

## 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 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 lead-contaminated house dust.<sup>1,2</sup> 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.<sup>3-5</sup> 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.<sup>6</sup> 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.<sup>7</sup> New lead dust hazards occur when lead in house paint is released during home renovation and remodeling activities.<sup>8,9</sup>

Two other contributors to lead in house dust are lead-contaminated soil and airborne lead.<sup>10-13</sup> Known sources of lead in soil include historical airborne emissions of leaded gasoline, emissions from industrial sources such as smelters, and lead-based paint.<sup>14,15</sup> 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.<sup>15</sup>

Lead-contaminated house dust is not the only source of childhood lead exposure. Direct contact with lead-contaminated soil,<sup>13</sup> ingestion of lead-based paint chips,<sup>16</sup> 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.<sup>5,17,18</sup> 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.<sup>17</sup> 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;<sup>5,19</sup> jewelry;<sup>20</sup> tobacco smoke;<sup>21</sup> imported candies, spices, and condiments;<sup>5,22</sup> and imported folk remedies.<sup>23,24</sup>

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.<sup>25</sup>

Childhood blood lead levels in the United States differ across groups in the population, such as those defined by socioeconomic status and race/ethnicity.<sup>26</sup> Children living in poverty and Black non-Hispanic children tend to have higher blood lead levels<sup>27</sup> and higher levels of lead-contaminated dust in the home<sup>6</sup> 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.<sup>6,28</sup> Blood lead levels may vary by nutritional status: conditions such as iron deficiency have been associated with higher blood lead levels in children.<sup>15</sup> 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.<sup>27,29</sup>

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,<sup>30</sup> and to the reduction in the number of homes with lead-based paint hazards.<sup>7</sup> 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.<sup>5</sup> 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.<sup>5</sup> However, because trace levels of lead may be present in these products, normal use may still result in lead exposure.<sup>5</sup>

The National Toxicology Program (NTP) has concluded that childhood lead exposure is associated with reduced cognitive function.<sup>31</sup> Children with higher blood lead levels generally have lower scores on IQ tests<sup>32-38</sup> and reduced academic achievement.<sup>31</sup> 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).<sup>31</sup> Studies have reported that lead exposure in children may contribute to decreased attention,<sup>38-43</sup> hyperactivity-impulsivity,<sup>44</sup> and increased likelihood of attention-deficit/hyperactivity disorder.<sup>44-52</sup> Other adverse behavioral outcomes that have been associated with childhood lead exposure in some studies include conduct disorders,<sup>53,54</sup> increased risks of juvenile delinquency and antisocial behaviors,<sup>55-57</sup> higher total arrest rates, and arrest rates for violent crimes in early adulthood.<sup>58,59</sup> 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.<sup>60,61</sup>

Mothers who are exposed to lead can transfer lead to the fetus during pregnancy and to the child while breast feeding.<sup>62,63</sup> The NTP has concluded that there is “limited evidence” that prenatal lead exposure is associated with cognitive and behavioral effects in children.<sup>31</sup> 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.<sup>64</sup>

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.<sup>15</sup> 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,<sup>36</sup> 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.<sup>65,66</sup>

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.<sup>67,68</sup> Childhood blood lead concentrations are also inversely associated with intellectual functioning in young adulthood.<sup>69</sup> 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.<sup>25,70,71</sup> Finally, childhood exposures to lead may contribute to a variety of neurological disorders and neurobehavioral effects in later life.<sup>25,71-73</sup>

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

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.

### **Indicator B1: Lead in children ages 1 to 5 years: Median and 95th percentile concentrations in blood, 1976–2010**

### **Indicator B2: Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2007–2010**

**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 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*.<sup>77</sup>

## **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.<sup>15</sup> All values are reported as micrograms of lead per deciliter of blood ( $\mu\text{g}/\text{dL}$ ).

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 2009–2010, NHANES collected lead biomonitoring data for 8,793 individuals ages 1 year and older, including 836 children ages 1 to 5. Lead was detected in 100% of all individuals sampled. The median blood lead level among all NHANES participants in 2009–2010 was 1.1  $\mu\text{g}/\text{dL}$  and the 95<sup>th</sup> percentile was 3.3  $\mu\text{g}/\text{dL}$ .

