. Statistical significance tests comparing the percentiles of blood lead levels in children ages 1 to 5 years, between pairs of race/ethnicity/income groups at different income levels, for 2005-2008.

				P-VALUES	
Variable	Percentile	RACEINC1	RACEINC2	Unadjusted	Adjusted (for age, sex)
lead	50	White non-Hispanic, < PL	White non-Hispanic, ≥ PL	0.355	0.002
lead	50	White non-Hispanic, < PL	Black non-Hispanic, ≥ PL	0.522	0.704
lead	50	White non-Hispanic, < PL	Mexican-American, ≥ PL	0.412	< 0.0005
lead	50	White non-Hispanic, < PL	Other, ≥ PL	0.270	< 0.0005
lead	50	Black non-Hispanic, < PL	White non-Hispanic, ≥ PL	< 0.0005	< 0.0005
lead	50	Black non-Hispanic, < PL	Black non-Hispanic, ≥ PL	< 0.0005	< 0.0005
lead	50	Black non-Hispanic, < PL	Mexican-American, ≥ PL	< 0.0005	< 0.0005
lead	50	Black non-Hispanic, < PL	Other, ≥ PL	< 0.0005	< 0.0005
lead	50	Mexican-American, < PL	White non-Hispanic, ≥ PL	0.001	0.162
lead	50	Mexican-American, < PL	Black non-Hispanic, ≥ PL	0.937	0.126
lead	50	Mexican-American, < PL	Mexican-American, ≥ PL	0.007	0.010
lead	50	Mexican-American, < PL	Other, ≥ PL	0.015	0.013
lead	50	Other, < PL	White non-Hispanic, ≥ PL	0.008	0.126
lead	50	Other, < PL	Black non-Hispanic, ≥ PL	0.195	0.557
lead	50	Other, < PL	Mexican-American, ≥ PL	0.012	0.016
lead	50	Other, < PL	Other, ≥ PL	0.009	0.030
lead	95	White non-Hispanic, < PL	White non-Hispanic, ≥ PL	0.187	< 0.0005
lead	95	White non-Hispanic, < PL	Black non-Hispanic, ≥ PL	0.519	0.008

				P-VALUES	
Variable	**Percentile**	**RACEINC1**	**RACEINC2**	**Unadjusted**	**Adjusted (for age, sex)**
lead	95	White non-Hispanic, < PL	Mexican-American, ≥ PL	0.145	< 0.0005
lead	95	White non-Hispanic, < PL	Other, ≥ PL	0.264	< 0.0005
lead	95	Black non-Hispanic, < PL	White non-Hispanic, ≥ PL	0.001	< 0.0005
lead	95	Black non-Hispanic, < PL	Black non-Hispanic, ≥ PL	0.005	< 0.0005
lead	95	Black non-Hispanic, < PL	Mexican-American, ≥ PL	< 0.0005	< 0.0005
lead	95	Black non-Hispanic, < PL	Other, ≥ PL	0.002	< 0.0005
lead	95	Mexican-American, < PL	White non-Hispanic, ≥ PL	0.213	< 0.0005
lead	95	Mexican-American, < PL	Black non-Hispanic, ≥ PL	0.667	0.407
lead	95	Mexican-American, < PL	Mexican-American, ≥ PL	0.025	< 0.0005
lead	95	Mexican-American, < PL	Other, ≥ PL	0.468	< 0.0005
lead	95	Other, < PL	White non-Hispanic, ≥ PL	0.297	< 0.0005
lead	95	Other, < PL	Black non-Hispanic, ≥ PL	0.719	< 0.0005
lead	95	Other, < PL	Mexican-American, ≥ PL	0.245	< 0.0005
lead	95	Other, < PL	Other, ≥ PL	0.394	< 0.0005

Table 3. Other statistical significance tests comparing the percentiles of blood lead levels in children ages 1 to 5 years, for 2005-2008 (trends from 1988-2008 and from 1999-2008).

					P-VALUES	
Variable	**Percentile**	**From**	**To**	**Against**	**Unadjusted**	**Adjusted***
lead	50	2005	2008	income	< 0.0005	< 0.0005
lead	50	1988	2008	yearnum	< 0.0005	< 0.0005
lead	50	1999	2008	yearnum	< 0.0005	< 0.0005
lead	95	2005	2008	income	0.043	< 0.0005
lead	95	1988	2008	yearnum	< 0.0005	< 0.0005
lead	95	1999	2008	yearnum	< 0.0005	< 0.0005

*For AGAINST = "income," the p-values are adjusted for age, sex, and race/ethnicity.
For AGAINST = "yearnum," the p-values are adjusted for age, sex, race/ethnicity, and income.

Table 4. Statistical significance tests comparing the percentiles of blood lead levels in children ages 1 to 5 years between 1991-1994 and 2005-2008.

						P-VALUES	
Variable	**Percentile**	**From**	**To**	**Against**	**Subset**	**Unadjusted**	**Adjusted***
lead	50	1991-1994	2005-2008	yearnum		< 0.0005	< 0.0005
lead	95	1991-1994	2005-2008	yearnum		< 0.0005	< 0.0005
lead	50	1991-1994	2005-2008	yearnum	White non-Hispanic	< 0.0005	< 0.0005
lead	95	1991-1994	2005-2008	yearnum	White non-Hispanic	< 0.0005	< 0.0005
lead	50	1991-1994	2005-2008	yearnum	Black non-Hispanic	< 0.0005	< 0.0005
lead	95	1991-1994	2005-2008	yearnum	Black non-Hispanic	< 0.0005	< 0.0005
lead	50	1991-1994	2005-2008	yearnum	Mexican-American	< 0.0005	< 0.0005
lead	95	1991-1994	2005-2008	yearnum	Mexican-American	< 0.0005	< 0.0005
lead	50	1991-1994	2005-2008	yearnum	Other	0.001	0.012
lead	95	1991-1994	2005-2008	yearnum	Other	< 0.0005	0.001

*For AGAINST = "yearnum,," where SUBSET is not missing, the p-values are adjusted for age, sex, race/ethnicity, and income.

For AGAINST = "yearnum,," where SUBSET is missing, the p-values are adjusted for age, sex, and income.