## **Data Presented in the Indicators**

Indicator B1 presents median and 95<sup>th</sup> percentile concentrations of lead in blood over time for children ages 1 to 5 years, using NHANES data from 1976–2010.

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 2007–2008 and 2009–2010.

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 95<sup>th</sup> percentiles of blood lead by race/ethnicity and income are presented in the data tables.

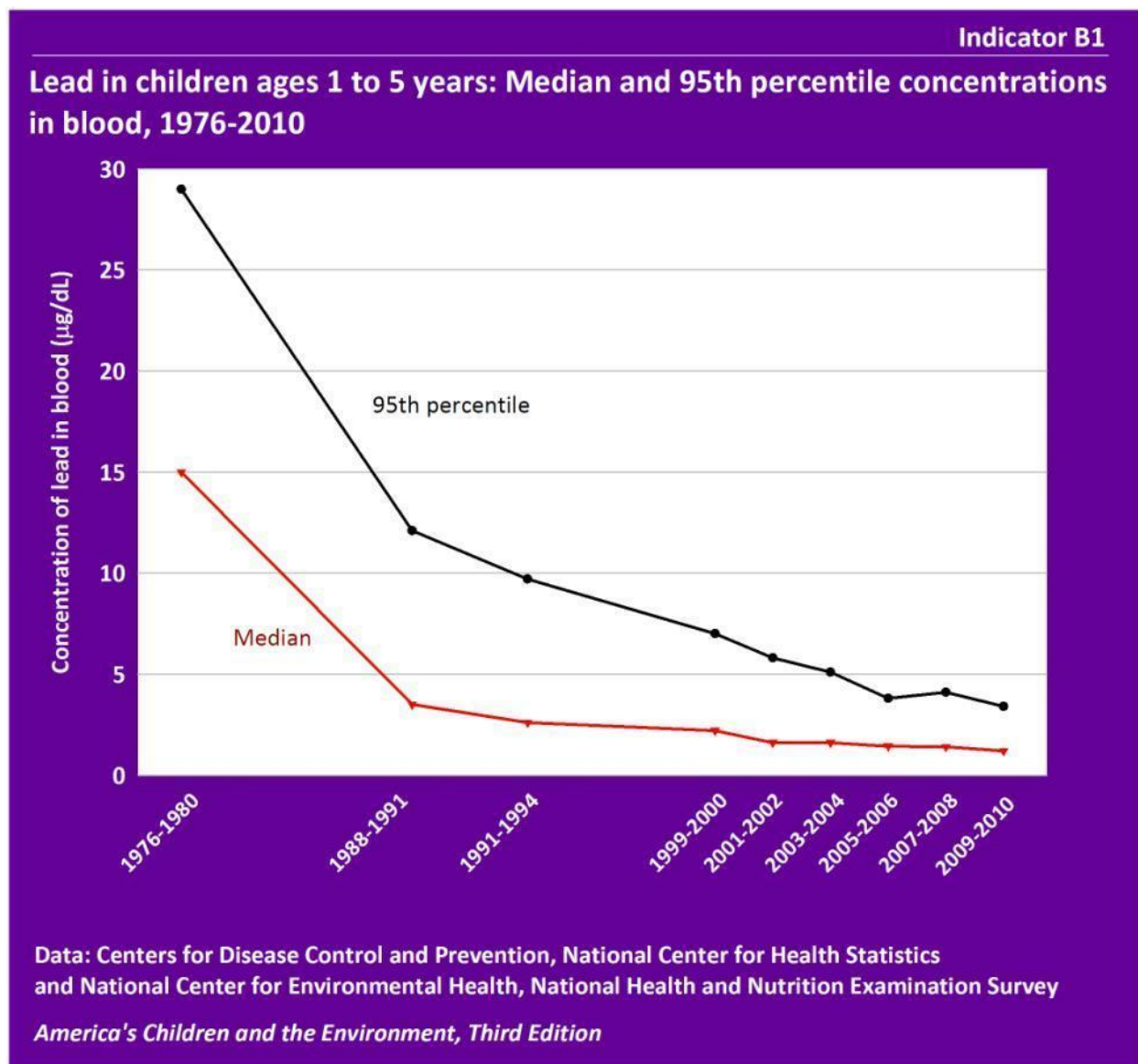
Four race/ethnicity groups are presented in Indicator B2: White non-Hispanic, Black non-Hispanic, Mexican-American, and “All Other Races/Ethnicities.” The “All Other Races/Ethnicities” category includes all other races and ethnicities not specified, together with those individuals who report more than 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 95<sup>th</sup> 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 95<sup>th</sup> 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 “95<sup>th</sup> percentile,” and information on the statistical significance testing applied to these indicators.



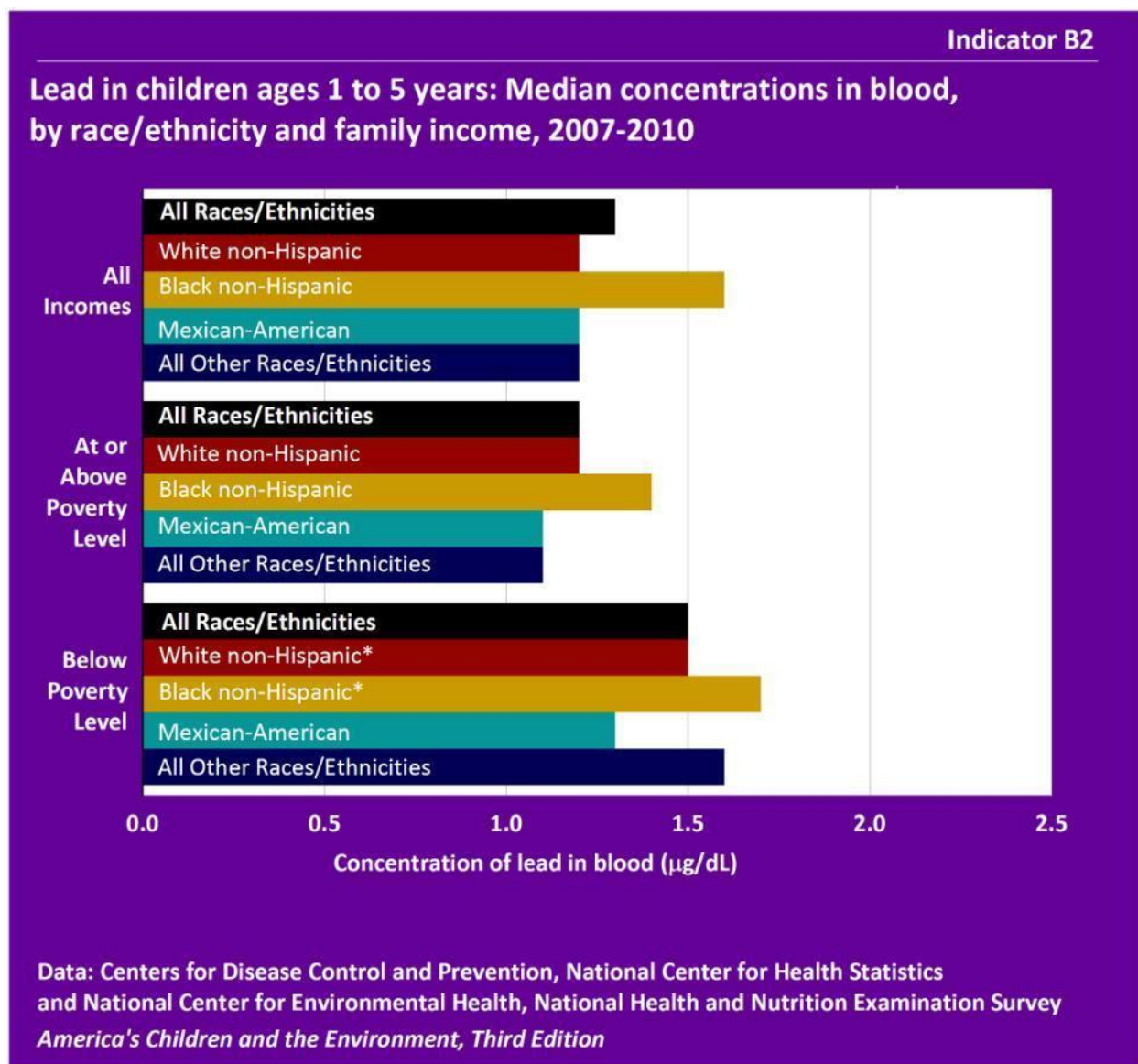
**Data characterization**

- 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 µg/dL in 1976–1980 to 1.2 µg/dL in 2009–2010, a decrease of 92%.
- The concentration of lead in blood at the 95<sup>th</sup> percentile in children ages 1 to 5 years dropped from 29 µg/dL in 1976–1980 to 3.4 µg/dL in 2009–2010, a decrease of 88%.
- 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 2009–2010, 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 95<sup>th</sup> percentile over the most recent 12 years (from 1999–2000 to 2009–2010).
- In 2009–2010, median blood lead levels by age group were: 1.2 µg/dL for age 1 year and age 2 years; 1.1 µg/dL for ages 3 to 5 years; 0.8 µg/dL for ages 6 to 10 years; 0.7 µg/dL for ages 11 to 15 years; and 0.7 µg/dL for ages 16 to 17. The 95<sup>th</sup> percentile blood lead levels were 4.2, 3.5, 2.8, 2.1, 1.7, and 1.4 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 95<sup>th</sup> 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 2007–2010 was 1.3 µg/dL. The median blood lead level in Black non-Hispanic children ages 1 to 5 years in 2007–2010 was



1.6 µg/dL, higher than the level of 1.2 µg/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 1.5 µg/dL, and for children living in families at or above the poverty level it was 1.2 µg/dL, a difference that was statistically significant.
- The 95<sup>th</sup> percentile blood lead level among all children ages 1 to 5 years was 3.9 µg/dL. The 95<sup>th</sup> percentile blood lead level in Black non-Hispanic children ages 1 to 5 years in 2007–2010 was 5.8 µg/dL, compared with 3.5 µg/dL for White non-Hispanic children and children of “All Other Races/Ethnicities,” and 3.3 µg/dL for Mexican-American children. (See Table B2a.)
  - The 95<sup>th</sup> percentile blood lead level in Black non-Hispanic children was statistically significantly higher than the 95<sup>th</sup> percentile for each of the remaining race/ethnicity groups.
- Among children ages 1 to 5 years in families with incomes below poverty level, the 95<sup>th</sup> percentile blood lead was 4.7 µg/dL, and among those in families at or above the poverty level, it was 3.3 µg/dL, a difference that was statistically significant after accounting for differences by age, sex, and race/ethnicity. (See Table B2a.)
- The 95<sup>th</sup> 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 within each race/ethnicity group. Black non-Hispanic children in families with incomes below the poverty level had the highest 95<sup>th</sup> percentile blood lead level, 6.8 µg/dL, which was 60% higher than for Black non-Hispanic children with families at or above the poverty level. (See Table B2a.)
  - The differences in 95<sup>th</sup> percentile blood lead levels between income groups were statistically significant for Black non-Hispanic children and children of “All Other Races/Ethnicities.” The difference was also statistically significant for Mexican-American children after accounting for differences by age and sex.
- Between 1991–1994 and 2007–2010, median blood lead levels among Black non-Hispanic children ages 1 to 5 years declined 63%: from 4.3 µg/dL to 1.6 µg/dL. Over the same time period, median blood lead levels among Mexican-American children ages 1 to 5 years declined 61%: from 3.1 µg/dL to 1.2 µg/dL, and median blood lead levels among White non-Hispanic children ages 1 to 5 years declined 48%: from 2.3 µg/dL to 1.2 µg/dL. The differences over time were statistically significant for each race/ethnicity. (See Table B2b.)

## Biomonitoring

### Lead

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**Lead (continued)**

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## Biomonitoring

### Lead

**Table B1: Lead in children ages 1 to 5 years: Median and 95<sup>th</sup> percentile concentrations in blood, 1976-2010**

	Blood lead concentration (µg/dL)								
	1976-1980	1988-1991	1991-1994	1999-2000	2001-2002	2003-2004	2005-2006	2007-2008	2009-2010
<b>Median</b>	15.0	3.5	2.6	2.2	1.6	1.6	1.4	1.4	1.2
<b>95<sup>th</sup> percentile</b>	29.0	12.1	9.7	7.0	5.8	5.1	3.8	4.1	3.4

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

**Table B1a: Lead in children ages 1 to 17 years: Blood lead concentrations by age group, 2009-2010**

	Blood lead concentration (µg/dL)						
	Ages 1 to 17 years	Age 1 year	Age 2 years	Ages 3 to 5 years	Ages 6 to 10 years	Ages 11 to 15 years	Ages 16 to 17 years
<b>Median</b>	0.8	1.2	1.2	1.1	0.8	0.7	0.7
<b>95<sup>th</sup> percentile</b>	2.2	4.2	3.5	2.8	2.1	1.7	1.4

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

**Table B2. Lead in children ages 1 to 5 years: Median concentrations in blood, by race/ethnicity and family income, 2007-2010**

	Median blood lead concentration (µg/dL)		
Race / Ethnicity	All Incomes‡ (n=1,653)	< Poverty Level (n=642)	≥ Poverty Level (n=898)
<b>All Races/Ethnicities</b> (n=1,653)	1.3	1.5	1.2
<b>White Non-Hispanic</b> (n=536)	1.2	1.5*	1.2
<b>Black Non-Hispanic</b> (n=338)	1.6	1.7*	1.4
<b>Mexican-American</b> (n=490)	1.2	1.3	1.1
<b>All Other Races/Ethnicities†</b> (n=289)	1.2	1.6	1.1

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

† The “All Other Races/Ethnicities” category includes all other races or ethnicities not specified, together with those individuals who report more than one race.

‡ Includes sampled individuals for whom income information is missing.

\*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.

**Table B2a. Lead in children ages 1 to 5 years: 95<sup>th</sup> percentile concentrations in blood, by race/ethnicity and family income, 2007-2010**

Race / Ethnicity	95 <sup>th</sup> percentile blood lead concentration (µg/dL)		
	All Incomes‡ (n=1,653)	< Poverty Level (n=642)	≥ Poverty Level (n=898)
<b>All Races/Ethnicities</b> (n=1,653)	3.9	4.7	3.3
<b>White non-Hispanic</b> (n=536)	3.5	4.5*	3.4
<b>Black non-Hispanic</b> (n=338)	5.8	6.8*	4.2
<b>Mexican-American</b> (n=490)	3.3	4.1	3.2
<b>All Other Races/Ethnicities†</b> (n=289)	3.5	4.2	2.7

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

† The “All Other Races/Ethnicities” category includes all other races or ethnicities not specified, together with those individuals who report more than one race.

‡ Includes sampled individuals for whom income information is missing.

\*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.

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

Race / Ethnicity	Median blood lead concentration (µg/dL)		
	All Incomes‡ (n=2,367)	< Poverty Level (n=974)	≥ Poverty Level (n=1,253)
<b>All Races/Ethnicities</b> (n=2,367)	2.6	4.0	2.2
<b>White Non-Hispanic</b> (n=623)	2.3	3.2*	2.1
<b>Black Non-Hispanic</b> (n=773)	4.3	5.1	3.5
<b>Mexican-American</b> (n=822)	3.1	3.7	2.6
<b>All Other Races/Ethnicities†</b> (n=149)	2.5	NA**	2.0*

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

† The “All Other Races/Ethnicities” category includes all other races or ethnicities not specified, together with those individuals who report more than one race.

‡ Includes sampled individuals for whom income information is missing.

\*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.

\*\* Not available. The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate), or the RSE cannot be reliably estimated.